

Oral and Maxillofacial Surgery

Edited by
Lars Andersson
Karl-Erik Kahnberg
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Oral and Maxillofacial Surgery

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Preface

We had the idea of writing a quality textbook on oral and maxillofacial surgery with an international appeal. As editors we have personal links with the Americas, Europe, and Asia, and we were very fortunate to be able to recruit an international cast of authors; by utilizing electronic means of communication, we have been able to assure a high level of coordination and standardization. In this way, we have attempted to cover the full scope of the specialty of oral and maxillofacial surgery. We have incorporated recent technical and biological developments within the specialty. We have all been part of a most exciting development within our surgical field

including both surgical techniques and instrumentation. We have been involved in innovative medical and dental research guiding us in our effort to give optimal care to our patients.

We hope that this textbook will have wide appeal among specialists in oral and maxillofacial surgery and residents in training and will be available as a resource for students, residents, and specialists in allied professions.

This book is dedicated to our teachers and mentors (we stand on the shoulders of giants) as well as the dedication and sacrifices of our wives Karin, Ingrid, and Ann.



Lars Andersson



Karl-Erik Kahnberg



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The above photograph (left to right: Karl-Erik Kahnberg, Lars Andersson, Tony Pogrel) was taken at the 18th International Conference in Oral and Maxillofacial Surgery in Bangalore, India in November 2007.

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Chapter 1

Patient Evaluation

Alan S. Herford and Wayne K. Tanaka

This chapter will cover how to obtain a patient history and carry out a full physical examination. It will cover comorbidities and systemic diseases of relevance to the oral and maxillofacial surgeon in the cardiovascular system (including cardiac disease and hypertension), the pulmonary system (including tobacco use and asthma), and also the endocrine systems, to cover obesity.

Suitable imaging and laboratory studies will also be discussed to arrive at a diagnosis of the patient's medical history, assess anesthetic and surgical risks, and determine whether a procedure would be best carried out in the office or as an inpatient taking into account the medical history.

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The goal of preoperative evaluation is to reduce patient risk and the morbidity of surgery and is based on the premise that it will modify patient care and improve outcome. The preoperative evaluation has several components and should be guided by the patient and surgery being contemplated. It is important to understand that some patients will require an in-depth, thorough examination prior to undergoing surgery, whereas others may benefit from a more focused examination. The type depends on many factors including the age and health of the patient, existing comorbidities, and the type of surgical procedure

planned. Thus, it is important for the clinician to understand how to perform a detailed, in-depth history and physical examination.

The Joint Commission for the Accreditation of Healthcare Organizations (JCAHO) requires that all patients receive a preoperative anesthetic evaluation and the American Society of Anesthesiologists (ASA) has approved Basic Standards for Preoperative Care which outline the minimum requirements for a preoperative evaluation. Preoperative patient assessment is important in order to develop a safe and appropriate surgical and anesthetic plan.¹⁻¹¹ During the preoperative assessment the clinician interviews either the patient or knowledgeable guardian to obtain information. This is followed by a physical examination with special emphasis on the cardiovascular and respiratory systems to help determine risk. Laboratory tests, imaging, and consultations are ordered as deemed necessary based on information obtained during the history and physical examination. Finally, diagnoses are formulated and the treatment options are discussed with the patient followed by obtaining an informed consent.

Obtaining a patient history

The importance of an accurate, detailed history cannot be overemphasized because it provides the framework on which the clinician builds an accurate diagnosis and treatment plan (Fig. 1.1) An inaccurate or incomplete evaluation may lead to a delay in treatment, unnecessary testing, or misdiagnosis.

It is often helpful to review previous medical records. This can provide important information and save time during the interview process. Information such as medications and doses and history of previous surgeries are some examples that can be gleaned from the previous medical records. The clinician may uncover a history of a difficult airway or a history of malignant hyperthermia, and the patient's response to surgical stress and specific anesthetics may be

4 Basic Principles

Schedule procedure:
Reason for procedure:
Brief HPI: Age _____ Gender _____ ☐ See admission/prior HPI dated _____
☐ Patient's records & chart reviewed ☐ ROS: cardiac & pulmonary unremarkable

Current medications:
Allergies: ☐ NKDA
Previous surgeries:
Previous hospitals:

☐ PHYSICAL EXAM (Check box if normal)
BP _____ / _____ PR _____ RR _____ /min Temp _____ Weight _____ lbs
☐ General appearance ☐ Mental status (Oriented ×3)
☐ AIRWAY
☐ HEART
☐ LUNGS
☐ ABDOMEN
☐ Other

ASA PHYSICAL STATUS (Circle) 1 2 3 4 5
(1) Normal (2) Mild systemic disease (3) Severe but not incapacitating (4) Incapacitating & threat to life (5) Moribund
☐ Risks, benefits and alternatives of sedation/analgesia and procedure discussed with patient/family and accepted.
☐ THE PATIENT IS AN APPROPRIATE CANDIDATE TO UNDERGO THE PLANNED PROCEDURE AND GENERAL ANESTHETIC

Doctor's Signature _____

Date _____

Fig. 1.1 Sample history and physical examination form.

evaluated. If the patient presents in an altered state, these records can be especially helpful. Preoperative questionnaires and computer-driven programs are becoming more common as a way to gather information and save time. This should help to alert the clinician on areas to focus on but should not be used as an alternative to interviewing the patient.

The clinician must assess the reliability of the person giving the history. Every patient should be asked about their chief complaint (CC). This should be transcribed into the medical record in the patient's own words. The chief complaint assists the clinician in establishing priorities during the history-taking process.

The patient should be asked to describe the history of the present illness (HPI). Information should be gathered regarding onset, intensity, quality, location, duration, radiation, and any exacerbating or relieving factors. Constitutional symptoms that relate to the present illness should also be noted. Examples of pertinent positives and negatives with regard to the chief complaint may include fever, chills, loss of weight, weakness, etc.

The past medical history (PMH) alerts the clinician to any coexisting illnesses that may have an impact on any planned surgeries. Information regarding the severity of an illness should be obtained. For example if a patient reports a history of asthma, the severity and frequency of episodes, previous hospital treatments, and current control should be ascertained. Past surgical history can also help to identify factors that may impact the ability for a patient to undergo a safe surgical procedure. Patients should be asked about medications they are taking as well as any over-the-counter and herbal products. Any allergies to drugs should also be documented, including what type of allergic response a patient may have experienced. A family history (FH) may reveal risk factors for patients as well as the possibility of inherited illnesses such as hemophilia or malignant hyperthermia.

The social history (SH) of a patient should include information regarding their social support system and also any habits such as tobacco, alcohol, or illicit drug use. These habits may adversely affect healing and also increase a patient's risk for undergoing a planned surgical procedure.

A review of systems (ROS) is a comprehensive method of inquiring about a patient's symptoms on an organ system basis. The review of systems may reveal undiagnosed medical conditions unknown to the patient. Concerns raised in the cardiovascular and respiratory systems for example may have importance with regard to a patient undergoing a safe surgical procedure. Patients should also be asked about the presence or recent history of an upper respiratory infection. Symptoms such as shortness of breath, cough, wheezing, stridor, snoring, or sleep apnea can alert the clinician to underlying illness that may increase the risk of morbidity associated with a procedure.

Physical examination

During the physical exam the clinician further reinforces or disproves impressions gained during the history-taking portion. Vital signs are recorded at the beginning of the physical exam. These include blood pressure, pulse rate, respiratory rate, and temperature. Next the patient's general appearance should be noted. It is important when documenting findings to only use commonly used, accepted abbreviations in order to avoid confusion. The physical exam should proceed in a stepwise, systematic manner. Evaluation typically involves inspection, palpation, percussion, and auscultation of the organ system being evaluated. The areas involved typically include the head, eyes, ears, nose, and throat (HEENT) region, in addition to the lungs, abdomen, heart, genitourinary, musculoskeletal, skin, and neurological exam. The detail in which these examinations take place is based on the health of the patient, including any comorbidities, and the type of surgical procedure planned. The examination may be cursory in healthy patients or extensive in patients with coexisting disease. For patients sustaining severe trauma a neurological examination should include a Glasgow Coma Score (Table 1.1). A cranial nerve exam should be performed to uncover any abnormalities (Table 1.2). For patients undergoing facial trauma, the eyes should be evaluated for any afferent pupillary light defects (Fig. 1.2).

Table 1.1 Glasgow coma scale.

EYE RESPONSE (E)

- 4 = Open spontaneously
- 3 = Open to verbal command
- 2 = Open to pain
- 1 = No response

VERBAL RESPONSE (V)

- 5 = Oriented, converses
- 4 = Disoriented, converses
- 3 = Inappropriate responses
- 2 = Incomprehensible
- 1 = No response

MOTOR RESPONSE (M)

- 6 = Obeys verbal command
- 5 = Localizes to pain
- 4 = Withdrawal from pain
- 3 = Decorticate (flex) to pain
- 2 = Decerebrate (extend) to pain
- 1 = No response to pain

NOTE: Scoring exam used to monitor changes in level of consciousness. Score is sum of eye, verbal, and motor responses. Range 3 (worst) to 15 (normal).

It is important to thoroughly assess the airway in patients undergoing anesthesia. This evaluation involves determination of the thyromental distance, the ability to flex the base of the neck and extend the head, and examination of the oral cavity including the dentition and interincisal opening. The Mallampati classification is useful for assessing the tongue size relative to the oral cavity, although by itself the

Table 1.2 Cranial nerves.

Cranial nerve	Function
I. Olfactory	Smell
II. Optic	Visual acuity/fields and fundoscopic examination of each eye
III, IV, VI. Oculomotor, Trochlear, Abducens	Eyelid opening, extraocular movements (IV, superior oblique; VI, lateral rectus; III, all others), direct and consensual pupillary light reflexes
V. Trigeminal (V, ophthalmic; V2, maxillary; V3, mandible)	Corneal reflex, facial sensation, jaw opening, bite strength
VII. Facial	Eyebrow raise, eyelid close, smile, frown, pucker, taste
VIII. Vestibulocochlear	Auditory acuity of each ear, Rinne (air vs bone conduction) and Weber (lateralizing) tests, oculocephalic reflex (doll's eye maneuver), oculovestibular reflex (ear canal caloric stimulation)
IX, X. Glossopharyngeal, Vagus	Palate elevation, swallowing, posterior taste, phonation, gag reflex
XI. Spinal Accessory	Lateral head rotation, neck flexion, shoulder shrug
XII. Hypoglossal	Tongue protrusion and strength on lateral deviation

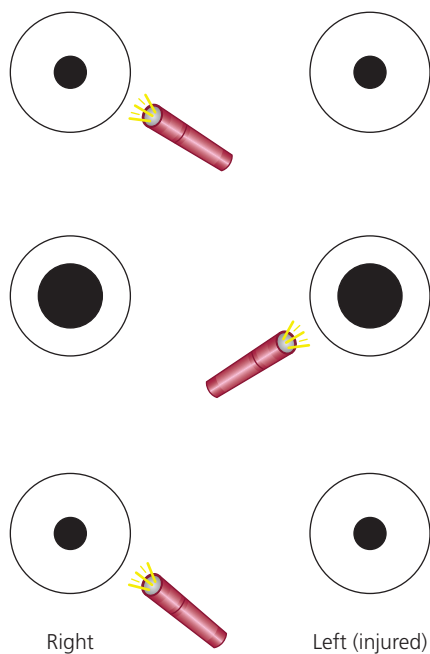


Fig. 1.2 Afferent pupillary light defect. When the optic nerve is damaged the sensory stimulus sent to the midbrain is reduced. The pupil responds less vigorously and dilates from its prior constricted state (Marcus Gunn pupil).

Mallampati classification has a low positive predictive value in identifying patients who are difficult to intubate (Fig. 1.3).^{12,13} The cervical spine should be assessed and cleared for patients that have undergone significant trauma.

The remainder of the physical examination should proceed in a systematic way. For a complete descrip-

tion of examination techniques the reader is advised to consult textbooks on physical diagnosis.

Comorbidities/systemic diseases

The clinician needs to assess potential risk factors and understand their effect on treatment. Changes in heart rate, rhythm, blood pressure, preload, afterload, and inotropy may occur during surgery and these can have deleterious effects especially in patients with comorbidities. The risks for complications are greatest when caring for patients who are already medically compromised. Many significant untoward events can be prevented by careful preoperative assessment along with attentive intraoperative monitoring and support.¹⁴

Cardiovascular system

Cardiac disease

Cardiac complications following non-cardiac surgery constitute an enormous burden of perioperative morbidity and mortality. More than one million operations annually are complicated by adverse cardiovascular events, such as perioperative myocardial infarction or death from cardiac causes.¹⁵ Common cardiac risk factors include diabetes, hypertension, family history of heart disease, hypercholesterolemia, and obesity. Certain populations of patients, such as the elderly, diabetics, or women, may present with more atypical features.

Methods for evaluating a patient's cardiac risk preoperatively include a careful history, including exercise tolerance, physical examination, and electrocardiogram (EKG). Based on this information, various risk indices, guidelines, and algorithms can assist the clinician in deciding which patients can undergo surgery without further testing and which patients may benefit from further cardiac evaluation or medical therapy prior to surgery. Risk assessment involves evaluating patients' comorbidities and exercise tolerance, as well as the type of procedure to be performed to determine the overall risk of perioperative cardiac complications. Exercise tolerance is a major determinant of cardiac risk and need for further testing. Beta blockade has shown clear benefits in risk reduction whereas revascularization procedures, such as coronary artery bypass grafting, have not been shown useful in reducing non-cardiac surgical risk.^{16–18}

The Goldman Index is a multifactorial index used to assess cardiac risk associated with non-cardiac surgeries. It is based on a study by Goldman *et al.*¹⁹ that prospectively studied 1001 patients. A risk index was formulated based on potential risk factors for cardiac complication and actual complications. For class I patients (0–5 points) only 0.7% had life-threatening complications whereas class IV patients

Class	Direct visualization, patient seated	
Class I	Able to visualize the soft palate, fauces, uvula, anterior and posterior tonsillar pillars	
Class II	Able to visualize the soft palate, fauces, and uvula; the anterior and posterior tonsillar pillars are hidden by the tongue	
Class III	Only the soft palate and base of uvula are visible	
Class IV	Only the soft palate can be seen (no uvula seen)	

Fig. 1.3 Mallampati classification.

(>26 points) experienced life-threatening complications 22% of the time. A previous history of congestive heart failure was the factor most predictive of complications, followed by a myocardial infarction within the previous 6 months. Detsky *et al.*²⁰ included unstable angina and remote myocardial infarction as additional risk factors. They simplified the scoring system into three classes attempting to improve predictive accuracy. In another update of the Goldman

Cardiac Risk Index, Lee *et al.*²¹ studied 4315 patients aged 50 years and older who were undergoing elective, major non-cardiac procedures. Six independent predictors of complications were identified (high-risk type of surgery, history of ischemic heart disease, history of congestive heart failure, history of cerebrovascular disease, preoperative treatment with insulin, and preoperative serum creatinine >2.0 mg/dl). Cardiac complication rates rose with an increase in

the number of risk factors present. Rates of major cardiac complications with 0, 1, 2, or 3 of these factors were 0.5, 1.3, 4, and 9% respectively, in the derivation cohort and 0.4, 0.9, 7, and 11% respectively, among 1422 patients in the validation cohort.

Angina pectoris can be a symptom of ischemic heart disease. It presents as substernal chest pain that may radiate to the arm, neck, or mandible and represents a reduced delivery of oxygen to the myocardium. It most commonly is the result of coronary heart disease and is classified as stable, unstable, or variant.²² Stable angina pectoris is characterized by no change over 2 months with regards to precipitating factors, frequency, intensity, and duration of attacks. Unstable angina pectoris represents a patient who has experienced worsening of the symptoms recently. Unstable angina pectoris is worrisome because patients are at an increased risk for developing a myocardial infarction.^{23,24} Variant pectoral angina (Prinzmetal's angina) may occur in patients without coronary artery disease and represents vasospasm of the coronary arteries. Patients with angina pectoris should be thoroughly evaluated to determine surgical risk.

The use of vasoconstrictors in local anesthetics in patients with coronary heart disease is controversial in the literature.²⁵ A study by Neves *et al.*²⁶ found no difference in blood pressure, heart rate, or evidence of ischemia in patients treated with or without vasoconstrictor.

Studies estimate the risk of perioperative myocardial infarction to be 0.13% for patients without a history of myocardial infarction and approximately 6% in patients with a history of previous myocardial infarction.^{27,28} Multiple studies have demonstrated an increased incidence of reinfarction if the myocardial infarction was within 6 months of surgery.^{23,29,30}

Congestive heart failure is the single most important risk factor for perioperative cardiac morbidity. Perioperative management includes optimizing fluid management, and maximizing drugs such as inotropes, vasodilators, and antidysrhythmics. Symptoms obtained during the history-taking portion may be worrisome for congestive heart disease. These include findings such as orthopnea and dyspnea. The patient should be questioned about limitation of physical activity such as ability to climb stairs or how far a patient can walk. Signs such as ankle edema, ascites, distended neck vein, and rales on pulmonary auscultation may help to identify congestive heart failure.

Valvular disease can affect the risk of a procedure. Symptoms of valvular disease should be sought, such as angina, syncope, or congestive heart failure from aortic stenosis which would require further evaluation. A history of valvular disease may dictate the need for subacute bacterial endocarditis prophylaxis (Table 1.3). Valvular heart disease presents with a murmur on physical exam. The clinician must determine the severity of the murmur and whether the patient requires preoperative antibiotic prophylaxis to prevent endocarditis.³¹ Aortic stenosis, aortic regurgitation, mitral valve stenosis, mitral valve insufficiency, mitral valve prolapse, and cardiomyopathy are some cardiac conditions which can be detected by cardiac auscultation. Goldman and Caldera recognized aortic stenosis as an independent risk factor for poor outcome increasing the risk for perioperative cardiac death by a factor of 14.³²

Algorithms for preoperative evaluation of cardiac patients undergoing noncardiac surgery are useful in guiding the need for further testing and evaluation. These algorithms are based on the available evidence and expert opinion that integrates clinical history, surgery-specific risk, and exercise tolerance. Implementation of American College of Cardiology / American Heart Association (ACC / AHA) guidelines for cardiac risk assessment prior to non-cardiac surgery reduces preoperative resource utilization, improves medical treatment, and preserves a low rate of perioperative cardiac complications.^{33,34} Studies have shown perioperative cardiac morbidity is greatly reduced by perioperative β -adrenergic blockade administration.^{35,36} The American College of Physician Guidelines uses the Detsky modification of the cardiac risk index.^{20,37} Patients that are class II or III are considered high risk. For patients that are classified as class I, other clinical factors can be used to further stratify risk. The Guidelines suggest that there is insufficient evidence to recommend diagnostic testing for non-vascular surgery patients.

In patients for whom further work-up is deemed necessary, this may include cardiovascular tests such as EKG, non-invasive cardiovascular tests such as an exercise EKG or pharmacologic testing in patients who are unable to exercise. The exercise EKG is the most cost-effective and least invasive method for detecting ischemia, with a sensitivity of 70–80% and

Table 1.3 AHA SBE prophylaxis regimens.

	Adult	Child
Dental, oral, upper respiratory tract	Amoxicillin	PO
	2 g PO 1 h prior	Amoxicillin
	Clindamycin ^a	50 mg/kg
	600 mg PO 1 h prior	Azithromycin
	600 mg IV/IM 30 minutes prior ^b	15 mg/kg
	Cephalexin or Cefadroxil ^a	Clindamycin
	2 g PO 1 h prior	10 mg/kg
	Azithromycin ^a	
	500 mg PO 1 h prior	IV/IM
	Ampicillin ^b	Ampicillin
	2 g IV or IM 30 minutes prior	50 mg/kg
	Cefazolin ^{a,b}	Clindamycin
	1 g IV/IM 30 minutes prior	10 mg/kg
		Gentamicin

^a Penicillin allergy.

^b PO intolerant.

specificity of 60–75% for identifying coronary artery disease. Pharmacologic stress thallium imaging is useful in those patients unable to exercise. Dipyridamole or adenosine is administered as a coronary vasodilator to assess flow heterogeneity. A redistribution defect is predictive of perioperative cardiac events. Dopamine can also be used to increase myocardial oxygen demand, by increasing the heart rate and blood pressure. Stress echocardiography is another preoperative test that can reveal regional wall motion abnormalities which represent areas at risk for myocardial ischemia. Echocardiography can assess cardiac ejection fraction and also provide information regarding valvular function. Coronary angiography provides information regarding the coronary anatomy. Unlike exercise or pharmacologic stress tests, coronary angiography provides anatomic, not functional, information.

Hypertension

Hypertension is a common disease which can increase perioperative cardiac risk. Hypertension has been associated with an increase in the incidence of silent myocardial ischemia and infarction.³⁸ The Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure recently revised their definition.³⁹ Hypertensive patients with left ventricular hypertrophy are at a higher perioperative cardiac risk than non-hypertensive patients.⁴⁰

Controversy exists regarding whether to delay a surgical procedure in a patient with untreated or poorly controlled hypertension. Aggressive treatment of high blood pressure does diminish long-term risk. A study often quoted as the basis for delaying surgery for patients with a diastolic blood pressure greater than 110 mmHg actually demonstrated no major morbidity in that group of patients.³² Other authors have found little association between blood pressures less than 180 mmHg systolic or 110 mmHg diastolic and postoperative outcomes. Patients with severe hypertension are more prone to perioperative myocardial ischemia, ventricular dysrhythmias, and lability in blood pressure. For patients with blood pressures greater than 180/110 mmHg there is no absolute evidence that postponing surgery will decrease the cardiac risk.⁴¹ For patients without end-organ changes, such as renal insufficiency or left ventricular hypertrophy, it may be appropriate to proceed with surgery. However patients with a markedly elevated blood pressure and new onset of a headache should have surgery delayed for further medical treatment. Patients with hypertension may have a contracted intravascular volume and therefore have an increased susceptibility to vasodilator effects of commonly used sedative and anesthetic agents. For elective surgery it is best to have the patient's blood pressure optimized prior to surgery.

Risk factors for hypertension include smoking, hypercholesterolemia, increasing age, family history of cardiovascular disease, and diabetes. Untreated hypertension commonly causes coronary heart disease, cardiomegaly, congestive heart failure, and end-organ damage. When evaluating a patient with hypertension, it is important to determine the presence of end-organ damage (heart, lung, and cerebrovascular systems). An elevated systolic blood pressure may be a better predictor of postoperative myocardial ischemia than elevated diastolic blood pressure.³⁹

Pulmonary system

Pulmonary complications are a major cause of morbidity for patients undergoing a surgical procedure. They occur more frequently than cardiac complications with an incidence of 5–10% in those having major non-cardiac surgeries.^{42,43} Perioperative pulmonary complications include atelectasis, pneumonia, bronchitis, bronchospasm, hypoxemia, and respiratory complications.⁴⁴ Both the site and type of surgery are the strongest predictors of complications. With regards to the surgical site, thoracic surgery is associated with the highest risk for perioperative pulmonary complications, whereas major head and neck surgery is associated with a 24–47% risk of pulmonary complications.^{45,46} A decrease in postoperative vital capacity and functional residual capacity contributes to hypoxemia and atelectasis.⁴⁷ General anesthesia also results in mechanical changes such as a decrease in the functional residual capacity as well as an altered diaphragmatic motion. This can lead to a mismatch between ventilation and perfusion creating shunting and dead space ventilation. The functional residual capacity may take up to 2 weeks to return to baseline.⁴⁸ A general anesthetic can also cause inhibition of mucociliary clearance, increased alveolar capillary permeability, inhibition of surfactant release, and increased sensitivity of the pulmonary vasculature to neurohormonal mediators. The duration of anesthesia has been shown to be a risk factor for postoperative pulmonary complications, with morbidity rates increasing after 2–3 hours.⁴⁹

For patients with an upper respiratory illness, surgery should be delayed if possible for at least 2 weeks after resolution of the illness. A recent study found a 10% incidence of severe complications, respiratory as well as cardiac arrest, pneumonia, and prolonged intubation due to increased sputum.⁵⁰

During the presurgical evaluation, the clinician should obtain information about exercise tolerance, chronic cough, or unexplained dyspnea. On physical exam, findings of rhonchi, wheezing, decreased breath sounds, dullness to percussion, and a prolonged expiratory phase are important. Preoperative pulmonary function tests are usually reserved for patients undergoing lung resection or those undergoing major surgery who have unexplained pulmonary

signs and symptoms after a history and physical examination.

Tobacco

Tobacco is an important risk factor. Even among smokers without chronic lung disease, smoking is known to increase carboxyhemoglobin levels, decrease ciliary function, and increase sputum production. Nicotine also stimulates the cardiovascular system. Discontinuing smoking for 2 days can decrease carboxyhemoglobin levels, abolish nicotine effects, and improve mucus clearance. However, a study by Warner showed that smoking cessation for at least 8 weeks was necessary to reduce the rate of postoperative pulmonary complications.⁵¹ Patients who smoke often show an increase in airway reactivity under general anesthesia. Administration of a bronchodilator such as fluticasone preoperatively may be beneficial in this group of patients.⁵²

Asthma

Asthma causes episodic narrowing of the small airways, which produces wheezing and dyspnea. Patients should be questioned regarding precipitating factors, frequency and severity of attacks, medications used, and current status. Frequent use of bronchodilators, hospitalizations for asthma, and the requirement for systemic steroids are all indicators of the severity of the disease. Airway hyper-reactivity may persist for several weeks after an episode of asthma.⁵³ Patients may require xanthine-derived bronchodilators, such as theophylline, and corticosteroids. Cromolyn sodium is useful for protecting against acute attacks but is ineffective once broncho-

spasm occurs. Sympathomimetic amines in aerosol form such as epinephrine or metaproterenol can be administered if wheezing begins. Clinicians should be aware of the role of anxiety in initiating bronchospasm and also the potential adrenal suppression in patients receiving corticosteroid therapy. Elective therapy should be delayed if a respiratory tract infection or wheezing is present. In addition to bronchodilators, perioperative steroids may be beneficial as prophylaxis for severe asthmatics. The possibility of adrenal insufficiency is also a concern for patients who have received extended treatment with steroids and should be administered "stress doses" of steroids perioperatively. The risks of complications are low for asthmatics treated with short-term steroids undergoing surgery.⁵⁴ There is no association with impaired wound healing or infections. It is recommended for patients using inhaled steroids that these be regularly administered starting at least 48 hours prior to surgery for optimal effectiveness.⁵⁵

Endocrine system

Endocrine disorders can affect the course of anesthesia and should be evaluated preoperatively (Table 1.4). A decrease in adrenal cortical activity such as seen in Addison's disease, may lead to a decreased production and availability of cortisol and aldosterone thus altering cardiovascular stability. Patients who are taking glucocorticosteroids may have suppression of the pituitary gland and may require supplementation of cortisol (rule of twos). In patients on long-term corticosteroids, the clinician should have a high index of suspicion for adrenal cortical suppression and Cushing's syndrome. Classic symptoms found in Cushing's syndrome include

Table 1.4 Clinical manifestations of endocrine diseases.

	Diabetes mellitus	Pheochromocytoma	Addison's disease	Hyperthyroidism	Hypothyroidism	Hyperparathyroidism
General	Polydipsia/polyuria	Heat intolerance, palpitations, intermittent hypertension, sweating	Fatigue, sweating	Heat intolerance, warm, moist skin	Cold intolerance	Polydipsia
Cardiovascular	Cerebrovascular disease	Tachycardia, bounding pulse	Sudden drops in blood pressure	Tachycardia, atrial fibrillation, congestive heart failure	Bradycardia, cardiomegaly,	Hypertension, heart block
Neurologic	Blurred vision, retinopathy	Headaches, diaphoresis	Dizziness, headache	Nervousness, tremor, hyperactive reflexes	Slow mental function, hyporeflexia	Weakness, lethargy, headache, insomnia
Gastrointestinal	Weight loss	Weight loss, abdominal pain	Weight loss, diarrhea	Diarrhea	Delayed gastric emptying	Anorexia, nausea, vomiting, constipation
Musculoskeletal	Charcot joints	Signs of poor perfusion	Muscle weakness, joint and muscle pain	Muscle weakness, bone resorption	Large tongue, amyloidosis	Bone pains, arthritis, pathologic fractures
Renal	Kidney disease	Urinary retention	Urinary loss of sodium	Chronic renal failure	Impaired free water clearance	Polyuria, hematuria

moon facies, striations of the skin, trunk obesity, hypertension, easy bruisability, and hypovolemia. These patients should have any fluid and electrolyte abnormalities corrected preoperatively.

Patients with a pheochromocytoma may present with overproduction of epinephrine and norepinephrine in the adrenal medulla which can lead to hypertension and tachycardia intraoperatively. The classic findings for pheochromocytoma include intermittent hypertension, headache, diaphoresis, and tachycardia. In patients with other endocrine tumors, a pheochromocytoma should be ruled out as part of a multiple endocrine neoplasia (MEN) syndrome.

Thyroid and parathyroid disease have clinical manifestations that are important to evaluate preoperatively. Disorders of the thyroid can present as hypothyroidism or hyperthyroidism. The presurgical evaluation should focus on the signs and symptoms of hyperthyroidism and hypothyroidism. Thyroid function tests including thyroid stimulating hormone (TSH), triiodothyronine (T3), and thyroxine (T4) are used to assess for disorders of the thyroid. Patients with hypothyroidism may exhibit cold intolerance, myxedema, fatigue, and/or depression. Severe hypothyroidism can lead to coma, cardiovascular collapse, and heart failure, and surgery should be postponed until it is corrected. Hyperthyroidism, such as seen in Grave's disease, presents with symptoms including hyperexcitability, weight loss, hypertension, and tachycardia. Thyroid storm can occur during anesthesia. Medications such as propylthiouracil or methimazole may be helpful preoperatively to reduce thyroxine secretion. Beta-blockers may be useful to stabilize the adrenergic activity during surgery. Large goiters may impact the patency of the airway and make intubation more difficult. Patients with hyperparathyroidism often have hypercalcemia and the serum calcium level should be determined preoperatively.

The pituitary gland has control of many glands and organs. Increased production of pituitary hormones can lead to secondary hyperthyroidism, secondary Cushing's syndrome, and acromegaly.

Diabetes mellitus is the most common endocrinopathy and has both acute and chronic disease manifestations. The disease process causes impairment of normal blood flow and subsequent end-organ damage. Type 1 diabetes is characterized by insulin dependency and typically occurs at a young age, whereas type 2 diabetes is often non-insulin dependent and occurs later in life. Preoperative assessment of patients with diabetes includes determining their degree of blood sugar control and evaluation of any end-organ damage. Perioperative complications increase with poorly controlled diabetes with end-organ damage. Diabetics have an increased risk of coronary artery disease, perioperative myocardial infarction, hypertension, and congestive heart failure. They are also at an increased risk for cerebral vascular, peripheral vascular, and renal vascular disease.

Myocardial infarction or ischemia may be "silent" if diabetic autonomic neuropathy is present. Diabetes accelerates the progression of atherosclerosis leading to a higher incidence of coronary artery disease than in non-diabetics. There is also a higher incidence of silent myocardial infarction in this group of patients. Eagle *et al.*³⁴ demonstrated that diabetes is an independent risk factor for perioperative cardiac morbidity. An EKG should be obtained to examine for the presence of Q waves. Laboratory assessments for diabetics include a blood sugar and HbA1c. A blood glucose level gives a value at one point in time whereas the glycosylated fraction of adult hemoglobin (HbA1c) gives a better assessment of control over the previous 2 months. Perioperative concerns with diabetic patients include poor healing with possible infection and diabetic ketoacidosis. Hypoglycemia should be avoided in order to prevent central nervous system damage. Managing surgical patients with diabetes must take into account the type of diabetes, how well the patient controls their blood sugar, and the stress associated with the surgical procedure.

Obesity

A patient is considered obese when their body weight is 20% or more above ideal weight. Obesity can be measured by the body mass index (BMI) which is derived by dividing the weight in kilograms by the height in meters squared ($BMI = Wt/ht^2$).

A BMI greater than 30 suggests increased morbidity due to stroke, heart disease and diabetes.⁵⁶ At a minimum, these conditions indicate the need for close evaluation of the patient's airway and cardiac and pulmonary status.⁵⁷ Even with an adequate airway, ventilation may be difficult because of the patient's size and a tendency toward hypoxemia. There may also be significant cardiovascular changes.

On the other hand, the clinician should not dismiss a low BMI, especially with evidence suggesting an eating disorder. Nutritional deficiency may be present along with significant cardiac changes, fluid and electrolyte imbalances, delayed gastric emptying, and severe endocrine abnormalities.⁵⁸

Other organ systems

Disease affecting the renal system has important implications for fluid and electrolyte management, as well as the metabolism of certain drugs. Liver disease is associated with altered protein binding and volume distribution of drugs, as well as coagulation disorders.

Patients should be questioned regarding bleeding problems. This includes questions regarding bruising, bleeding, and the use of medications that influence platelet function such as aspirin, other non-steroidal anti-inflammatory drugs, and anticoagulants. Medications such as acetyl-salicylic acid and other non-selective non-steroidal anti-inflamma-

Table 1.5 Clotting indices.

Measurement	Normal result	Comments
Prothrombin time (PT)	11–15 s	Measures intrinsic and common pathways (Factors I, II, V, VII, and X)
Activated partial thromboplastin time (aPTT)	20–35 s	Measures extrinsic and common pathways (Factors I, II, V, VIII, IX, X, XI, and XII)
Bleeding time	2–7 minutes	Tests platelet and vascular phases (independent of coagulation cascade)
Thrombin time	6.3–11.1 s	Measures ability to form initial clot from fibrinogen
International normalized ratio (INR)	1.0	(PT patient/PT control) × international sensitivity index (ISI)
Fibrinogen	200–400 mg/dl	Factor I

tory analgesics can inhibit platelet function. Liver disease may decrease the amount of clotting factors. Inherited disorders such as von Willebrand's disease and hemophilia A and B may require administration of various factors preoperatively to minimize bleeding risk.⁵⁹ Reduced ristocetin cofactor levels are often seen in patients with von Willebrand's disease.⁶⁰

Screening tests for bleeding disorders include prothrombin time (PT) or international normalized ratio (INR) to assess the extrinsic and final common pathway and activated partial thromboplastin time (PTT) to test the intrinsic and final common pathways (Table 1.5). Platelet counts may be helpful in patients with thrombocytopenia. Bleeding times measure qualitative platelet function. They are used less and may be unreliable.

Patients with transient ischemic attacks (TIAs) or stroke should undergo a thorough evaluation. Patients who have had unstable TIAs or a stroke within the previous 6 months should have their surgery delayed if possible. Surgery can lead to a hypercoagulable state which may exacerbate cerebral arterial disease. Questioning should include the degree of compliance with medical therapy.

Controversy exists regarding the management of patients on anticoagulation therapy. Some clinicians prefer to admit a patient to the hospital and begin therapy with heparin. This can then be stopped immediately prior to surgery followed by restarting the heparin after the surgical procedure and while beginning oral therapy with warfarin. Low-molecular-weight heparin can be considered as a way to bridge anticoagulation during the interruption of warfarin for high bleeding-risk procedures.⁶¹ A study by Ferrieri *et al.*⁶² noted a low 1.96% risk of bleeding complications in patients undergoing oral surgical procedures with an INR ≤ 5.5 who did not stop their oral anticoagulant therapy. Other authors have found that it is not necessary to reduce oral anticoagulant therapy.^{63–65}

Disorders of the musculoskeletal system have been associated with an increased risk of malignant hyperthermia. Osteoarthritis may result in difficulty exposing the glottic opening during intubation. Rheumatoid

arthritis is a multisystem disease and a thorough review of systems should be performed. These patients may have restrictive lung disease, temporomandibular joint manifestations with restricted opening and hypoplastic jaw, pleural effusions, pericarditis, and anemia. Epilepsy is a common neurological disorder. Patients should be questioned on frequency, duration, and type of seizures they experience. Risks of pulmonary aspiration and respiratory insufficiency during seizure episodes should be taken into account.

Imaging

A patient's presentation will dictate which films are required. Radiographs such as plain films, cone beam, fanning computed tomography (CT), nuclear scans, and arteriography are helpful in various circumstances. The risks associated with these studies should be weighed against the added benefit from them.

Laboratory studies

Many institutions have preadmission screening test algorithms based on factors such as age of the patient (Table 1.6). Preoperative laboratory tests should be ordered based on defined indications such as positive findings on a history and physical exam. A thorough history and physical examination can be used to identify those medical conditions that might affect perioperative management and direct further laboratory testing. A study by Golub *et al.*⁶⁶ reviewed the records of 325 patients who had undergone preadmission testing prior to surgery. Of these 272 (84%) had at least one abnormal screening test, while only 28 surgeries were canceled or delayed. Only three patients potentially benefited from preadmission testing, including a new diagnosis of diabetes in one and non-specific EKG changes in two. Another study by Narr *et al.*⁶⁷ demonstrated minimal benefits from routine testing and proposed that routine laboratory screening tests were not required in healthy patients.

Table 1.6 Sample preadmission screening test algorithm. (EBL, estimated blood loss; HTN, hypertension; IVDA, intravenous drug abuse; LMP, last menstrual period; ABG, arterial blood gases; CBC, complete blood count; PT, prothrombin time; PTT, partial thromboplastin time; LFTs, liver function tests; CXR, chest X-ray; EKG, electrocardiogram; HCG, human chorionic gonadotropin; UA, urinalysis; PFTs, pulmonary function tests; T/S, type and screen.)

CIRCLE APPROPRIATE LABS TO ORDER												
Preoperative condition	ABGs	CBC	PT/PTT	Lutes	BUN/Creat	Blood/Glucose or Accucheck	LFT	CXR	EKG	Hcg preg/UA	PFTs	T/S
Possible EBL >500 ml		X										X
Neonates		X										
Age: >40 yr									X			
Age: >75 yr		X				X			X			
Cardiovascular disease/ chronic HTN		X			X			X	X			
Use of diuretics, digoxin				X*	X				X			
Severe pulmonary disease/ prethoracotomy	X	X						X	X		X	
Malignancy/radiation/ chemotherapy		X,plt		X	X							
Hepatic disease		X,plt					X					
Chronic alcoholism		X,plt		X	X		X		X			
Renal disease (dialysis)		X		X*	X*			-/+	-/+			
Bleeding disorder/ anticoagulant therapy		X,plt*	X*									
Diabetes				-/+	X	X*			>30 yr			
Possible pregnancy/ gyn surgery										X*		
IVDA									>30 yr			

Note: Not all diseases are included. Therefore, the physician should use own judgment regarding patients having diseases that are not listed.

In patients with stable medical conditions, labs and EKGs within the last 3 months, and CXR within the last year, will be acceptable.

X Items should be done within 72 hours of surgery.

* Urine pregnancy test if LMP >21 days with possibility of pregnancy or menstruating females <18 years of age, all women undergoing tubal ligation and all women having a hysterectomy who are in their reproductive years or who are experiencing the first year of menopause.

Type & screen

Type & cross _____ units PRBCs

Other Labs – Specify

Provider signature

Table 1.7 American Society of Anesthesiologists physical status classification.

Status	Disease state
ASA class 1	No organic, physiologic, biochemical, or psychiatric disturbance
ASA class 2	Mild to moderate systemic disturbance that may not be related to the reason for surgery
ASA class 3	Severe systemic disturbance that may or may not be related to the reason for surgery
ASA class 4	Severe systemic disturbance that is life threatening with or without surgery
ASA class 5	Moribund patient who has little chance of survival but is submitted to surgery as a last resort (resuscitative effort)
Emergency operation (E)	Any patient in whom an emergency operation is required

In a follow-up study a cohort of patients was followed who had no preoperative testing and was found to include no deaths or major morbidity.⁶⁸ By combining information from the history, physical examination, exercise tolerance, and stress of a proposed surgical procedure, inappropriate testing can be reduced, whereas, more importantly, appropriate screening tests will be performed.

Arriving at a diagnosis

During the history-taking portion of the preoperative evaluation the surgeon is developing a list of possible diagnoses. By examining the patient he or she then proceeds to further narrow the possible diagnoses and solidify clinical impressions developed during the history. Laboratory data and imaging may further clarify the diagnosis or diagnoses. Once the clinician has obtained enough information a treatment plan can be developed. The patient is informed of the risks, alternatives, and possible benefits to a potential procedure.

Assessing anesthetic/surgical risk

Once the clinician has gathered information by interviewing and examining the patient, he or she can classify them according to the American Society of Anesthesiologists (ASA) Classification of Physical Status (Table 1.7). Patients with a lower ASA classification represent a lower surgical risk than do patients with severe systemic disease. This system is commonly used and is helpful in identifying risk factors so that modifications in the treatment plan can be undertaken. The surgical procedure influences the scope of preoperative evaluation required by determining the potential range of physiologic flux during the perioperative period. The AHA/ACC guidelines describe risk stratification for non-cardiac surgery.⁶⁹ These can be divided into low, intermediate, and high with a reported risk of cardiac death and non-fatal myocardial infarction of <1, <5, and >5% respectively. Head and neck surgeries are considered intermediate-risk procedures. Examples of low-risk procedures would be cataract surgery and superficial procedures, whereas high-risk procedures would be surgeries such as emergency major operation or anticipated prolonged surgical procedures associated with large fluid shifts and/or blood loss.

Office vs inpatient

Once the clinician has gathered pertinent information during the preoperative work-up, he must decide where best to perform the surgical procedure.

Safety continues to be the guiding factor in deciding where various types of procedures should be per-

formed. Options available include office surgery, ambulatory surgery centers, and traditional hospital-based locations. Many variables are considered when deciding on whether to perform a surgery in the office or perform the surgery elsewhere.

Oral and maxillofacial surgeons routinely perform a large number and variety of procedures in the office setting. There the surgeon can perform the procedures under local anesthetic, intravenous (IV) sedation or general anesthetic. Oral and maxillofacial surgeons have developed an excellent track record with a long history of patient safety. Perrott *et al.* looked at outcomes of office-based ambulatory anesthesia by oral and maxillofacial surgeons.⁷⁰ The study involved 34191 patients, of whom 71.9% received deep sedation or general anesthesia, 15.5% received conscious sedation, and 12.6% received local anesthetic. The complication rate was 1.3% and almost all complications were minor and self-limiting. Only two patients had complications requiring hospitalization. In fact, oral and maxillofacial surgeons have been performing surgeries in an office setting much longer than other specialties. Their training and ability qualifies them to manage a patient's airway as well as any emergencies, thus minimizing surgical risk.

The type of procedure is an important determinant of where the surgery should be performed. For local bone graft harvest, the invasiveness of the surgery is similar to surgically removing impacted third molar teeth, and the risk to patients is low. Harvesting bone from the posterior iliac crest and cranium, on the other hand, is more invasive, and although the risk of major complications is low, the risk is higher than for local graft harvest. These complications include bleeding from the surgical site and postoperative infection. It is important when performing these types of procedures in the office or ambulatory surgery center to avoid compromising the sterility of the procedure.

Patient factors should also be an important part of the decision on where to perform the procedure. Patients with poorly controlled medical conditions such as morbid obesity or poorly controlled hypertension should be carefully evaluated, and appropriate preoperative testing should be performed to determine their surgical risk. Patient factors such as increased age, an operating time longer than 120 minutes, cardiac diagnoses, peripheral vascular disease, cerebrovascular disease, malignancy, and immunodeficiency can place patients at higher risk for immediate hospital admission.⁷¹

Advantages of performing surgery in a hospital setting include the addition of another health care provider to administer anesthetic during the surgical procedure. Imaging techniques such as ultrasonography, CT, and chest radiographs are readily available as are blood chemistries to rapidly diagnose and treat complications. Also procedures such as interventional radiology, for such things as embolization, are available. Ultimately the decision on where to perform a

surgery depends on both the surgeon and informed patient considering the type and length of the procedure, patient health factors, and safety.

Summary

The process of preoperative evaluation is essential in assessing the medical condition of patients, evaluating their overall health status, determining risk factors, and educating them. The goal of preoperative evaluation is to reduce patient risk and the morbidity of surgery.

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Chapter 2

Radiographic Imaging in Oral and Maxillofacial Surgery

Arne Petersson

This chapter covers current imaging modalities for the maxillofacial region. Although plain film radiography is covered, the majority of the chapter is devoted to the use of computed tomography (both conventional and cone beam CT), magnetic resonance imaging, and positron emission tomography (PET). The most appropriate imaging technique for impacted teeth, inflammatory lesions, cysts and benign tumors, and malignant tumors is discussed.

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Introduction

The most common radiographic examinations of oral and maxillofacial surgery patients are intraoral and panoramic radiographs. However, today computed tomography (CT) and magnetic resonance imaging (MRI) are common examinations in imaging of many different conditions. A useful investigation is one in which the result – positive or negative – will alter management or add confidence to the clinician's diagnosis.¹ It is sometimes difficult to decide which examination technique(s) should be used and it is often helpful to discuss this with a specialist in oral and maxillofacial radiology before deciding the investigation. The radiological examination must be based on the clinical information and the specific questions that should be answered. It is also important to try to minimize the radiation dose to the patient and especially CT can potentially give very

high absorbed doses to the patient. The trend today is to use a low-dose technique for CT, but this can be at the expense of the image quality and its use depends on the clinical problem.

Computed tomography (CT)

CT is a digital technique providing images of thin slices of the patient with a variable thickness. The slice thickness can be less than 1 mm by use of very small X-ray detectors and a fan-shaped X-ray beam transmitted through the patient. By simultaneously scanning several slices of the body (multislice CT), the scan time can be reduced significantly and the smallest details can be imaged within short scan times. Multislice CT enables a wide range of clinical applications and, through the use of computer software, three-dimensional (3D) images can be produced. Images can be viewed in the axial, coronal, or sagittal planes depending on the diagnostic task. This is referred to as multiplanar reformatted (MPR) imaging. Images can also be viewed in any other plane decided by the operator. CT has the advantage over other radiographic techniques that it eliminates superimposition of images of structures outside the area of interest. It has an inherent high-contrast resolution and differences between tissues that differ in physical density by less than 1% can be distinguished. For image display, each pixel is assigned a CT number (Hounsfield units – HU) representing density. The density of air is defined as –1000HU, water as 0HU and bone tissue has more than +400HU. To allow the observer to interpret the image, only a limited number of HU are displayed. A clinically useful gray scale is achieved by setting the window level and window width on the computer console to a suitable range of HU, depending on the tissue being studied. The term 'window level' represents the central HU of all the numbers within the window width. The window width covers the HU of all the tissues of interest and these are displayed as various shades of gray.

Cone-beam computed tomography (cone-beam CT)

This technique has been commercially available for the last few years. Cone-beam CT is based on volumetric tomography, in contrast to conventional fan-beam CT where slices are scanned. From this volume slices can be reconstructed in various planes. One advantage with cone-beam CT compared to conventional CT is the lower radiation dose. The radiation dose is reduced by up to 98% compared with conventional CT² and is comparable to 3–28 average panoramic radiographs.³ The dose varies substantially, however, depending on the device, imaging field and selected technique factors.³ The scan time is relatively short (around 20s) and the resolution is high (i.e. 0.125mm in Accuitomo®, Morita). The software is usually adapted to maxillofacial imaging and is real-time interactive, for example for implant planning. In both cone-beam CT and conventional CT artifacts are produced by metal objects and it is important to try to avoid exposing metal fillings and crowns.

Magnetic resonance imaging (MRI)

MRI does not use ionizing radiation. There are, however, some contraindications as the presence of ferromagnetic metals is a potential hazard. Patients with metallic foreign objects, pacemakers, and metal clips must not be examined. Pregnancy is a relative contraindication. The advantage of MRI is that it offers the best resolution of tissues of low inherent contrast and it has an excellent soft tissue contrast resolution. Disadvantages are relatively long imaging times and patients suffering from claustrophobia cannot be examined. MRI physics is very complicated and an understanding of the basic concepts is important in order to manipulate the scan parameters to improve the quality of the images. All imaging should be discussed with the radiology department to get optimal information.

Impacted teeth

A preoperative examination of an impacted tooth usually consists of two intraoral radiographs exposed at different angles (Fig. 2.1) or a panoramic radiograph. Using intraoral films in three different projections gave an insight into the true anatomy of third molars when the radiographic appearance was compared to the clinical observation.⁴ Intraoral and panoramic radiographs are usually sufficient to show the relationship between the roots of the third molar and the mandibular canal. However, narrowing of the canal, increased radiolucency (“dark band”) and interruption of the radiopaque border of the mandibular canal can justify a CT examination.^{5–7} Recently, cone-beam CT has been shown to have a high diagnostic accuracy in predicting neurovascular bundle

exposure during extraction of impacted mandibular third molars.⁸ Fig. 2.1 shows an example of an impacted mandibular third molar with a complicated root anatomy examined with cone-beam CT.

CT is also valuable when examining impacted teeth in other regions.⁹ Cone-beam CT has been shown to be indicated for localization of impacted maxillary canines¹⁰ and conventional CT demonstrated root resorption better of the adjacent incisors compared to conventional radiography.^{11,12} Fig. 2.2 shows a cone-beam CT examination of a non-erupted maxillary canine causing resorption of the root of the lateral incisor.

Pathological conditions – inflammatory lesions, cysts, benign and malignant tumors

The aims of the radiographic examination are to give information that leads to the most probable diagnosis and to the best treatment. The examination must cover the whole pathological area in at least two dimensions. A combination of different radiographic techniques can lead to a more certain diagnosis, but it should always be kept in mind that the treatment also must be affected positively by the extended examinations in order to be justified. There are some radiological signs that always must be looked for when interpreting radiographs of pathological conditions, such as:

- location and size;
- periphery and shape;
- internal structures;
- effects on surrounding structures.

Inflammatory lesions

There are several lesions that present as a radiolucent area in the jaws. Inflammatory lesions located in the periapical area are by far the most common changes. It is not possible to differentiate radiologically between a radicular cyst and apical periodontitis. Radicular cysts tend to be larger than periapical granulomas, but a large variation in size has been shown for both types of lesions.¹³ This is not a diagnostic problem for small periapical radiolucencies as endodontic treatment has a high success rate¹⁴ and the prevalence of true cystic lesions is low.¹⁵ At follow-up after endodontic treatment and periapical surgery it is important to standardize the radiographic examination regarding projection and density/contrast of the image to be able to compare different examinations. An example is given in Fig. 2.3 where the projection was changed between the two radiographs taken on the same occasion and it appears that the size of the periapical bone destruction has changed.

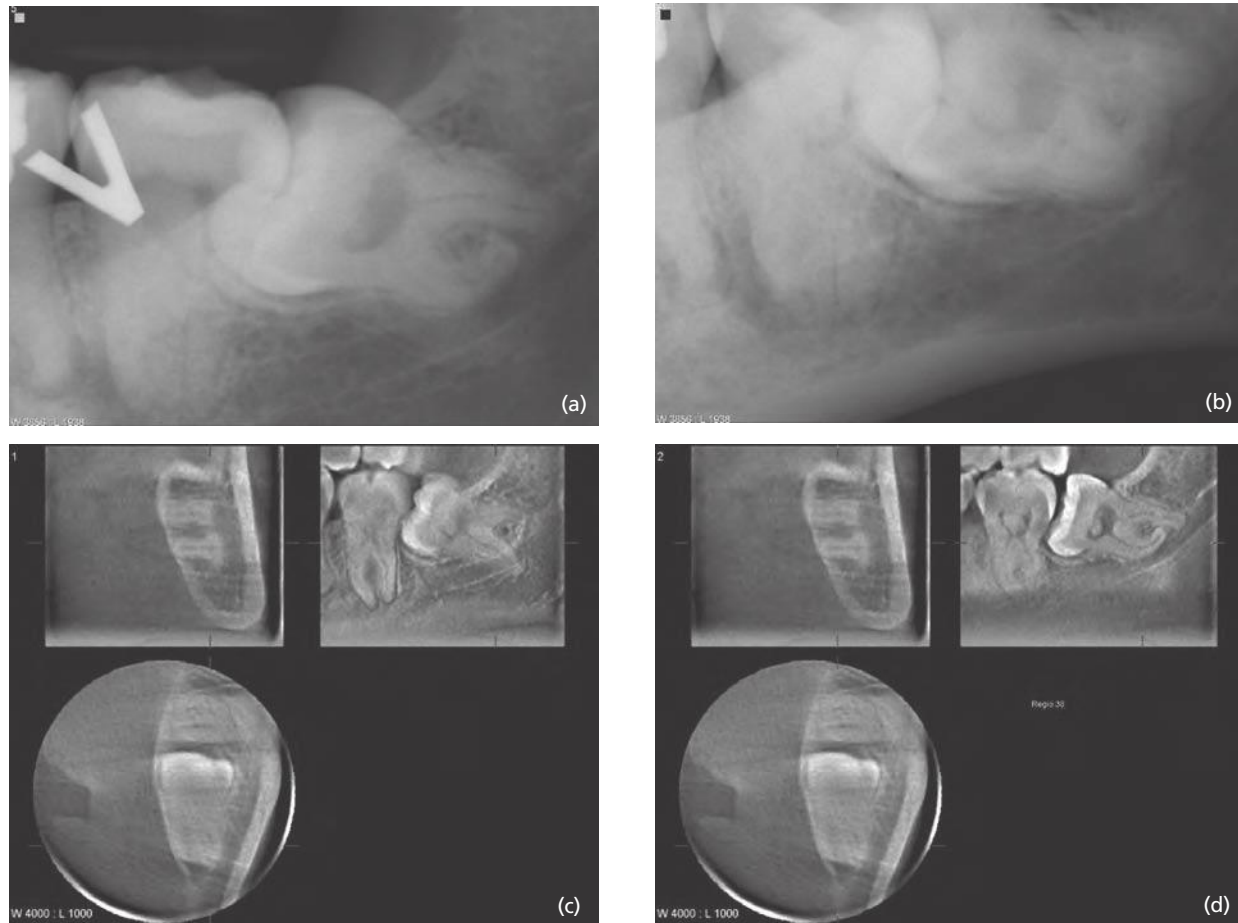


Fig. 2.1 Impacted lower third molar in a mesioangular position. (a) Periapical radiograph taken with +10° vertical angulation of the X-ray tube. Two mesial roots (one straight and one curved) and one distal root are seen. (b) Periapical radiograph taken with -10° vertical angulation of the X-ray tube. The mandibular canal seems to be buccal to the curved root. (c) Cone-beam CT with 1 mm thick sections. Upper left image is a cross-section of the mandible through the roots. The mandibular canal is seen below the mesial roots, lingual to the buccal root and buccal to the lingual root. Upper right image is a sagittal view of the buccal part of the tooth and the straight mesio-buccal root is shown. Lower section shows an axial view of the tooth, which is situated close to the lingual compact bone. (d) Same as (c), but the section is lingually placed. The curved mesio-lingual root is shown in the sagittal view.

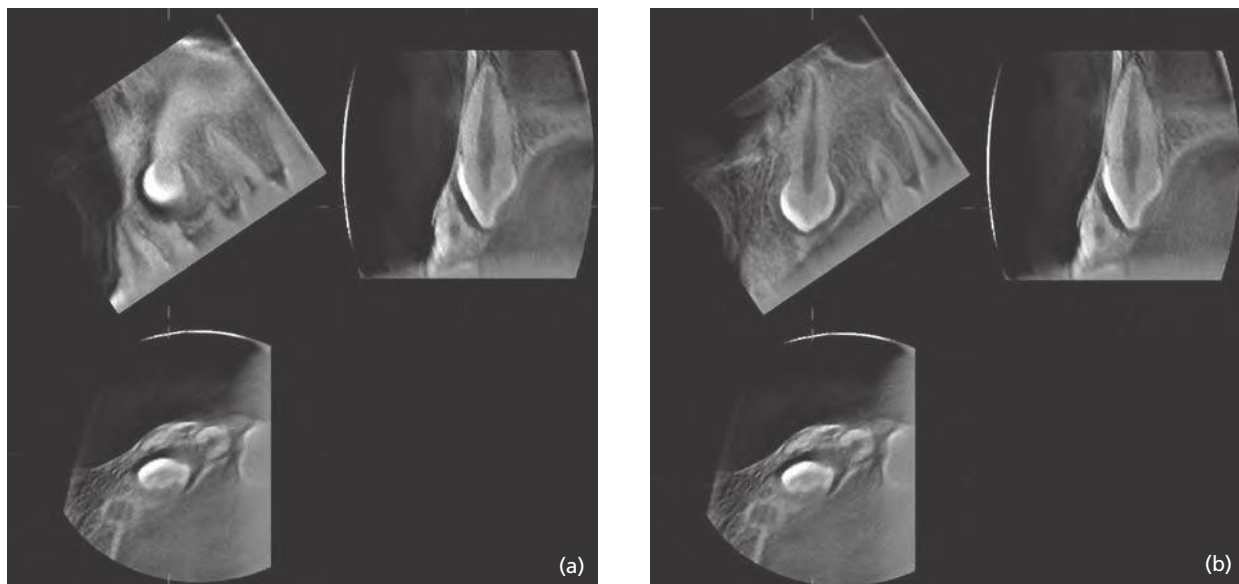


Fig. 2.2 Cone-beam CT examination of a non-erupted maxillary canine causing resorption of the root of the lateral incisor. The crown of the canine is situated palatal to the root of the incisor. (a) Upper left, sagittal view. Upper right, cross-section of the jaw. Lower, axial view. (b) Same as (a), but the 1 mm thick section is placed more palatal in the sagittal section. The root tip of the canine is curved mesially.

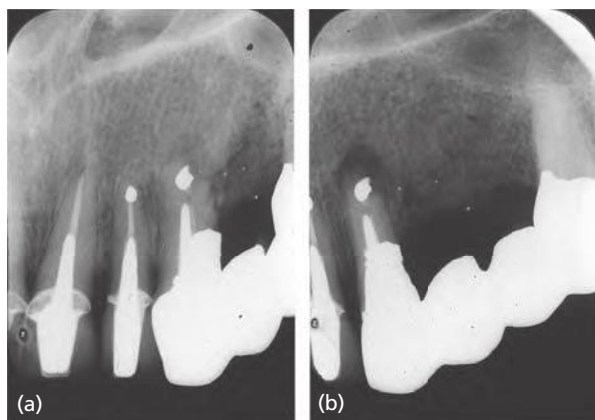


Fig. 2.3 Evaluation of healing after apical surgery of the upper left canine. Radiographs in (a) and (b) are taken at the same occasion, but with different projections. The apical bone destruction has a different appearance depending on the change of projection

Cone-beam CT should be considered when no detectable pathology is found in periapical radiographs and clinical tests indicate pathology, as more periapical lesions are found with cone-beam CT.^{16,17} This is especially important in patients with chronic maxillary sinusitis, as a dental cause was found in 40% of patients with chronic maxillary sinusitis.¹⁸

Radiographs of the paranasal sinuses are not indicated routinely when sinusitis is suspected. Panoramic radiography should not be used for the detection of small osteolytic lesions in the maxillary sinus¹⁹ and soft tissue changes can be difficult to detect in panoramic radiographs depending on whether the surrounding structures are projected into the maxillary sinuses. CT is more rewarding than conventional radiography in the examination of the paranasal sinuses.²⁰ Low-dose high-resolution CT is recommended,²¹ when medical treatment has failed, when complications arise or if malignancy is suspected.¹ Fig. 2.4 shows a case of chronic maxillary sinusitis examined with panoramic radiography and low-dose CT.

Osteomyelitis, osteonecrosis, and osteoradionecrosis are conditions that have similarities in their radiographic appearance. Osteomyelitis is an inflammatory condition of bone that involves the medullary cavity and has a tendency to progress along this space and involve the adjacent cortex, periosteum and soft tissue.²² Osteonecrosis of the jaws is a condition that shows as a painful bone exposure, often after bisphosphonate treatment.²³ Another form of osteonecrosis is the feared complication of radiation therapy (osteoradionecrosis).

In a review article of diagnostic modalities of skeletal infections, plain films are recommended as the first step in the imaging assessment of osteomyelitis, but the sensitivity of plain films is low.²⁴ CT provides more diagnostic information than plain films about bone changes and also shows soft tissue changes. Bone scintigraphy has a higher sensitivity than radi-

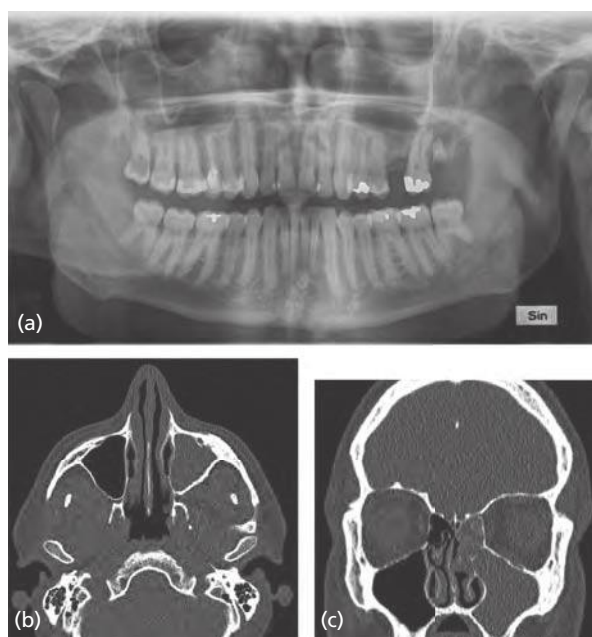


Fig. 2.4 A patient with chronic maxillary sinusitis. Panoramic radiography (a) and low-dose CT (b, c) were performed. (a) Panoramic radiograph showing root fragments of the upper left third molar with periapical bone destruction. The maxillary sinuses are difficult to evaluate. (b) Axial CT showing complete radiopacification of the left maxillary sinus. The bony walls are slightly sclerotic. (c) Coronal reconstruction showing the same picture as (b).

ography and changes may be visualized in early phases.²⁴ However, MRI has also proven to have a high sensitivity in the early detection of osteomyelitis.²⁴ In the jaws osteomyelitis is more common in the mandible and can be difficult to diagnose. Radiographically it is most important to confirm the clinical suspicion and to localize the condition. It is also important to examine its extent and to assess the response after treatment. In the mandible the extent of inflammation was better appreciated with CT than with conventional radiography, especially in cases with diffuse changes such as cortical plate disruption and periosteal reaction.²⁵ Fig. 2.5 shows a child with chronic osteomyelitis illustrating the superiority of CT over panoramic radiography in showing bony involvement. Four radiographic patterns have been identified from computed tomograms: the most common was a mixed pattern with osteosclerosis and osteolysis, the second most common was sclerotic pattern, the third most common was a lytic pattern, and finally the least common was sequestration with or without other bony changes.²⁵ Periosteal reaction was found in 40% in cases of osteomyelitis, especially in younger patients.²⁶ Cone-beam CT offers a good alternative to conventional CT in examining osteomyelitis of the mandible.²² MRI using a combination of T1- (low-intermediate signal) and T2-weighted (high-intermediate signal) images showed larger areas of abnormality than plain radiography or CT.²⁷ A combination of T1-weighted and so-called STIR

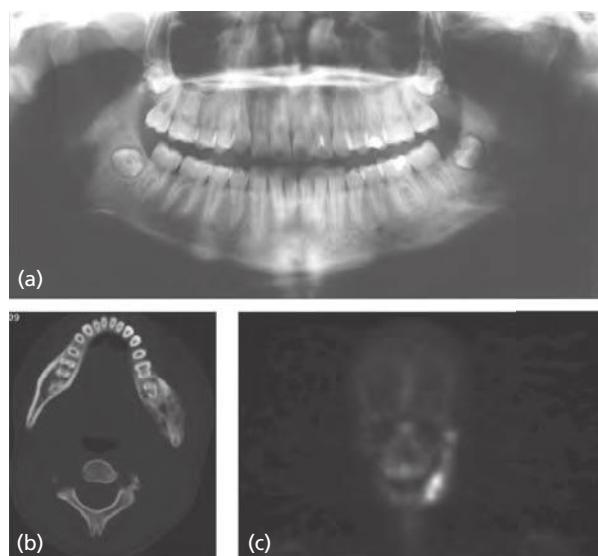


Fig. 2.5 13-year old girl with pain and swelling of the left mandible. The diagnosis was chronic osteomyelitis. (a) Panoramic radiograph showing sclerosis, thick periosteal reaction, and destruction of the cortical delineation of the mandibular canal and tooth bud of the third molar on the left side. (b) CT showing the expansion of the mandible and bone destructions more clearly than panoramic radiography. (c) Bone scan using ^{99m}Tc methylene diphosphonate. An area of high intensity of uptake is shown in the left mandible. The uptake is extending up in the condyle.

images is also useful for the detection of mandibular osteomyelitis.²⁸

Head and neck cancers are sensitive to radiation. Radiotherapy alone, or combined with surgical resection is part of the treatment and especially the mandible may be included in the radiation field.²⁹ Osteoradionecrosis can be defined as radiological evidence of bone necrosis within the radiation field, where tumor recurrence has been excluded.²⁹ It has been suggested that microorganisms play a very minor role in the pathophysiology of osteoradionecrosis of the jaws, differentiating it from osteomyelitis.³⁰ The radiological findings are cortical thinning and destruction, sclerosis, sequestration, and loss of cancellous bone.³¹ CT is superior to panoramic radiography in visualizing the features and extent of osteoradionecrosis of the mandible.³¹ Fig. 2.6 illustrates a patient with osteoradionecrosis developing several years after radiation therapy. Contrast-enhanced MRI can be used to better show the bone marrow involvement as marked contrast enhancement of the osteoradionecrotic bone marrow is shown.³²

Cysts and benign tumors

Radicular cysts are the most common cysts found in the jaws, followed by dentigerous cysts and odontogenic keratocysts.³³ Large cysts in the region of the maxillary sinus may be difficult to image with conventional radiographs and CT is usually indicated to see the extension of the cyst (Fig. 2.7). Dentigerous

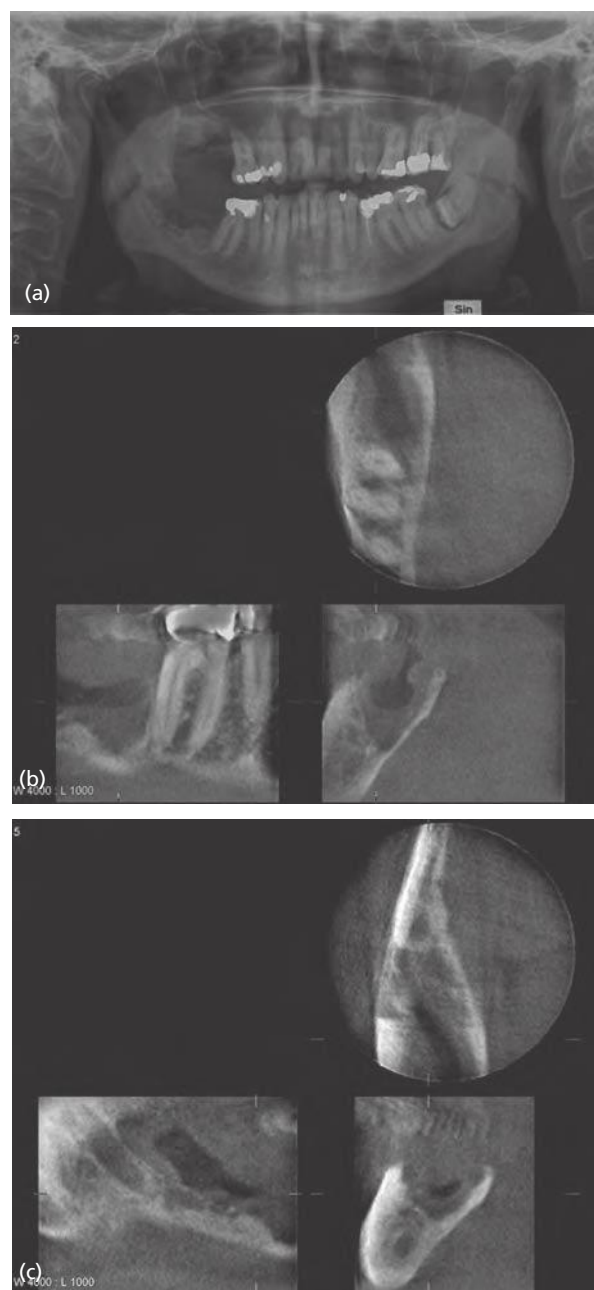


Fig. 2.6 The patient was treated with radiation therapy 9 years ago for a cancer in the tonsil region. He now has pain in the right side of the mandible. (a) Panoramic radiograph showing bone destruction in the right mandibular molar region and mandibular ramus. (b, c) Cone-beam CT shows the large extent of the bone destruction in a bucco-lingual direction.

cysts and keratocysts are usually incidental findings in panoramic radiographs, with keratocysts predominantly found in the mandible (Fig. 2.8). In the most recent WHO classification, the odontogenic keratocyst has been identified as an odontogenic tumor and it was named keratocystic odontogenic tumor.³⁴ CT may be indicated to locate the relation of the mandibular canal to the cyst and teeth (Fig. 2.9). Benign tumors are relatively uncommon lesions in the oral and maxillofacial region. There is, however, a significant geographical difference in prevalence, e.g. the ameloblastoma occurs most frequently in all Asian,

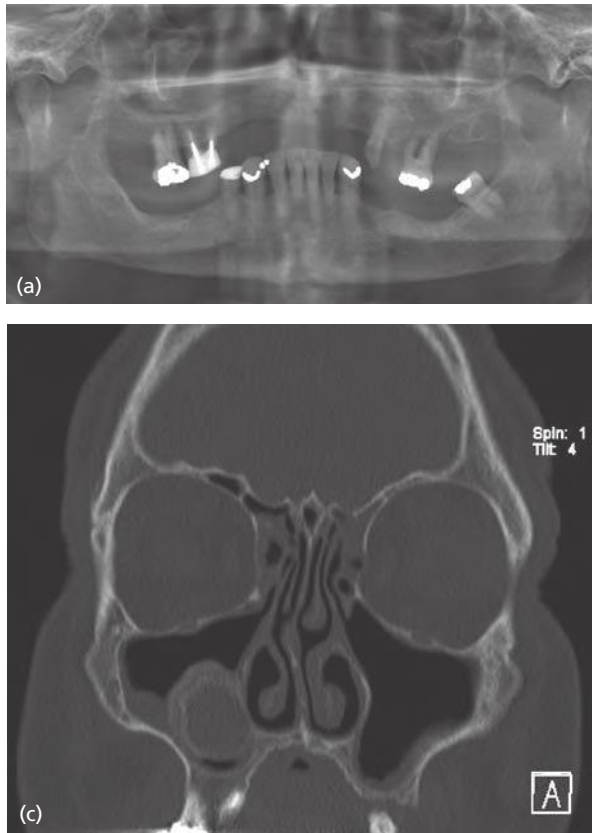


Fig. 2.7 Patient with a fistula in the maxillary right canine region. Buccal swelling and symptoms of sinusitis. He mentions that a tooth was extracted in the region about 10 years ago when he had similar symptoms. The final diagnosis was proved to be residual cyst. (a) Panoramic radiograph which is difficult to interpret. (b) CT with an axial section showing well defined bone destruction in the right maxillary canine region. (c) Coronal section showing the cystic lesion with thickened bone around the cyst. Soft tissue swellings are seen in the maxillary sinus. (d) Axial CT taken 10 years earlier, when the patient had symptoms of sinusitis. A cystic lesion is seen around the root tip of the right maxillary canine. The tooth was later extracted.



African and South American countries.³⁵ Sometimes it is difficult to differentiate cysts and tumors on the basis of conventional radiographs. MRI could be helpful to differentiate between cysts and tumors.³⁶

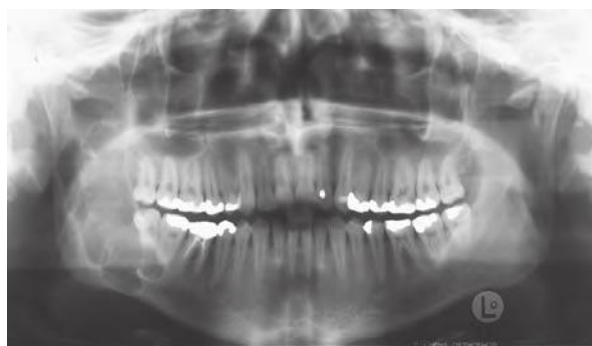


Fig. 2.8 Panoramic radiograph showing a multilocular bone destruction in the right mandibular ramus area. The patient had no symptoms and the cyst was detected in bitewing radiographs taken by his dentist. The tentative radiological diagnoses were ameloblastoma or odontogenic keratocyst. The diagnosis from the pathologist's report was odontogenic keratocyst.

Malignant tumors

Primary malignant bone tumors are uncommon in the jaws. Squamous cell carcinoma is the most common head and neck cancer and it may invade the underlying bone. The typical appearance on a panoramic radiograph of a malignant lesion involving the jaw bone is bone destruction with a border that is ill defined. Fig. 2.10 shows an example of a carcinoma of the maxillary sinus involving the upper jaw detected on a panoramic radiograph. Rapidly growing malignant lesions destroy the alveolar bone and usually no root resorption is present. A typical sign is that the teeth may appear to be floating in space: "floating teeth". Some malignant tumors such as osteosarcomas and chondrosarcomas can produce hard tissue with irregular margins. The posterior part

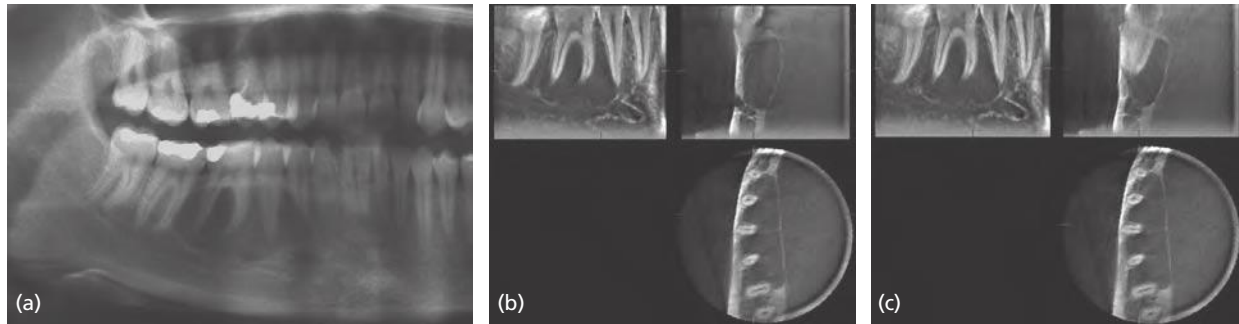


Fig. 2.9 A cystic lesion that at surgery was diagnosed as a traumatic bone cyst. (a) Panoramic radiograph showing the cystic lesion in the right mandibular premolar and first molar regions. (b, c) Cone-beam CT was performed to locate the mandibular canal. The lingual cortical bone is thin. In the anterior section the mental foramen and mandibular canal are visualized.

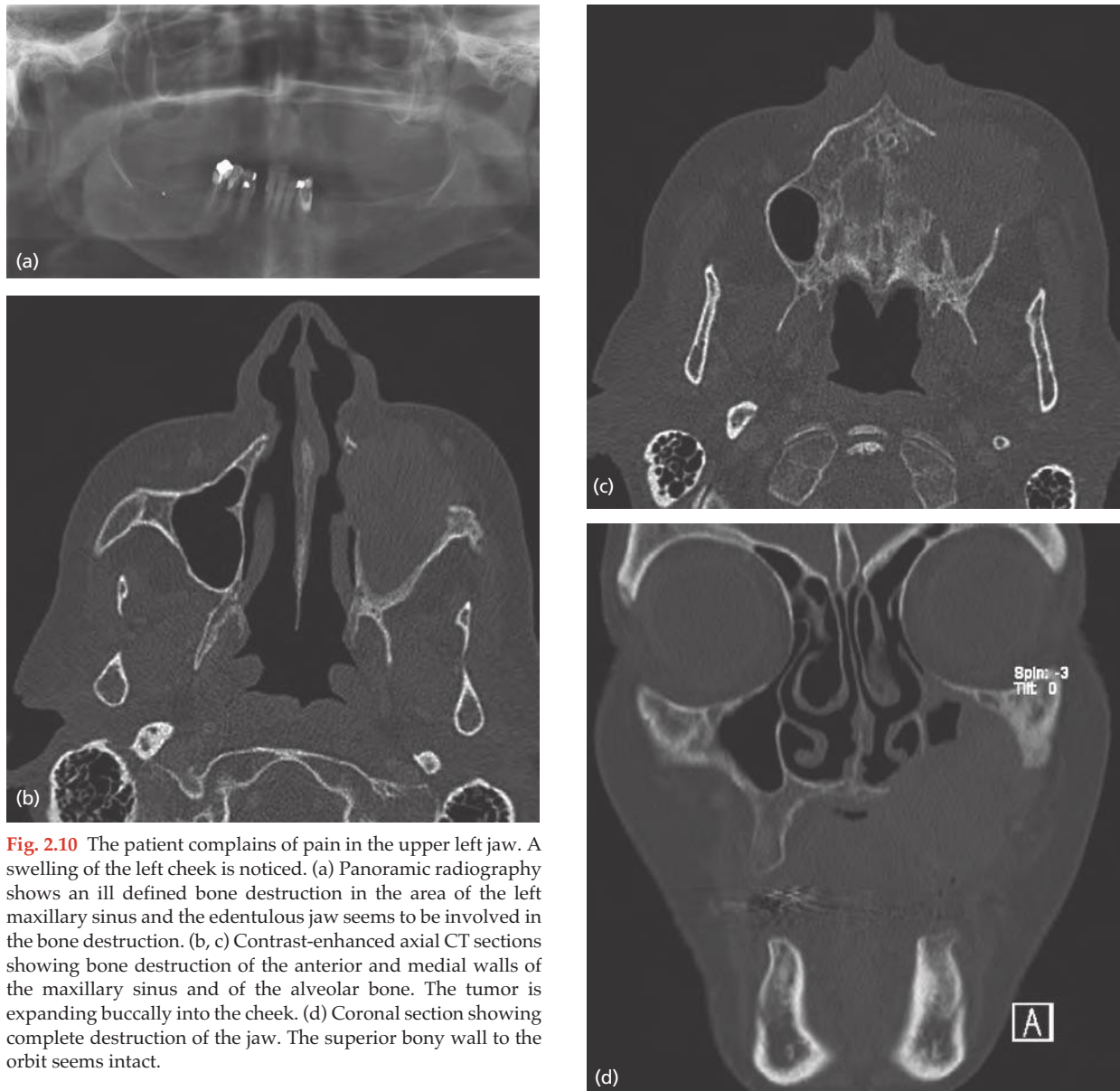


Fig. 2.10 The patient complains of pain in the upper left jaw. A swelling of the left cheek is noticed. (a) Panoramic radiography shows an ill defined bone destruction in the area of the left maxillary sinus and the edentulous jaw seems to be involved in the bone destruction. (b, c) Contrast-enhanced axial CT sections showing bone destruction of the anterior and medial walls of the maxillary sinus and of the alveolar bone. The tumor is expanding buccally into the cheek. (d) Coronal section showing complete destruction of the jaw. The superior bony wall to the orbit seems intact.

of the mandible is the most common location of metastatic tumors in the jaws and for females the most common site of origin is the breast and for men the prostate.³⁷

The radiographic examination of malignant tumors often comprises CT and MRI to determine the extent of the tumor and to evaluate cervical lymphadenopa-

thy.³⁸ Post-treatment examinations are usually performed to evaluate the effect of treatment. Recently, a combination of CT and positron emission tomography (PET) has been introduced and PET/CT is now widely used as an advanced clinical tool for the diagnosis, staging, and restaging of cancer, and for the assessment of tumor therapy.³⁹

Temporomandibular joint (TMJ)

The majority of patients with temporomandibular disorders (TMD) achieve good relief of symptoms after conservative therapy.⁴⁰ Imaging of TMD patients plays a minor role in the management of these patients as it has been shown that the treatment outcome is not affected by the radiological findings.⁴¹ Despite the success of conservative care, some patients do not improve and TMJ surgery may be indicated.⁴² In these cases radiography is indicated, as well as in patients with trauma, tumors, ankylosis and developmental anomalies. Further, radiographic examination of patients with polyarthritic conditions, such as rheumatoid arthritis, can be recommended to evaluate the degree of joint destruction.⁴³ Bone scanning with ^{99m}technetium phosphate might be indicated to determine the level of growth activity in condylar hyperplasia.⁴⁴

There are different techniques for imaging the TMJ: panoramic radiography, plain radiography, conventional and computed tomography, arthrography, and MRI. Panoramic radiography is not a reliable method of accurately showing the shape of the mandibular condyle⁴⁵ and the temporal component is poorly visualized. Plain radiography of the TMJ depicts the mineralized part of the joint, but superimposition of adjacent anatomic structures can make interpretation difficult. Conventional tomography improves the depiction of the bone structures.⁴⁶ However, minor bony changes will not be shown in conventional tomography.⁴⁷ A conventional tomo-

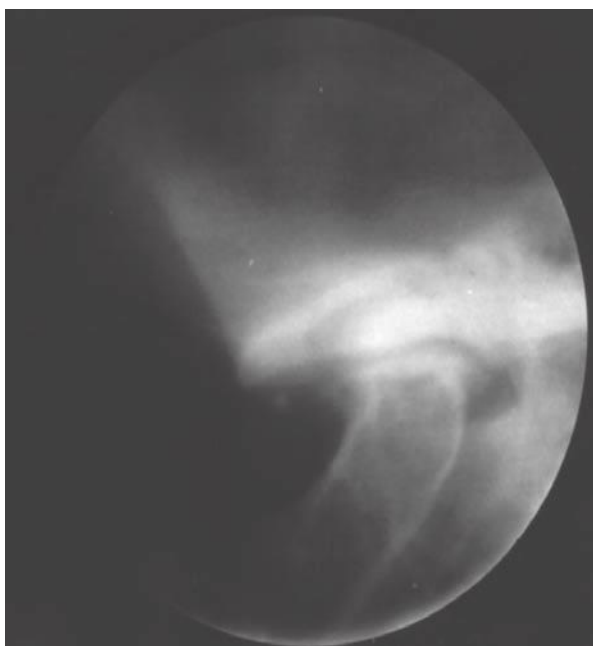


Fig. 2.11 The patient has pain and crepitation from the left TMJ. The sagittal tomogram obtained with hypocycloidal tomography shows flattening, sclerosis and an anterior osteophyte of the condyle. The articular eminence is also flattened. The diagnosis is osteoarthritis.

gram from a patient with osteoarthritis is shown in Fig. 2.11. CT imaging provides exquisite detail for bony abnormalities, such as ankylosis, fractures (Fig. 2.12), osseous tumors and arthrosis⁴⁸ and 3D images can be produced; 3D reconstructions of a patient with condylar aplasia are shown in Fig. 2.13.

MRI has gradually replaced arthrography and information about disk position, joint fluid, bone marrow changes, and bone structure at multiple levels of the joint can be obtained from MRI. MRI is emerging as the prime diagnostic imaging technique in TMD patients.⁴⁸ The technique, however, is expensive and there are no studies showing when the results of the MRI examination will result in a better treatment outcome for the typical TMD patient.⁴⁹ Imaging of the TMJ is definitely indicated prior to TMJ surgery and the preferred method is MRI if the soft tissue should be shown and CT if the hard tissue is of prime interest. In cases of tumors the methods often are combined.

A classification system for disk displacement was developed by Tasaki *et al.*⁵⁰ Disk displacement presents as a spectrum of displacements in all directions with anterolateral and anterior disk displacement being the most common.⁵⁰ A combination of sagittal and coronal MR images will show anterior as well as sideways disk displacements.⁵¹ A systematic review of MRI of the TMJ concluded that the most commonly used MRI technique was a combination of sagittal and coronal images using proton density and T2 sequences.⁴⁹ Fig. 2.14 illustrates a patient with anterior disk displacement without reduction. The presence of joint effusion can be seen in the T2-weighted image in Fig. 2.14b, but the diagnostic value of this finding is not clear from the literature. Fig. 2.15 illustrates a case with changes in the bone marrow where both cone-beam CT and MRI have been used.

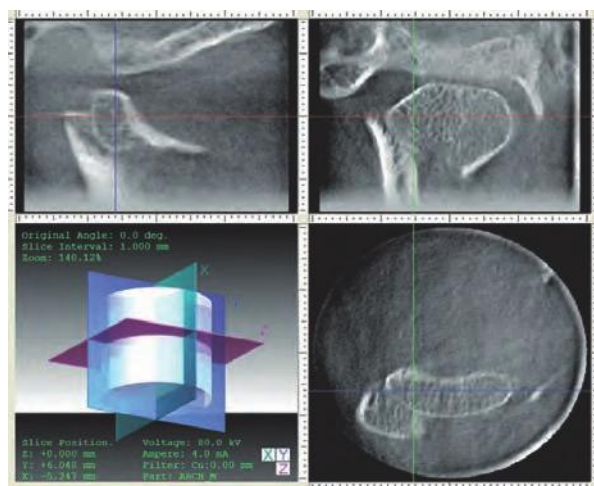


Fig. 2.12 Cone-beam CT of a fracture of the condylar neck after a bicycle accident. Upper left, sagittal view; upper right, coronal view; lower right, axial view. The condylar fragment has been dislocated medially and inferiorly. The images are taken with 3D Accuitomo (Morita corp.).

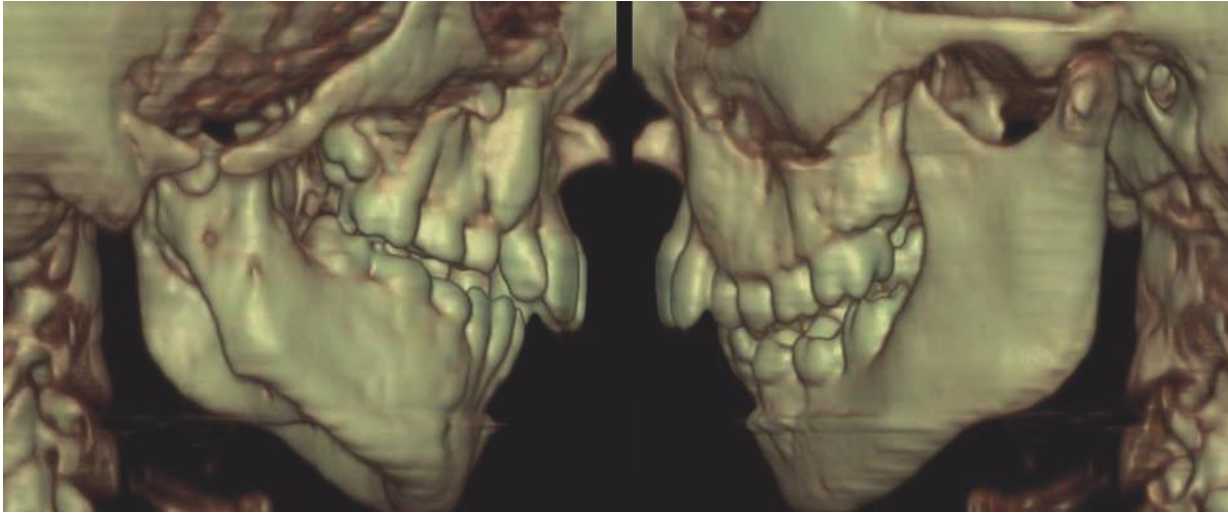


Fig. 2.13 CT 3D reconstructions of a patient with hemifacial microsomia and aplasia of the right condyle. The patient is missing the ear and a defective zygomatic arch is seen on the right side. The left side has developed normally.

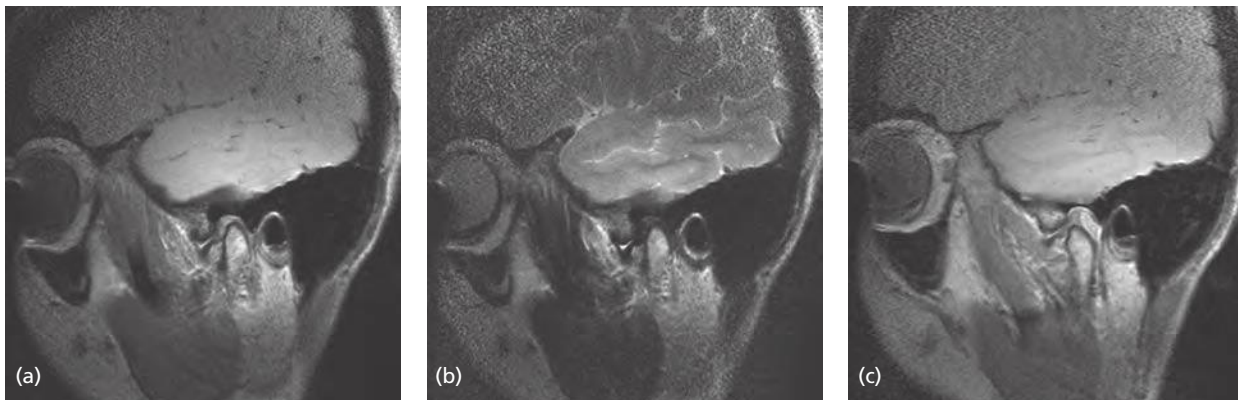


Fig. 2.14 The patient has pain from the right TMJ and difficulty in opening the mouth wide. (a) Proton density MR image taken at intercuspal position. The disk is anteriorly displaced. (b) T2-weighted image showing an area with bright signal in the upper joint space: joint fluid. (c) Maximum mouth opening. The disk is situated anterior of the condyle: anterior disk displacement without reduction.

Implant treatment

Panoramic radiography is the first choice for the radiological appraisal before implant treatment. The technique is, however, dependent on proper positioning of the patient during exposure and objects located outside the center of the sharply depicted plane are reproduced with distortions. Reliable measurements have been found for digital panoramic radiography,⁵² but both over-⁵³ and underestimation⁵⁴ of vertical linear measurements have been found in other studies of panoramic radiography. The inherent errors in panoramic radiography should always be kept in mind whenever an exact assessment of a distance is required. Panoramic radiography is inferior to conventional tomography and CT in visualization of the mandibular canal and in measurements related to the mandibular canal.^{55,56} For proper preoperative planning of oral implants, the need has increased for cross-sectional tomographic examinations.⁵⁷ The use of tomography increased the efficacy of periapical and panoramic images, with respect to the prediction of the appropriate implant size when planning single

tooth implants.⁵⁸ CT has today almost totally replaced conventional tomography in oral radiology, although conventional tomography still may be in use for cross-sectional imaging. There is a choice between multislice CT and cone-beam CT for preoperative examination of implant patients. Loubéle *et al.*⁵⁷ found no significant differences between multislice CT and cone-beam CT when measuring distances on dry skulls. Cone-beam CT should be preferred as this technique gives lower radiation dose compared to multislice CT. However, when a completely edentulous patient is examined several exposures with cone-beam CT with a narrow field size are necessary and in such cases multislice CT should be preferred (Fig. 2.16). CT is indicated whenever the bone volume must be evaluated accurately and when localizing the mandibular canal. Fig. 2.17 shows cone-beam CT of the mandibular canal. Another indication of CT is when evaluating bone grafting, as two-dimensional radiographs underestimate bone resorption.⁵⁹ Postoperative follow-up examinations after implant treatment are usually performed with intraoral radiographs taken in a standardized way.

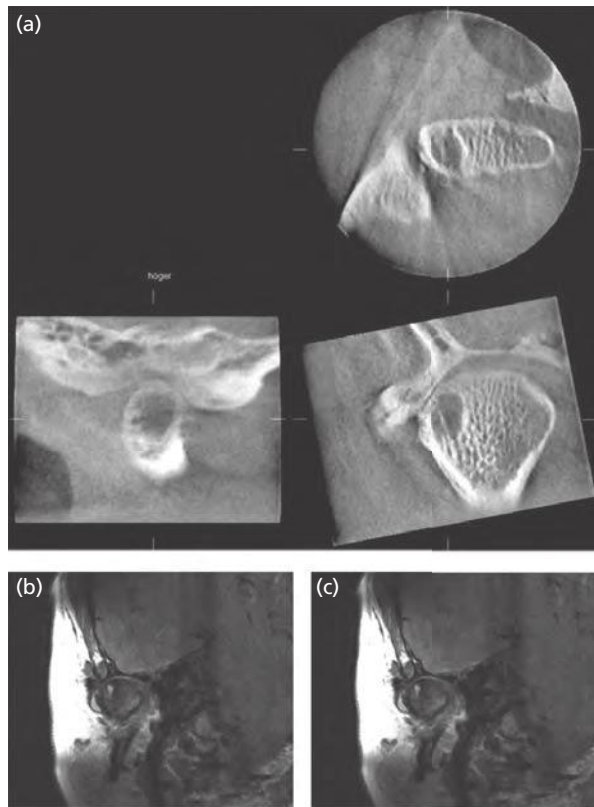


Fig. 2.15 Cone-beam CT and MRI of a patient with symptoms of osteoarthritis of the right TMJ. (a) Cone-beam CT showing a subchondral cyst in the lateral part of the condyle. Upper right, axial section; lower left, sagittal section; lower right, coronal section. (b) MRI, proton density coronal image showing the cyst with medium signal intensity. (c) T2-weighted coronal image showing the cyst with high signal intensity. Joint fluid (high signal intensity) is also seen in the lateral part of the lower joint compartment.

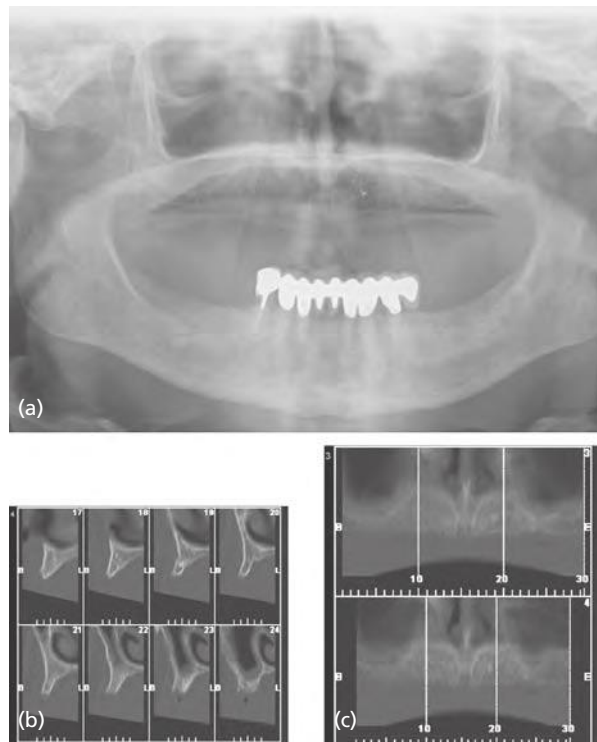


Fig. 2.16 Multislice CT examination for planning of implant treatment of an edentulous maxilla. (a) Panoramic radiograph. The bucco-palatal bone width of the maxilla was judged to be questionable. (b) 3 mm thick paraxial reconstructions made perpendicular to the alveolar bone (cross-sections) of the left side from the incisor to the premolar region. The images are produced in scale 1:1. B = buccal, L = lingual. (c) 3 mm thick panoramic reconstructions. The number of the vertical lines can be identified in the paraxial sections in order to locate the section.

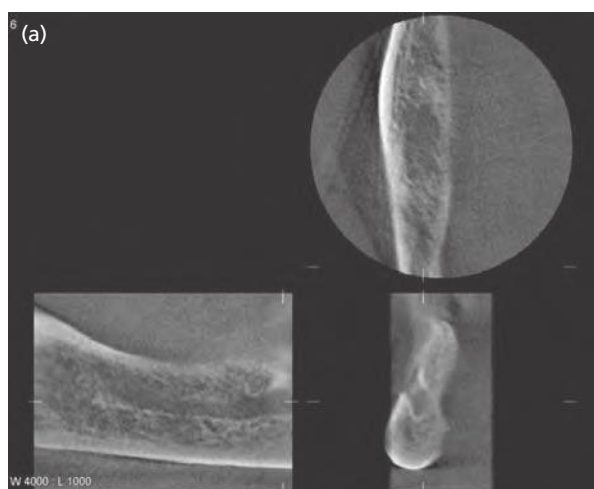


Fig. 2.17 Cone-beam CT of the mandible to visualize the mandibular canal. Images are produced in scale 1:1. (a) Anterior section showing the mental foramen (coronal section, lower right). (b) Posterior section in the molar region showing the mandibular canal (coronal section, lower right).

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Chapter 3

Medical Aspects – High-risk Patients

Earl G. Freymiller

Underlying medical conditions can place patients at increased risk during surgery. This chapter covers several high-risk diseases commonly found in patients undergoing oral and maxillofacial surgery. Divided into major organ systems, each system will discuss one or two specific diseases with oral and maxillofacial surgical implications. Some diseases increase the risk of surgical complications (e.g. bleeding in von Willebrand's disease, wound infection in immunocompromised patients). For other diseases, the surgery itself can affect control of the underlying disease (e.g. surgical stress and its effect on coronary artery disease, diabetes mellitus, etc.).

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Cardiovascular system

Ischemic heart disease

In ischemic heart disease, the mismatch between myocardial oxygen supply and demand places patients at increased risk when undergoing oral and maxillofacial surgery. During an anginal attack, the myocardial ischemia causes a temporary decrease in the effectiveness of the cardiac pump, in addition to placing the patient at risk for the initiation of a cardiac arrhythmia. More severe hypoxia resulting in myocardial infarction causes necrosis of part of the myocardium which can produce a permanent decrease in cardiac function, initiate congestive heart failure, trigger arrhythmias, or cause death.

The main risk factors for coronary artery disease are hypertension, smoking, diabetes mellitus, dyslipidemia, family history, and obesity. Patients with no prior history of ischemic heart disease but with several risk factors should be considered to have ischemic

heart disease until proven otherwise. Medical treatment of patients with coronary artery disease includes changes in lifestyle (weight loss, smoking cessation, exercise, etc.) and controlling other medical risk factors (diabetes, hypertension, dyslipidemia). Medical therapy includes aspirin to help decrease platelet adhesion and limit thrombus formation, beta-blockers and/or calcium channel blockers to help control the pressure against which the heart must contract and control heart rate, statins to reduce cholesterol, nitrates, and angiotensin converting enzyme (ACE) inhibitors. Surgical treatment is aimed at revascularizing the ischemic heart through percutaneous cardiac angioplasty with stent placement, or coronary artery bypass grafting.

Preoperative evaluation of the patient with ischemic heart disease is aimed at determining the severity of the underlying disease, thus determining the amount of cardiac reserve which directly relates to surgical risk. Much information can be obtained from the medical history, by determining the patient's

ability to tolerate cardiac stress during an exercise like climbing stairs. Specific cardiac tests augment this information. A preoperative electrocardiogram (EKG) may be of some use (e.g. T waves indicative of prior myocardial infarction). However, the resting EKG can be completely normal between ischemic events. Thus, a stress EKG gives more information. If the patient cannot exercise, pharmacological stress can be placed on the heart (e.g. dobutamine). Nuclear imaging during stress using thallium and technetium will aid in localizing and determining the extent of ischemic areas, thus increasing the diagnostic sensitivity. Similarly, a stress echocardiogram can detect abnormal wall motion during ischemia, and can also be used to evaluate the extent of any underlying valvular heart disease. Cardiac angiography, although more invasive, identifies the specific location and severity of atherosclerotic plaques restricting blood flow within the coronary arteries.

During oral and maxillofacial surgery and in the immediate postsurgical period, the goal is to prevent cardiac hypoxia. Patients with significant coronary artery disease may not tolerate a decrease in oxygen-carrying capacity; it is believed that the hemoglobin concentration should not be allowed to fall to a level that would be acceptable in an otherwise healthy patient, and thus there is a much lower threshold for transfusion in patients with coronary artery disease. Some studies have demonstrated that a low hemoglobin level increases the postsurgical mortality rate in patients with cardiac disease.¹ However, prospective, randomized clinical trials have demonstrated that restricting transfusions to maintain the hemoglobin concentration at 7–10 g/dl (as opposed to 10–12 g/dl) actually results in decreased mortality in some groups of critically ill patients (those less acutely ill and those less than 55 years of age).² For patients with clinically significant cardiac disease, no difference in mortality was seen when patients' hemoglobin levels were allowed to drop to the 7–10 g/dl range. Except for those patients recovering from an acute myocardial infarction or those with unstable angina, restricting transfusions may actually be beneficial.² Besides possible disease transmission or transfusion reactions, there are other concerns associated with transfusion. Immunomodulation resulting from allogeneic transfusions has been shown to increase the risk of postsurgical bacterial infections.³ Especially important to the oral and maxillofacial surgeon, some studies have implied that allogeneic transfusions increase the risk of tumor recurrence after surgery for head and neck cancers,⁴ and/or shorten the time to recurrence,⁵ also due to down-regulation of the immune system. All of these factors must be considered when deciding whether or not to transfuse the patient with ischemic heart disease.

Fluid overload which results in increased cardiac workload must be avoided in patients with ischemic heart disease. Consideration should also be given to

cardioprotective perioperative beta-blockade to reduce heart rate and control after-load, thus decreasing oxygen consumption by the heart. Perioperative beta-blockers are probably beneficial for high cardiac risk patients (more than one clinical risk factor), but their use is uncertain for those with a single risk factor.⁶ Unless significant bleeding will represent a major problem for surgery, many recommend continuing daily low-dose aspirin through the pre- and postsurgical periods, because aspirin withdrawal precedes up to 10.2% of acute cardiovascular events, with the mean time to occurrence of a cardiac event after aspirin withdrawal being just over a week (8.5 days).⁷

For an acute ischemic event in the postoperative period, patients should be treated with supplemental oxygen and nitrates. An EKG may demonstrate flipped T waves, the development of Q waves, and ST changes. Elevation of cardiac-specific markers such as creatinine kinase-MB and troponin indicate myocardial infarction. Pain control (morphine) and anti-platelet therapy (aspirin) should be administered. Consideration of low molecular weight heparin and possible early percutaneous transluminal coronary angioplasty (PTCA) or thrombolysis (tissue plasminogen activator, t-PA) is indicated. Beta-blockers in combination with calcium channel blockers should also be considered.

Valvular heart disease

The patient with valvular heart disease undergoing oral and maxillofacial surgery presents two main concerns. The first concern is for the decreased effectiveness of the cardiac pump. The second is for the patient's increased risk for developing bacterial endocarditis. Patients with valvular heart disease frequently present with a heart murmur graded from 1 to 6. However, the louder murmur is not always indicative of a more severe problem. Valvular heart disease is initially diagnosed with a stethoscope, by determining where on the chest wall the murmur is heard loudest, in which part of the cardiac cycle it occurs (systole or diastole), the quality of the murmur (e.g. crescendo-decrescendo), and where it radiates. However, the final diagnosis is almost always confirmed with an echocardiogram.

In mitral stenosis, the stenotic mitral valve reduces left ventricular filling so cardiac output is decreased and left atrial pressure is increased. The condition can worsen when the patient becomes tachycardic. Tachycardia decreases the time for left ventricular filling, and cardiac output can fall. Perioperative tachycardia should thus be avoided in patients with mitral stenosis. The resultant left atrial enlargement also predisposes the patient to develop atrial fibrillation. Atrial fibrillation with mitral stenosis requires anticoagulation therapy to reduce the risk of embolic stroke. With mitral regurgitation, the incompetent mitral valve also decreases cardiac output, as a por-

tion of blood flows back into the left atrium during systole. This can also result in an enlarged left atrium, and predisposes the patient to atrial fibrillation. Aortic stenosis is of special concern. The limitation to left ventricular outflow not only reduces cardiac output, but also causes significant left ventricular hypertrophy with a concomitant increase in myocardial oxygen demand. Frequently fatal if left untreated, aortic stenosis presents a significant surgical risk. With aortic insufficiency, systemic blood flows back into the left ventricle during diastole resulting in left ventricular dilation (but not necessarily hypertrophy) and an increase in stroke volume. Patients may present with an increased systolic pressure, but with retrograde regurgitant flow during diastole, the pressure rapidly drops and systemic diastolic pressure is low. Therefore, an enlarged pulse pressure is seen with bounding carotid pulses.

Treatment of valvular heart disease is directed toward controlling blood pressure and heart rate, and in treating any accompanying congestive heart failure (e.g. diuretics). Surgical treatment includes valvuloplasty (possibly via cardiac catheterization) or valve replacement with subsequent anticoagulation. The American Heart Association guidelines for prevention of bacterial endocarditis in patients with valvular heart disease now only include antibiotic prophylaxis for patients at the very highest risk. Patients recommended for coverage include those with prosthetic valves (or other prosthetic material used to repair a valve), patients with unrepaired cyanotic congenital heart defects (and those with residual defects after repair), those within 6 months of repair of a congenital heart defect, patients with a prior history of infective endocarditis, and those who develop valve defects after cardiac transplant.⁸

Congestive heart failure

Insufficient cardiac output results in increased back pressure with fluid overload in the pulmonic vascular system. Congestive heart failure has approximately a 50% 5-year mortality rate.⁹ Causes of congestive heart failure are multiple, and include long-standing hypertension, prior cardiac damage secondary to infarction, valvular heart disease, congenital heart diseases, etc. Congestive heart failure usually begins with left-sided failure. The pulmonary fluid overload causes rales, dyspnea on exertion, shortness of breath, fatigue, weakness, paroxysmal nocturnal dyspnea, and orthopnea. These symptoms are worsened at times of exertion or with gravitational patient positioning. Long-standing left-sided failure places more workload on the right ventricle and can progress to include right-sided heart failure with jugular venous distention, pitting pedal edema, and liver enlargement from hepatic congestion.

Serum measurements of beta natriuretic peptide released from the right and left ventricular myocytes

during volume and/or pressure overload can help confirm the diagnosis, and is especially useful to rule out other primary pulmonary causes of dyspnea. Beta natriuretic peptide levels are more accurate than the history, physical exam, and other lab tests in identifying congestive heart failure as the underlying cause of dyspnea.¹⁰ Chronic medical treatment for the patient with congestive heart failure includes exercise and weight loss, with fluid and sodium restriction. Medications include ACE inhibitors, beta-blockers to reduce the workload of the heart, diuretics to reduce the fluid volume, and digoxin to improve the cardiac inotropic function.

The oral and maxillofacial surgeon must take into consideration the volume of intravenous (IV) fluids administered to patients with congestive heart failure. Patient positioning during outpatient surgery is important, and the patient should be reclined a limited amount. For an acute episode of pulmonary edema, in addition to supplemental oxygen, the patient should be treated as an emergency with morphine, nitroglycerin, and a diuretic such as furosemide.

Respiratory system

Asthma

The prevalence of asthma is increasing, and it is now the leading chronic illness of childhood.¹¹ In the reactive airway, chronic inflammation of the bronchioles produces edema, smooth muscle contraction, and increased mucus production, all of which contribute to dyspnea and wheezing with cough. Asthma attacks can be triggered by exercise, allergy, exposure to respiratory irritants, or respiratory infections. Some patients will eventually outgrow the disease whereas others may develop it later in life. Chronic treatment is determined by the severity of the disease. For patients with mild and only intermittent attacks of asthma, treatment with a beta-2 agonist inhaler on a PRN basis is employed. If the exacerbations are more frequent but still with mild symptoms, a daily low-dose steroid inhaler is frequently prescribed, using the beta agonist as a rescue inhaler during acute flares. For the patient with somewhat more severe and persistent symptoms, in addition to the daily steroid inhaler and a long-acting bronchodilator, oral leukotriene antagonists or theophylline may be used. The most severe asthmatics may require treatment with oral corticosteroids.

Treatment of an acute asthmatic attack during outpatient oral and maxillofacial surgery should initially be addressed by placing the patient in an upright position and administering oxygen and a beta-2 agonist bronchodilator inhaler such as albuterol. If the symptoms become severe, subcutaneous epinephrine and corticosteroids should be given, with consideration of intubation.

Chronic obstructive pulmonary disease

COPD is usually the direct result of smoking, and more rarely of other inhaled environmental irritants. It commonly occurs as emphysema with destruction of the septae of the small airway spaces resulting in enlargement of the alveoli. Chronic bronchitis presents with persistent bronchial inflammation and increased mucus production causing cough. COPD often presents as a combination of both. Symptoms include shortness of breath, dyspnea, wheezing, and a productive cough. Cessation of smoking will help to reduce surgical anesthetic complications; however a rebound increase in sputum production may initially occur. Thus, a cessation period of 8 weeks is usually needed to minimize the pulmonary risks associated with smoking.¹²

Surgical risks in patients with COPD are not limited to those associated with the respiratory complications during anesthesia. Smokers are at increased risk for other systemic conditions such as myocardial infarction and stroke. Poor wound healing and a greater risk for wound dehiscence and infection are more pronounced in smokers.¹³

Chronic medical management of COPD includes cessation of smoking and the use of bronchodilators (e.g. beta-2 agonists or anticholinergic inhalers), steroid inhalers, and mucolytic agents. In severe cases, portable supplemental oxygen therapy may be required, with systemic oral glucocorticosteroids reserved for acute and severe exacerbations.

Renal system

Renal failure

Normal blood flow to the kidneys is high, around 20% of cardiac output. About 10% of the plasma passing through the renal arteries is filtered in the glomeruli. Renal failure has many causes including long-standing hypertension, diabetes, prolonged hypovolemia or hypoxia. The kidneys are oxygen sensitive, and are one of the early organs to show signs of damage after periods of hypovolemia or hypoxia. Intravenous contrast dye and drugs such as gentamicin and non-steroidal anti-inflammatory medications are nephrotoxic.

Renal disease is usually divided into three categories. Prerenal disease includes any process resulting in a limitation of blood flow to the kidney. Postrenal disease occurs when there is restriction to urine outflow with resultant back pressure into the kidney. Intrinsic renal disease involves direct injury or failure of the kidney filtration system.

For surgical patients with renal disease, close monitoring of urine output is essential. As a rule, appropriate urine output in any surgical patient should be at least 4ml/kg/h for the first 10kg in

patient weight, 2ml/kg/h for the second 10kg, and 1ml/kg/h for each additional kilogram. Therefore, for a 70kg adult, minimum urine output should be about 110ml/h. For patients with renal disease, it is crucial to maintain renal perfusion, so hypovolemic and hypotensive anesthetic techniques should be avoided whenever possible. In addition the surgeon should avoid contrast dye in radiographic studies and avoid or limit the use of nephrotoxic drugs such as non-steroidal anti-inflammatory medications.

For the patient with acute renal failure, low-dose dopamine (2µg/kg/min) has been used in the intensive care setting in attempt to improve renal perfusion, but the evidence to support its use is weak.¹⁴ Although low-dose dopamine has been shown to reduce renal vascular resistance in patients without acute renal failure, in those with acute renal failure low-dose dopamine can actually worsen renal perfusion.¹⁵

Uremia affects platelet function, so bleeding may be prolonged in patients with chronic renal failure. To reduce the risk of bleeding in patients on dialysis, elective oral and maxillofacial surgery should usually be scheduled for the day following dialysis when risk of uremia is low; dialysis is resumed 2 days after surgery at which time dialytic heparinization should be safe from a surgical hemostatic standpoint.

Endocrine system

Diabetes mellitus

Of all the endocrine diseases, diabetes mellitus is probably of most concern for the oral and maxillofacial surgeon. It is one of the most commonly encountered endocrine disorders in surgical patients. Not only can diabetes affect the outcome of surgery (e.g. increased risk of infection), the stress of surgery can affect the control of the disease, especially in the brittle diabetic patient. Diabetics are also at increased risk for many other systemic diseases such as coronary artery disease, cerebrovascular accidents, renal failure, etc.

Type 1 diabetes typically occurs in younger patients and is the result of autoimmune destruction of the beta cells of the pancreas. Hence, type 1 diabetic patients require exogenous insulin, either injected subcutaneously or via an insulin pump. Type 2 diabetes, which often occurs later in life, is due to insulin resistance at the target cells and is often followed by failure of the beta cells to fully compensate and produce sufficient insulin. As type 2 diabetes may develop in association with excessive weight gain, it may be completely or at least partially controlled with weight loss. Oral agents are used to control blood sugar in the type 2 diabetic patient, but insulin may be required for full control.

Exogenous insulins are classified primarily based on their time to onset and length of action. They are

classified as rapid acting (e.g. regular insulin, peak effect 2–4 hours), intermediate acting (e.g. NPH or lente, peak effect 6–12 hours) and long acting (e.g. ultralente, peak effect 10–16 hours) and are frequently used in combination. All insulin marketed in the USA is human insulin produced by genetically engineered *Escherichia coli*. Lantus is long acting and humalog and novalog are short acting. Nasally inhaled insulin is also available, but has only about 10% of the activity of subcutaneous insulin, is affected by smoking, and may cause pulmonary changes.

Oral agents for type 2 diabetes are targeted at various points in the glucose metabolism pathway. The sulfonylureas (e.g. glyburide, glipizide) act to increase insulin production by the beta cells. Biguanidines (e.g. metformin) act to decrease glucose production by the liver and increase glucose uptake in the periphery by sensitization of peripheral tissues to insulin. The alpha-glucosidases inhibit intestinal polysaccharide breakdown. Thiazolidinediones act to improve endogenous insulin sensitivity in target organs. Oral medications can also be used alone or in combination to control blood sugar in people with type 2 diabetes.

Blood sugar must be closely monitored and controlled in the perioperative period. A preoperative glycosylated hemoglobin level (HgA1C) will give an indication of the degree of blood glucose control over the prior 2–3 months, and thus is a better indicator of possible chronic sequelae of diabetes (e.g. coronary artery disease, diabetic nephropathy) than is one isolated fasting blood sugar value. A target value for glycosylated hemoglobin is less than 7%. However, in the perioperative period, serum glucose levels must be frequently monitored and controlled. This may be difficult in the brittle diabetic patient due to various surgical factors (surgical stress, nil per os (NPO) status, etc.) and so no standard regimen can be used by the oral and maxillofacial surgeon that will fit every diabetic patient.

Consideration must be given to the patient's overall diabetic status (well controlled versus brittle), timing of the procedure (preferably first case in the morning), length of the procedure, and time until oral intake can resume (especially important in patients undergoing oral and maxillofacial surgery). Often, short-acting insulin or short-acting oral medications are held on the day of surgery and longer-acting agents are given at a reduced dose. In the brittle diabetic patient these decisions should be made in consultation with the patient's endocrinologist. In the perioperative period, patients with type 1 diabetes are controlled with a sliding scale insulin dosage schedule.

Those patients with type 2 diabetes often require an insulin sliding scale during the immediate postoperative hospital stay. Dietary caloric control, while maintaining adequate nutrition for healing, is also important during hospitalization.

Gastrointestinal system

Liver failure

Due to the numerous functions of the liver, surgery presents an especially high risk for patients with reduced hepatic function. Liver failure can be a direct result of hepatitis, ethanol abuse, medications, etc. The liver functions to metabolize toxins and drugs, produce and excrete bile, synthesize proteins such as albumin and clotting factors, regulate metabolism (e.g. gluconeogenesis), etc. All of these functions are crucially important in the surgical patient.

Special consideration must be given to the patient in liver failure about to undergo surgery. The interval between dosing of medications may need to be increased due to slower metabolism of certain drugs. Hepatotoxic medications such as acetaminophen should be avoided. Patients with compromised liver function are at risk for poor wound healing. As serum albumin levels fall, wound healing becomes compromised. A more sensitive nutritional marker than albumin, serum prealbumin levels should be measured prior to surgery in patients with chronic liver failure. Since prealbumin is not a precursor of albumin, the more accurate name is transthyretin (serum transport protein for thyroxine and retinol-binding protein).¹⁶ (It was originally called prealbumin only because it preceded albumin on gel electrophoresis.) Since serum prealbumin levels respond more rapidly than albumin (due to albumin's longer half-life and larger body pool) prealbumin is a more sensitive test for monitoring changes in nutritional status. Low levels (<10 mg/dl) correlate with an increase in surgical blood loss, morbidity, and mortality.¹⁷

Control of bleeding is also a concern in the patient with liver failure. With the exception of factor VIII which is also produced by endothelial cells, all of the clotting factors are produced by the liver. Since bile is needed for vitamin K absorption, the vitamin K dependent clotting factors (II, VII, IX and X) are especially limited. Portal hypertension secondary to hepatic failure can cause splenomegaly, with associated platelet sequestration. The resultant thrombocytopenia further compromises blood clotting and can contribute to excessive surgical bleeding. For a patient in liver failure who has an international normalized ratio (INR) greater than 1.5 or platelet count less than 50 000, consideration should be given to transfusion of fresh frozen plasma and/or platelets prior to surgery.

Hematologic system

Disorders of hemostasis

The main components of blood include cells (red blood cells (RBCs), white blood cells (WBCs)), cellu-

lar particles (platelets), proteins (albumin, globulin, coagulation proteins, etc.) and serum. In surgery consideration certainly must be given to the quantity and quality of red cells for adequate tissue oxygenation and white cells to adequately prevent, resist, and/or combat infection. However, of all health care providers, it is the surgeon who is most influenced by the blood's ability to clot. Therefore, recognition and treatment of underlying bleeding disorders are of utmost concern to the oral and maxillofacial surgeon.

Put simply, blood clotting consists of primary hemostasis, where platelet aggregation forms the initial plug, followed by secondary hemostasis, where the plasma coagulation factors cascade to form the cross-linked fibrin clot. Both quantitative and qualitative defects of platelets and/or coagulation proteins produce disorders of hemostasis. These defects can be inherent to the platelets or clotting factors, secondarily acquired from other underlying medical conditions or iatrogenically induced.

Quantitative platelet disorders

Some inherent diseases causing thrombocytopenia include immune thrombocytopenia purpura (ITP) and thrombotic thrombocytopenia purpura (TTP). Examples of thrombocytopenia acquired secondary to other medical disorders include sequestration of platelets in patients with splenomegaly, failure of platelet production in myeloproliferative diseases such as leukemia, and acquired immunodeficiency syndrome (AIDS)-related thrombocytopenia, thought to be due to the effect of human immunodeficiency virus (HIV) on megakaryocytes. Iatrogenically induced thrombocytopenia occurs with bone marrow suppression from chemotherapy and is sometimes seen as a complication of heparin therapy. Treatment for thrombocytopenia usually involves platelet transfusion.

Qualitative platelet disorders

An inherent qualitative platelet disorder occurs with von Willebrand's disease. von Willebrand's factor has roles both in platelet adhesion and in stabilizing factor VIII, so patients with von Willebrand's disease have a qualitative platelet adhesion disorder. A qualitative platelet disorder secondary to another medical condition is seen in uremic patients with renal failure who demonstrate decreased platelet adhesion. Iatrogenically induced alterations in platelet quality occur with clopidogrel (Plavix) and with non-steroidal anti-inflammatory medications. (Of these, aspirin is of special concern because its effect on platelet adhesion is irreversible. To reverse aspirin's effect, sufficient time must pass after stopping the aspirin to allow for production of an adequate number of new platelets.) Treatment of qualitative platelet problems

usually involves treating the underlying etiology (e.g. dialysis to correct uremia or abstinence from aspirin for 7–10 days prior to surgery). For von Willebrand's disease, DDAVP (desmopressin, a synthetic analogue of vasopressin) can be effective, as it induces the release of von Willebrand's factor from endothelial cells, resulting in a virtually immediate increase in von Willebrand factor.¹⁸

Disorders of coagulation factors

With respect to the coagulation factors, many inherent conditions exist. The more common include hemophilia A (factor VIII deficiency), hemophilia B (factor IX deficiency), and von Willebrand's disease (instability of factor VIII). These inherent coagulopathies are corrected with the appropriate recombinant replacement factor (VIII or IX), with fresh frozen plasma or with cryoprecipitate. In addition to causing the release of von Willebrand's factor from endothelial cells, DDAVP also causes factor VIII release from the liver, resulting in a three- to five-fold increase in plasma factor VIII within 30 minutes of its administration,¹⁹ so DDAVP may be useful in hemophilia A (but not B). von Willebrand's disease has several subtypes, and DDAVP is useful for some subtypes but not others. Other medical conditions affecting the coagulation proteins include vitamin K deficiency and chronic liver failure as previously described. Iatrogenically induced coagulopathies are seen with the effects of warfarin (affecting the extrinsic pathway of coagulation and measured with prothrombin time (PT) and INR) and with heparin (affecting the intrinsic coagulation pathway, and measured with activated partial thromboplastin time (PTT)).

For patients with hematologic conditions predisposing them to excessive bleeding, the oral and maxillofacial surgeon can also consider local measures to prevent the subsequent breakdown of clots. For example, epsilon aminocaproic acid and tranexamic acid (antifibrinolytic agents) can both be used topically as an intraoral mouthwash.²⁰

Immunologic system

HIV/AIDS

HIV is a retrovirus that attaches to the CD4 receptor of host cells, including leukocytes, causing a defect in cell-mediated immunity. The immunocompromised patient is at risk for opportunistic bacterial, viral, fungal, and protozoal infections (e.g. pneumocystis or mycobacterium pneumonias, fungal infections, etc.) and at increased risk for cancer (e.g. Kaposi's sarcoma, non-Hodgkin's lymphoma). HIV, transmitted by blood-borne mechanisms and sexual contact, affects multiple organ systems, with cardiac and respiratory complications, neurologic sequelae (e.g. AIDS-related

dementia), kidney effects (e.g. HIV-associated nephropathy), hematologic effects (e.g. AIDS-related thrombocytopenia), etc. Although the time from initial HIV exposure to the development of AIDS can vary tremendously, the median incubation period is reported to be about 10 years.²¹ Drugs used to treat HIV/AIDS are aimed at inhibiting reverse transcriptase, inhibiting viral proteases, and blocking HIV entrance into cells.

Two specific tests are commonly followed in patients with HIV/AIDS. Measurement of viral load is important, not only to diagnose the disease, but also to monitor the effectiveness of antiviral therapy. The level of immunocompromise is evaluated with the CD4 count, which is inversely correlated with the risk for opportunistic infection. The CD4 count is also important for determining when to initiate certain antiviral therapies. A normal CD4 count is greater than 600 cells/ μ l. In the asymptomatic patient, antiviral therapy may be recommended when the CD4 count drops below 350 cells/ μ l.

For AIDS patients undergoing elective oral and maxillofacial surgery, certain preoperative lab tests are recommended. These include a complete blood count (CBC) with platelets, liver function tests with coagulation studies, blood urea nitrogen (BUN) and creatinine to evaluate HIV-associated nephropathy, chest radiography and EKG to evaluate any cardiorespiratory complications, and a CD4 count to determine the patient's immune status.²² For patients with low or no detectable viral titers and normal CD4 counts, no specific precautions need to be taken for surgery. However, patients with suppressed CD4 counts should be covered with prophylactic antibiotics.

Neurologic system

Seizure disorders

Seizures present in various forms. They can be generalized or localized, tonic-clonic (grand mal) or absence (petit mal). Generalized tonic-clonic seizures can result from many conditions such as high fever (especially in children), cerebral hypoxia, intracranial space-occupying lesions (e.g. brain tumor), head trauma, stroke, meningitis, drug withdrawal (e.g. ethanol), local anesthesia overdose, etc. Epilepsy is recurrent seizures due to abnormal cortical neural pathway activation.

Medications (barbiturates, benzodiazepines, hydantoins, succinimides and others) are the first-line treatment for epilepsy, used either alone or in combination. Other treatments include implantation of a vagal nerve stimulator, use of a strict ketogenic diet, and neurosurgical ablation.

Prior to elective surgery on a patient with epilepsy, the oral and maxillofacial surgeon should confirm

that the patient has been compliant with prescribed antiseizure medications, as there is an increased risk of seizure, including status epilepticus, with acute withdrawal of antiseizure medications. In addition, the surgeon may want to confirm that blood levels of the antiseizure medications are in the therapeutic range.

Treatment of a seizure that occurs in the outpatient surgical setting includes administration of a benzodiazepine (e.g. diazepam 5–10mg or midazolam 2–4mg), protection of the patient from injury during the event, and supportive care in the postictal period. Immediate medical evaluation is not always required, but should be considered for a first-time seizure (to rule out other underlying etiologies), if the patient was injured during the seizure, if the patient has persistent mental status changes after the seizure, or if the ictal phase of the seizure fails to resolve within about 5 minutes, as status epilepticus carries a 7–39% mortality rate due to cardiovascular and respiratory compromise.²³

Cerebrovascular accident

Strokes result from a sudden decrease in perfusion of a discrete region of the brain, and are caused either by occlusion of a cerebral artery (thrombotic or embolic) or by rupture with subsequent intracranial hemorrhage. The neurologic deficits that ensue (sensory deficits, motor deficits, aphasia, visual loss, loss of consciousness, etc.) depend on the anatomic location of the stroke. Risk factors for stroke include hypertension, diabetes, smoking, dyslipidemia, cardiac arrhythmias, valvular heart disease, use of oral contraceptives, etc. Since a history of stroke or transient ischemic attack is a strong predictor of perioperative stroke, the surgeon should fully investigate and treat the cause of any stroke or transient ischemic attack that has occurred within the previous 6 months.²⁴

If a patient is suspected of suffering a stroke, *immediate* evaluation and implementation of treatment is indicated. According to the Stroke Council of the American Stroke Association/American Heart Association guidelines,²⁵ if intravascular recombinant tissue plasminogen activator (t-PA) is to be used in attempt to dissolve an occlusive clot and allow for cerebral reperfusion, it must be initiated within 3 hours of the onset of the stroke. Imaging studies (computed tomography (CT) or magnetic resonance (MRI)) first will be needed to determine if the stroke is occlusive or hemorrhagic. In the immediate postoperative period, intravascular t-PA may be contraindicated due to increased risk of surgical bleeding; however direct intra-arterial t-PA may be relatively safe.²⁴ For patients with hemorrhagic strokes, supportive care is provided while the blood pressure is controlled. To maintain cerebral perfusion, blood pressure is controlled gradually to avoid worsening the outcome by additional hypotensive ischemia. In

some situations neurosurgical evacuation of the clot may be beneficial.

Musculoskeletal system

Bisphosphonate-related osteonecrosis of the jaws (BRONJ)

Bisphosphonate medications are prescribed for conditions where a reduction in bone resorption is beneficial. The two most common indications are (1) to increase bone density in patients with osteoporosis and (2) to limit the extension of intrabony malignancies and decrease hypercalcemia in patients with primary bone tumors (e.g. multiple myeloma) or metastatic bone tumors (e.g. breast or prostate carcinoma). These medications are most frequently prescribed in oral weekly doses for osteoporosis (e.g. alendronate, risedronate) and monthly IV doses for patients with primary or metastatic bone tumors (e.g. pamidronate, zoledronate). However, annual IV dosing for osteoporosis now is being advocated.^{26,27}

Some patients taking bisphosphonate medications develop areas of non-healing or slowly healing exposed necrotic bone in the jaws. These areas can enlarge, become secondarily infected, and may eventually result in jaw fracture and/or require resection. The American Association of Oral and Maxillofacial Surgeons defines BRONJ as exposed bone in the maxillofacial region that has persisted for more than 8 weeks in a patient who has received bisphosphonate medications and with no history of radiation therapy to the jaws.²⁸

BRONJ can occur spontaneously, or after a surgical procedure that exposes the jaw bone in a susceptible patient. For patients on monthly IV bisphosphonate therapy, approximately half of the cases of BRONJ occur after a dental procedure, and half spontaneously (although half of these may be attributable to uncontrolled periodontitis).²⁹ The risk is very much greater for patients on monthly IV bisphosphonate therapy for control of bone cancer as compared to those taking oral bisphosphonates weekly or monthly for osteoporosis. Risks have been estimated at 5–15% for the former²⁹ but at only 0.7/100 000 per year of drug use for the latter.³⁰ The risk is known to be cumulative, with patients being at increasingly greater risk the longer they have been taking the bisphosphonate. The risk accumulates faster for those on monthly IV forms (zoledronate at approximately 6 months, pamidronate at approximately 12 months) as compared with weekly oral doses (alendronate at about 36 months). However, cases of BRONJ have been seen after only one or two IV doses (author experience). Concurrent factors such as chemotherapy, steroid use, smoking, etc. are felt to increase the risk.

For patients taking monthly IV bisphosphonates, surgical jaw procedures should be avoided whenever

possible. Root canal therapy with crown amputation should be performed for non-salvageable teeth, as opposed to extraction. For patients taking weekly oral bisphosphonates, dentoalveolar surgery is not absolutely contraindicated. In this group, the risk of developing BRONJ is less and the condition is often less aggressive and more easily treated when it does occur. Nevertheless, for elective surgical procedures it may be beneficial to hold the oral bisphosphonate for several months prior to the planned procedure (depending on the length of time the patient has been taking the oral medication) and resume it several months after healing has occurred.

For patients with established BRONJ, conservative treatment is usually recommended. The exposed bone should be kept clean, and oral chlorhexidine rinses prescribed twice daily. Antibiotics are reserved for episodes of acute infection. Conservative debridement of exposed irritating bone can be performed, so long as no periosteum is elevated that exposes additional bone. Aggressive debridement or resection is reserved for the most severe cases (e.g. jaw fracture, infection not controlled with antibiotics). The possibility of holding the bisphosphonate medication can be discussed with the patient's physician, and may lead to resolution over time, especially for patients on oral bisphosphonates.

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Chapter 4

Medical Emergency Care

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- Emergency situations are to be expected in any active surgical practice.
- Cardiac angina, arrest, asthma attacks, anaphylaxis, syncope, hypoglycemia, and seizures are the most common emergencies encountered in the office setting.
- Prevention, the most important component of management of medical emergencies, is discussed.
- Early recognition and diagnosis of various medical emergencies allows for prompt and successful management. The presentation and pathophysiology of various medical conditions are reviewed.
- Treatment of medical emergencies begins in the office, although knowing when to involve emergency medical services is of utmost importance.
- Treatment for the most common medical emergencies is discussed in detail.

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Surgical care in the outpatient setting requires knowledge and preparedness in management of medical emergencies. Surveys of both American and Japanese dental practitioners found that between 19% and 44% of dentists were faced with a medical emergency in any 1 year.¹⁻³ If the numbers were limited to oral and maxillofacial surgeons, it is assumed the percentages would be higher. This chapter will discuss recognition and management of medical emergencies as they pertain to the oral and maxillofacial surgeon.

The first step for surgeons is to accept the fact that emergencies do occur. Preparedness requires sufficient time, effort, and money. Surgeons are seeing an increasing number of elderly and medically compromised patients, making emergencies inevitable. It is emphasized that all members of the surgical office staff must be trained to promptly recognize and efficiently perform appropriate duties in emergency situations.

Sempowski and Brison⁴ classified outpatient offices on the basis of risks for medical emergencies. High-risk offices included those that are in a rural or remote location, have no local hospital, perform invasive procedures in the office, frequently give parenteral medications, or have no access to emergency medical services. Also considered high risk are large group or high-volume practices, as well as those exposed to inclement weather. Oral and maxillofacial surgery offices would be considered in the high-risk category due to the invasiveness of procedures, the provision of anesthesia and the often high volume of work in a variety of locations.

Undoubtedly, the best way to manage medical emergencies is before they happen through a thorough preoperative evaluation. The practitioner must consider not only the pathophysiology of a particular patient's medical comorbidity, but also how they will be affected by the stress of the anticipation and experience of a surgical procedure. This chapter will focus on the most common medical emergencies encountered, including angina, cardiac arrest, asthma, syncope, drug hypersensitivity, hypoglycemia, and seizure.

Syncope

The most common medical emergency encountered in the office is syncope. Syncope is defined as the transient loss of consciousness and postural tone leading to spontaneous recovery without neurological deficit. Vasovagal syncope is the most common cause of unconsciousness in the office setting. It can be psychogenic resulting from fright, anxiety, or certain situations such as the sight of blood. Non-psychogenic causes include prolonged standing and dehydration. Although, syncope is usually a benign, self-limiting event, the surgeon must still be vigilant and rule out other more serious etiologies of unconsciousness.

Cardiac causes include obstructive outflow disease including aortic stenosis, hypertrophic obstructive cardiomyopathy, or acute left ventricular failure secondary to myocardial infarction. More commonly, syncope can be the result of acute arrhythmias such as bradycardia. Neurogenic causes of unconsciousness include some types of seizures, transient ischemic attack, migraines, and subclavian steal syndrome. Stroke is an extremely rare cause of syncope and presents with focal neurologic signs and symptoms well in advance of any loss of consciousness.

Orthostatic hypotension can lead to syncope in patients with depleted intravascular volume and can occur as a side-effect of certain drugs such as antidepressants and antihypertensives. Orthostasis also can occur in patients with autonomic instability, such as diabetes mellitus, and prevalence increases with advanced age. Lastly, hypoglycemia is a rare cause of syncope.

Vasovagal or vasodepressor syncope is generally the etiology in young, healthy patients without the above risk factors. Loss of consciousness usually occurs in three stages, with loss of consciousness occurring after a warning or presyncopal stage. The first stage, the presyncopal stage manifests as a feeling of warmth in face and neck, diaphoresis, and pallor. The heart rate may rise significantly while blood pressure often stays near the patient's baseline. As presyncope progresses, the pupils dilate, respiratory rate increases, and the blood pressure and heart rate drop resulting in loss of consciousness. The patient's breathing may be irregular and the patient may gasp or even become apneic. Convulsions may be observed in the hands, legs, or facial muscles and may often be confused for seizure activity. The pulse is thready and blood pressure can drop to extremely low levels. Generalized muscle relaxation occurs and can lead to loss of the airway. The period of unconsciousness can last from seconds to several minutes after placement in the supine position.⁵ If the patient's unconsciousness lasts for more than 5 minutes after being supine, other causes of syncope should be considered, especially if the patient is over 40 years old and had no prodrome to the loss of consciousness.⁶ Postsyncope

or recovery is usually rapid, although pallor, nausea, and diaphoresis often persist along with some confusion or disorientation.⁷

Management

Upon recognition of presyncope symptoms, the procedure should be stopped immediately, and the patient should be placed in the Trendelenberg position and administered oxygen. This can prevent actual syncope or shorten the syncopal episode. If unconsciousness does ensue, one should resort to the basic life support protocol, first calling for help, then assessing the airway, breathing, and circulation. A simple chin lift or jaw thrust will usually open the airway. Failure to lay a syncopal patient flat can result in brain damage or even death.⁸ If breathing and circulation are present, an ampule of ammonia can be crushed under the patient's nose to hasten recovery. If not done already, vital signs should be obtained and a rhythm analyzed. Vasovagal syncope is very unlikely if unconscious lasts beyond 10–15 minutes. Other, more ominous etiologies of syncope are likely and emergency medical services should be activated if this has not been done already. Definitive management of other causes of syncope often requires work-up in an inpatient setting and treatment is for the underlying cause of the syncope.

Chest pain

Chest pain is an exceedingly common complaint in the surgical patient. Given the high prevalence of cardiovascular disease and the ageing population, it accounts for a large percentage of office emergencies. Many etiologies of chest pain exist, although most commonly it results from angina pectoris and myocardial infarction.

Pathogenesis

Coronary artery disease results from atherosclerosis of the coronary arteries that develops in a predictable manner. The progression of atherosclerosis is accelerated by endothelial dysfunction, inflammation, and thrombosis. The advanced atherosclerotic lesion has a core of lipids and necrotic tissue surrounded by a fibrous cap. The fibrous cap is prone to rupture which can lead to further thrombus formation and occlusion and represents the most common cause of the acute coronary syndrome. The complete occlusion of a coronary artery due to ruptured plaque leads to transmural ischemia and eventually infarction of the myocardium. This is represented as an ST elevation myocardial infarction (STEMI) on 12-lead EKG. Non-occlusive thrombus causes ischemia at the distal aspect of the blood flow of the vessel, the subendocardium, leading to non-ST elevation myocardial infarction (NSTEMI).

Patients with classic angina pectoris usually have fixed atherosclerotic lesions resulting in greater than 70% blockage.⁹ Angina results from an imbalance in oxygen supply and demand in the myocardium. Exertion or stress from a surgical procedure leads to higher oxygen demand that cannot be met by a stenotic coronary vessel and leads to ischemia and pain. Coronary blood flow is normally autoregulated over a wide range of perfusion pressures by vasodilation and constriction. The vascular bed beyond a significant stenosis is usually already dilated maximally at rest and cannot adapt to increasing demand. Oxygen demand is determined by heart rate, systolic blood pressure and wall stress, with the latter determined by ventricular pressure, volume and wall thickness. Patients with long-standing hypertension often have hypertrophied myocardium leading to increased wall stress and are often intolerant of increased heart rate or blood pressure.

Diagnosis and history

When presented with a patient with chest pain, the clinician must decide if the pain has a cardiac or non-cardiac origin. Pain secondary to cardiac ischemia is rarely sharp and well localized, rather it is often described as uncomfortable pressure, tightness, or fullness substernally or just left of the sternum. The feeling is most often not described as pain at all, but rather pressure or discomfort. Cardiac pain is usually constant and without respiratory variation. Pain due to angina, classically subsides gradually over 2–5 minutes with adequate rest. If the pain continues, it is more likely due to infarction of the myocardium. The discomfort may radiate to the jaw, shoulders, the medial aspect of the arm and fingers most commonly on the left side. Patients may also be diaphoretic and describe a feeling of impending doom (*angor animi*). Centrally located dyspnea, especially in older patients, is often considered an “anginal equivalent” and could be the only presenting sign of cardiac ischemia.

Non-anginal chest pain can result from other serious conditions including aortic dissection, spontaneous pneumothorax, pulmonary embolism, esophageal spasm or rupture, and musculoskeletal injury (Table 4.1). Pain from aortic dissection usually is tearing and radiates to the back. Pneumothorax is often associated with shortness of breath and hemodynamic compromise if under tension. Pain from a pulmonary embolism is often pleuritic in nature and can, but not necessarily, result in hypoxia. Musculoskeletal pain is reproducibly tender to palpation.

It is often difficult to discern angina from actual myocardial infarction. A diagnosis of angina requires that the patient have a history of angina and the presenting pain must be consistent with the patient’s prior chest pain. In all other scenarios, the pain cannot be attributed to angina and must be treated as myocardial infarction until proven otherwise.

Table 4.1 Cardiac angina vs non-cardiac pain.

	More likely to be cardiac	Less likely to be cardiac
Description of pain	Dull; constant	Sharp, stabbing; intermittent
Onset	Gradual	Rapid
Location	Substernal	Lateral chest wall, back
Associated symptoms	Radiation to left arm or jaw, diaphoresis, nausea, feeling of impending doom	Not present
Reproducibility	On exertion	With inspiration
Tenderness to palpation?	No	Yes, may reproduce pain complaint

Management

After a brief targeted history into the characteristics of the pain, further assessment of a patient with chest pain suggestive of ischemia includes assessment of airway, breathing, circulation, and measuring of vital signs including oxygen saturation. Immediate treatment is aimed at restoring the balance between oxygen supply and demand. Patients experiencing chest pain generally prefer upright or erect positions. As indicated, the surgeon may administer morphine, oxygen, nitroglycerin, and aspirin. This can be easily remembered as the mnemonic MONA.¹⁰ Start oxygen at 4–6 L/min through a nasal cannula. Prior to giving nitroglycerin, assess whether the patient has taken a 5-phosphodiesterase inhibitor such as sildenafil or tadalafil within the last 24 hours, as serious hypotension can result if combined with nitroglycerin. If not, a nitroglycerin tablet or spray (0.4 mg) should be given sublingually every 5 minutes. Anginal pain will likely be relieved by nitrates in 1–2 minutes. One must be aware that the shelf life on nitroglycerin tablets is 3–4 months after opening. If the patient’s tablets are used and fail to relieve chest pain, one should then use nitroglycerin spray from the office emergency kit. Pain that persists despite a dose of metered aerosol nitroglycerin likely represents myocardial infarction. The adult patient with no history of cardiovascular disease that develops chest pain should be considered myocardial infarction until proven otherwise. In addition, patients whose chest pain returns after initially being relieved by nitroglycerin should also be managed as infarction. Emergency medical services should be sought immediately for transfer to a medical center capable of thrombolysis.

While waiting for help to arrive, continue to monitor vital signs. The patient should be kept in the upright or erect position. Aspirin (160–325 mg) should be chewed by the patient. Pain is further treated with IV morphine (2–5 mg) every 5–10 minutes.¹¹ If no IV is available, nitrous oxide can be administered at 35–40% without titration for relief of ischemic pain.¹² The surgeon should prepare to manage complica-

tions of myocardial infarction including acute dysrhythmias and cardiac arrest. Dysrhythmias usually occur within 1–2 hours of MI and can include bradycardia, ventricular fibrillation/tachycardia, or asystole.¹³ Always have an automated external defibrillator (AED) close by and be prepared to implement advanced cardiovascular life support (ACLS). Early transfer to the hospital also allows for better management of complications.

If available, obtain a 12-lead EKG to direct further management. This will allow differentiation into STEMI (ST segment elevation myocardial infarction) representing transmural infarction, NSTEMI (non-ST segment elevation myocardial infarction), and unstable angina. Management of MI in the acute period has improved dramatically. Angioplasty, stenting, or intravenous fibrinolytic therapy can be used to reopen occluded vessels. To be most effective, however, interventions should be initiated as soon as possible as many centers require less than 1 hour from symptom onset for primary angioplasty.¹⁴

Cardiac arrest

Oral and maxillofacial surgeons are treating an increasing number of elderly and medically compromised patients and therefore are more likely to encounter serious medical emergencies including cardiac arrest. Of all cardiovascular deaths in the USA, about half occur out of the hospital. The most frequent etiologies are ventricular dysrhythmias, commonly ventricular fibrillation or tachycardia (VF/VT), which may deteriorate rather quickly to asystole, a rhythm typically refractory to further treatment.^{15,16} See Table 4.2.

Ultimately, the patient that develops an arrhythmia or cardiac arrest needs to be managed in an emergency department with full ACLS protocols. As response times of emergency medical services (EMS) may vary, it is of utmost importance that offices have the tools to manage the initial phases of resuscitation.

Table 4.2 Symptomatic arrhythmias: signs and symptoms of hemodynamic compromise. Adapted from Haro *et al.*, Arrhythmias in the office. *Med Clin N Am* 2006; 90: 417–36.

Symptoms
Lightheadedness
Chest pain/pressure
Dyspnea
Palpitations
Orthostasis
Syncope
Signs
Hypotension
Heart rate >130 or <50 beats per minute
Cyanosis
Cool extremities
Altered mental status
Tachypnea

Unfortunately, the survival to hospital discharge after an out-of-hospital cardiac arrest is dismal. It has been reported that for each minute of untreated cardiac arrest, the probability of successful rhythm conversion decreases by 7–10%.^{17,18} Improvements in survival after sudden cardiac arrest have been related to institution of the “chain of survival” paradigm.¹⁵ The four links of the chain include: (1) early recognition and call for EMS; (2) initiation of basic life support including cardiopulmonary resuscitation (CPR); (3) defibrillation; and (4) ACLS drug intervention. Performing CPR early to increase perfusion and oxygenation will help make the myocardium more amenable to defibrillation or cardioversion. Preparedness for cardiac arrest in the office requires staff basic life support training, a simple office emergency protocol, necessary equipment, and rapid notification of emergency medical services.

The automated external defibrillator

Early defibrillation in cardiac arrest caused by VF/VT is of utmost importance. The automated external defibrillator (AED) is a device that can analyze a cardiac rhythm and instruct a user to deliver a shock when indicated. AEDs use microprocessors to analyze the frequency, amplitude and structure of an electrocardiogram (EKG) signal. Today, nearly all AEDs use biphasic currents to deliver a shock. Studies show that AEDs have a sensitivity for detecting VF/VT of greater than 90% and a specificity of greater than 95%.¹⁹ False positives can occur most likely due to patient movement by a rescuer or secondary to breathing. These errors can be minimized by proper verification of unresponsiveness, apnea, and pulselessness before an AED is used.

All AEDs are used in the following manner:

1. Power on the AED.
2. Attach electrode pads (Fig. 4.1). The right electrode is placed to the right of the sternum and below the clavicle. The left electrode is placed just lateral and inferior to the left nipple about 15 cm inferior to the axilla. The pads must achieve direct contact with the patient’s skin. If hirsute, hair can be removed rapidly with quick removal of an electrode pad.
3. Press “Analyze” to assess the rhythm.
4. State “Clear” and deliver shock if so prompted by the AED.

Successful resuscitation efforts with an AED depend on proper training and maintenance. A recommendation for all offices to have AEDs has been met with some resistance, which is likely secondary to perceived lack of cost-effectiveness. However, AED use has been integrated into all basic life support courses which allow non-physicians to deliver what is thought to be the most important life-saving measure for a patient in cardiac arrest without the

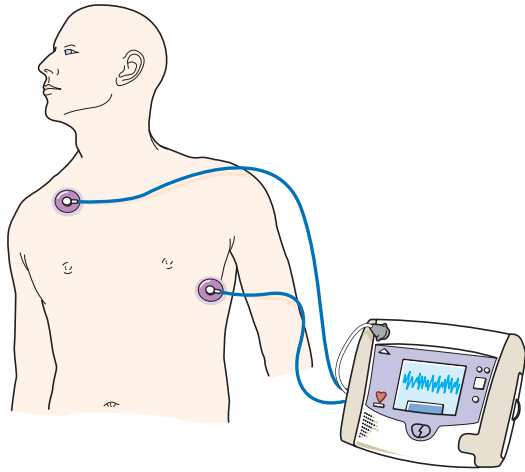


Fig. 4.1 Correct placement of the electrodes for an AED. The right electrode is placed to the right of the sternum and below the clavicle. The left electrode is placed just lateral and inferior to the left nipple approximately 15 cm inferior to the axilla.

requirement of experience and knowledge of complex cardiac rhythms. Importantly, one needs to recognize that defibrillation itself does not restart the heart, but actually terminates all electrical activity so that normal pacemakers can resume activity. Early CPR helps restore perfusion to the myocardium and other organs, which facilitates the heart's ability to then resume its spontaneous rhythm. Despite some controversies, it is these authors' opinion that an AED should be readily available for any office- based surgical practice treating adults that are at risk for sudden cardiac arrest.

General approach to the collapsed patient

Always assess the level of responsiveness of the patient. If some response occurs, check the level of alertness, chief complaint, and vital signs. Further treatment is dictated by the particular abnormality. If the patient is unresponsive, EMS should be notified immediately, ask for an AED and begin the primary survey as for all unresponsive patients as follows:

- **A: Airway.** Assess the airway by first opening the airway with a head tilt–chin lift or jaw thrust maneuver. Inspect the mouth for foreign bodies, vomitus, or blood.
- **B: Breathing.** Keeping the airway open, look, listen and feel for breathing. If the patient is not breathing after opening the airway, give two rescue breaths using a bag–mask.
- **C: Circulation.** Check for a pulse at the carotid artery. If there is no pulse, cardiac arrest is confirmed. Perform cycles of compressions and ventilations. The correct ratio is 30 chest compressions at a rate of 100 compressions/minute followed by two breaths. This cycle should be performed until an AED arrives.

- **D: Defibrillate.** Attach pads of AED and follow AED prompts as it assesses the rhythm. Deliver a shock if indicated by the prompts. Follow each shock immediately with more cycles of CPR.

The priority during cardiac arrest is for high-quality CPR and early defibrillation when indicated (Fig. 4.2). Secondary considerations include advanced airway and IV drug administration. No drug given during cardiac arrest has been shown to improve survival to hospital discharge or improve neurologic function, so CPR should not be delayed for locating and administering drugs.¹⁰

Considerations in transfer of the patient

It is well known that an unstable patient is best cared for in a hospital setting that is fully equipped for medical emergencies. Transfer of patients inherently results in great vulnerability. Efforts by both the surgeon and EMS should be aimed at stabilizing the patient as above before transfer is made.

Asthma

Asthma is a very common disease that affects an estimated 6.7% of adults and 8.5% of children in the USA.²⁰ It accounts for nearly 2 million emergency room visits yearly and approximately 40% of adults with asthma require emergency room care each year.²¹

Pathophysiology

Asthma is characterized by airway hyperresponsiveness leading to bronchiolar smooth muscle constriction, reversible obstruction and inflammation. The most frequent triggers include viral or bacterial respiratory infections, exposure to cold, allergens, irritants such as smoke, as well as exercise. Exposure to emotional distress or medications such as aspirin, non-steroidal anti-inflammatory drugs (NSAIDs), or beta-blockers can also incite an attack.²²

Prevention

Given the high prevalence of asthma throughout the world, the oral and maxillofacial surgeon must always be prepared to deal with an asthmatic emergency. The goal in the management of asthmatic patients is prevention of acute attacks through a careful history and physical examination prior to commencing treatment. The clinician should ascertain the extent of the patient's asthma by finding out about how often they have attacks, baseline peak flow rate, use of inhaled agents including corticosteroids, and if the patient has ever needed emergency care or intubation.

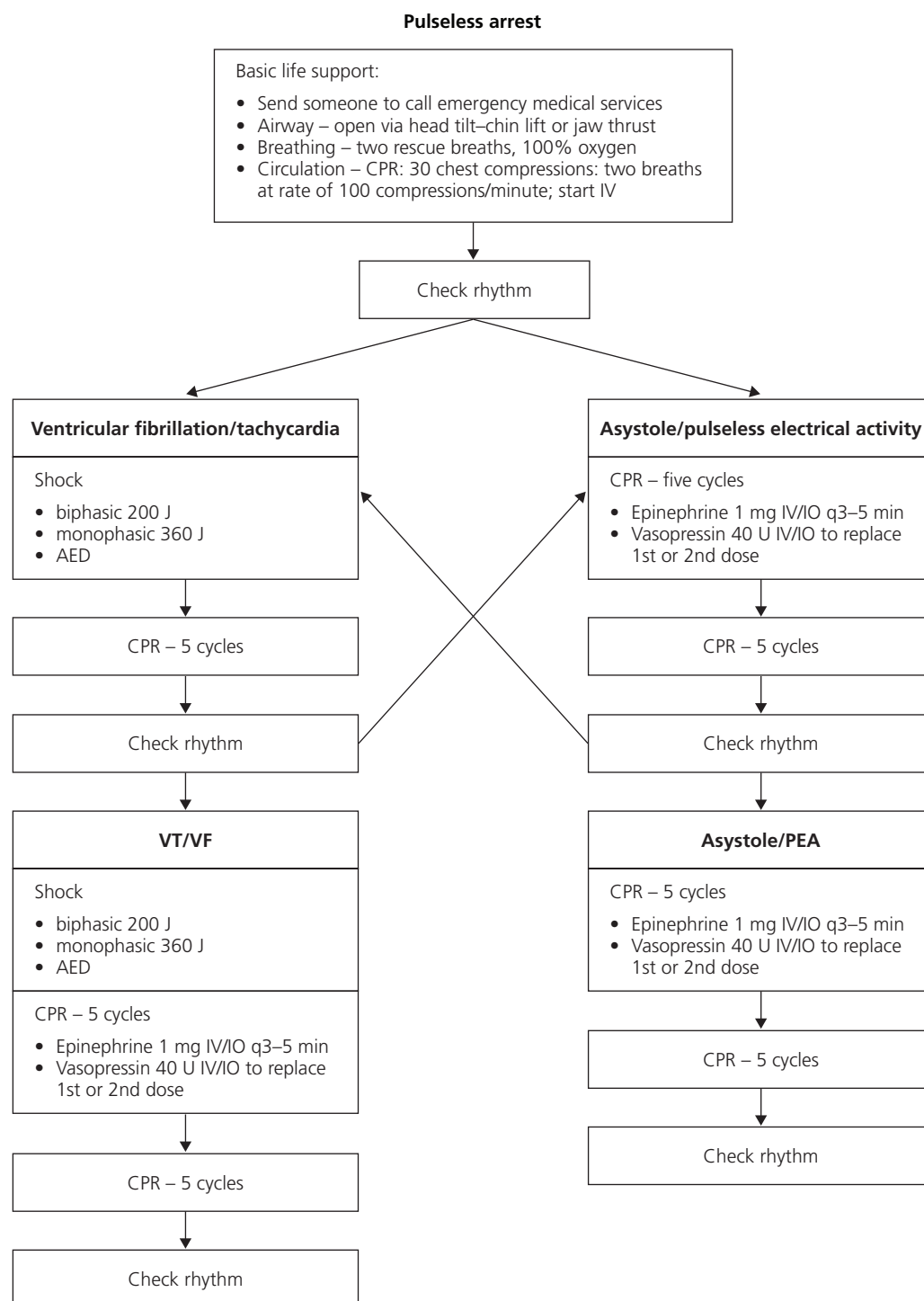


Fig. 4.2 Pulseless arrest algorithm. Adapted from: *Advanced Cardiovascular Life Support, Provider Manual*, 2006.

Emotional distress in fearful patients can trigger attacks and methods to reduce stress should be discussed. Common anesthetic drugs such as opioids and barbiturates can induce bronchospasm in asthmatics and should be used very carefully. Opioids lead to histamine release and barbiturates are thought to sensitize the respiratory reflexes, easily triggering a response in a predisposed patient.⁸ Aspirin sensitivity is present in 21% of adult asthmatics and 5% of child asthmatics²³ and nearly one third of asthmatics with nasal polyps.²⁴ This is thought to be diversion of arachidonic acid down the 5-lipoxygenase pathway

to form leukotrienes with aspirin's inhibition of the cyclooxygenase pathway. Leukotrienes are thought to significantly contribute to bronchoconstriction.²⁵ A significant amount of cross-reactivity exists between NSAIDs and aspirin, and they must also be used with caution in severe asthmatics.⁸

Clinical presentation

A typical asthma attack presents as congestion or tightness of the chest, cough which is often non-productive, and inspiratory and expiratory wheezing.

Some asthmatic patients are very self-aware of increased asthma symptoms while others will not perceive reduced airflow until it is severe. A peak flow meter is vital for differentiating severity of attacks and can be used in the office to assess respiratory compromise.²⁶ The analysis must be compared to the patient's baseline, so obtaining it in the history is crucial.

Patients exhibiting severe airway obstruction often experience increasing dyspnea and have to lean forward, with arms braced using accessory respiratory muscles to assist with breathing. Patients can become diaphoretic, tachycardic, and often hypertensive. Speech becomes very difficult for the patient and occurs in staccato-like short phrases. Upon auscultation, wheezing can be heard due to turbulent airflow through obstructed airways. Also, crackling sounds characteristic of atelectasis due to mucus plugging can be heard. Wheezing has no correlation with severity of attack, however, as the audible sound can disappear with severe narrowing of airways. Hypoxia is a late sign and if present should prompt immediate treatment.

Management

The surgical procedure should be stopped immediately and all materials removed from the mouth of any patient suspected of having an asthma attack. First, one should assess the severity of the attack based on clinical signs above and peak flow measurements. If capnography is available, alveolar carbon dioxide level (PaCO_2) can be assessed. Respiratory drive is increased in these patients and PaCO_2 should be lower than normal. Elevated or even normal PaCO_2 indicates severe airway narrowing and air trapping. Oxygen should be administered and beta-2 agonist administered via metered dose inhaler (MDI) for bronchodilation. The typical drug given is albuterol via MDI. Six puffs of albuterol MDI (90 μg /puff) are recommended and can be repeated every 20 minutes as needed.

The patient with moderate to severe bronchospasm or obstruction that does not adequately respond to bronchodilators is considered to be in status asthmaticus, a true medical emergency. EMS should be alerted immediately. From increased work of breathing, these patients can quickly fatigue, ultimately leading to hypoxia and shock. Epinephrine, a beta-1 and beta-2 agonist, should be injected subcutaneously. It is available as a preloaded syringe as 0.3 ml of 1:1000 dilution. It should only be administered subcutaneously due to its cardiovascular side-effects when given intravenously. Epinephrine can be given every 20–30 minutes for up to three doses. In the severe attack, parenteral administration of glucocorticoids is required to ultimately relieve the airway inflammation, the underlying etiology.²⁷ Although the benefit of administration is not seen for 3–6 hours it is thought to be better to administer corticosteroid early in the

attack.²⁸ Although the ideal dose of corticosteroid remains unknown, a patient in status asthmaticus should be given 125 mg of methylprednisolone or equivalent in the office or once they arrive in the emergency department.²⁹

Patients with severe hypoxia and respiratory failure require intensive care admission and often intubation. Failure of the patient to respond to initial therapy in the office and continued worsening always warrants emergency department evaluation and definitive management.

Hypoglycemia

According to the Center for Disease Control, more than 7% of adults in the USA are known to have diabetes mellitus.³⁰ Complications that can occur include hypoglycemia, diabetic ketoacidosis, and hyperosmolar non-ketotic coma. Generally, in the setting of the surgical office, hypoglycemia is the most worrisome complication due to its acute nature. The hyperglycemic complications are more often the end result of a more chronic hyperglycemic state and only very rarely encountered in the outpatient setting. Hypoglycemia is defined as blood glucose levels less than 50 mg/dl in adults and less than 40 mg/dl in children.³¹

Hypoglycemia most commonly occurs in the patient being treated for diabetes but may also present in the non-diabetic. Hypoglycemia is the most common endocrine emergency in the outpatient setting. In the Diabetes Control and Complications Trial (DCCT), an estimated 10–30% of type 1 diabetics experienced one hypoglycemic episode requiring treatment each year.³² Previous hypoglycemic episodes, lower glycosylated hemoglobin levels, and intensive therapy predicted hypoglycemic events in the study. In the first 10 years of the UK Prospective Diabetes Study (UKPDS), hypoglycemic episodes requiring third-party intervention occurred at an incidence of 1.2% for type 2 patients treated with insulin.³³ Recent studies suggest an incidence of severe hypoglycemia in type 2 diabetes approximating that of type 1 diabetes regardless of treatment.³⁴ Intensive therapy for tight control of glucose is thought to help prevent the microvascular complications of diabetes mellitus, but also increases the incidence of symptomatic hypoglycemia.

Normal physiological response to hypoglycemia

Glucose is the preferred metabolic substrate for the brain, which relies on a continuous supply to meet its energy requirements as it has no ability to store glucose. Multiple counter-regulatory mechanisms oppose insulin's action to increase peripheral uptake of glucose and lower blood glucose. The central nervous system coordinates the response to an acute

drop in systemic blood glucose through the mechanisms of glucagon, cortisol, and epinephrine to maintain glucose supply to the brain. This involves a decrease in the secretion of insulin followed by a concomitant rise in the systemic counter-regulatory hormones which increase glycogenolysis and gluconeogenesis, and also inhibit uptake by insulin-dependent tissue. In concert, these mechanisms limit glucose use by peripheral tissues and increase endogenous glucose production with resultant recovery from hypoglycemia. This process can often be disrupted by various drugs (exogenous insulin and beta-blockers) or disease states (diabetes, liver or renal failure) that can impair the counter-regulatory response and hence impair recovery from hypoglycemia. Diabetics also often have an attenuated sympathoadrenal response that masks the warning signs seen in hypoglycemia and results in an unawareness of hypoglycemia. The lack of signs and symptoms in these patients leads to a substantially increased risk of severe hypoglycemia.³⁵

Surgical treatment provides many opportunities for complications in the diabetic patient including severe hypoglycemia. Although the stress of a procedure will actually increase the counter-regulatory response and raise blood glucose, other factors may lead to hypoglycemia. Surgical treatment often requires NPO status and insulin-dependent patients require adjustment of morning insulin or oral hypoglycemic doses. In addition, maxillofacial surgery will often alter patients' eating habits for variable periods of time, increasing the risk of hypoglycemia if normal hypoglycemic medication doses are used.

Signs and symptoms of hypoglycemia

The initial symptoms of sweating, anxiety, palpitations, hunger, and tremor occur as the blood glucose level drops below 60 mg/dl. Mild cognitive dysfunction is noted in normal subjects at blood glucose levels of around 50–55 mg/dl. Older patients are more likely to have cognitive impairment than younger patients, and are less likely to have other symptoms warning them of the hypoglycemia.³⁶

More severe neurologic symptoms occur with progressive hypoglycemia. Blood glucose levels below 45–50 mg/dl can lead to lethargy and obtundation. Coma can occur at a plasma glucose concentration of about 30 mg/dl, with convulsions and ultimately death below 20 mg/dl.³⁷

Management

An initial blood glucose level should be obtained to determine and document the level of hypoglycemia. This should not delay glucose administration in any way if clinical suspicion is present. The potential detrimental effects of delayed treatment of hypoglycemia far outweigh any ill effect of hyperglycemia, so the

threshold for administering glucose should be low. For the conscious patient it is appropriate to give glucose orally via glucose tablets or glucose-containing fluids, candy, or food (Table 4.3). A reasonable initial dose is 20 g of glucose. If the patient is unable or unwilling to take oral glucose due to combativeness or unconsciousness secondary to the hypoglycemia, parenteral therapy is necessary. Intravenous dextrose, 25 g initially, is preferable. This can be given very quickly via injection of an ampule of 50% dextrose in water (D50W). With high suspicion in an unconscious patient, dextrose should be administered while the airway, breathing, and circulation phases of resuscitation are being completed. The amount of blood glucose increase from one ampule is variable and continued monitoring is important. EMS should be contacted for any patient that becomes unresponsive secondary to hypoglycemia or doesn't respond to administration of dextrose.

Fifty percent dextrose should not be given to infants or young children as it can cause venous sclerosis and lead to severe rebound hypoglycemia. Instead, for children younger than 8 years old, one should use 25% or even 10% dextrose. One can dilute the 50% dextrose in sterile water. The dose for children is 0.5–1 g/kg equating to 2–4 ml/kg of 25% dextrose.

Glucagon can also be administered to raise blood glucose. It can be administered intravenously and is usually given in 1 mg doses. If intravenous therapy is not available, 1–2 mg of glucagon can be given subcutaneously or intramuscularly. The onset of action is 10–20 minutes and peak effect is seen at 30–60 minutes. This may be repeated as needed. One milligram of glucagon has a similar effect on blood glucose as one ampule of D50W. Glucagon, however is ineffective in patients whose hypoglycemia is secondary to ethanol as glycogen stores are depleted.

The clinician must be aware that these urgent treatments raise plasma glucose concentrations only

Table 4.3 Summary of diagnosis and treatment for hypoglycemia. Adapted from: Marx J. *Rosen's Emergency Medicine: Concepts and Clinical Practice*, 6th edn. Philadelphia: Mosby Elsevier, 2006.

1. Hypoglycemia?
 - Check serum glucose; obtain sample before treatment
 - High clinical suspicion of hypoglycemia – give glucose before assessing fingerstick
2. Correct serum glucose:
 - Awake and cooperative patient – administer sugar-containing food or beverage PO. Sugar frosting can be administered sublingually
 - If patient is unable to take PO:
 - 25–75 g glucose as D50W (1–3 ampules) IV
 - Children: 0.5–1 g/kg glucose as D25W IV (2–4 ml/kg)
 - If unable to obtain IV access:
 - 1–2 mg glucagon IM or SC; may repeat every 20 minutes
 - Children: 0.025–0.1 mg/kg SC or IM; may repeat every 20 minutes

transiently. The patient's glucose concentration, as well as clinical status, should be monitored carefully after treatment. In a conscious patient that only required oral carbohydrate, one should monitor the patient for 1 hour before permitting the patient to leave the office. For the initially unresponsive patient, vital signs and blood glucose should be monitored at least every 5 minutes until medical assistance arrives. Many patients will require hospital admission for observation and continuous intravenous glucose infusion is often necessary.³⁸

Anaphylaxis

Any office that directly provides medications to patients, especially parenteral medications, needs to be prepared to deal with an anaphylactic reaction. Anaphylaxis is a true life-threatening medical emergency that is rarely anticipated. It accounts for 1500 deaths in the USA annually.³⁹ Anaphylaxis, a type I hypersensitivity reaction, results from binding of the offending antigen to preformed IgE antibodies on mast cells and basophils leading to systemic release of the immunologic mediators.

Presentation

The clinical presentation of anaphylaxis varies, but most often has cutaneous signs including urticaria and/or angioedema (Table 4.4). The presence of anaphylaxis is considered highly likely if any of the three criteria seen in Table 4.5 present within minutes to hours of administration of a medication.⁴⁰

Respiratory compromise and cardiovascular collapse are the usual cause of death in anaphylaxis.⁴¹

Table 4.4 Anaphylaxis: frequency of signs and symptoms. Adapted from: The diagnosis and management of anaphylaxis: an updated practice parameter. *J Allergy Clin Immunol* 2005;115(3 Suppl 2): S483–523.

Signs and symptoms	Frequency (%)
Cutaneous	90
Urticaria, angioedema	85–90
Flush	45–55
Pruritis, without rash	2–5
Respiratory	40–60
Upper airway edema	45–50
Dyspnea, wheeze	50–60
Rhinitis	15–20
Gastrointestinal symptoms: nausea, vomiting, diarrhea, crampy abdominal pain	25–30
Dizziness, syncope, hypotension	30–35
Other	
Headache	5–8
Susternal pain	4–6
Seizure	1–2

Up to 50% of the intravascular volume can shift to the extravascular space in a matter of 10 minutes leading to anaphylactic shock even before the onset of cutaneous findings.⁴²

The most common mediators of anaphylaxis encountered in the surgical practice include drugs, such as penicillin and aspirin (Table 4.6), and exposure to latex. The incidence of anaphylactic reactions to latex during medical procedures has increased over the last decade and has been reported to account

Table 4.5 Clinical criteria for diagnosing anaphylaxis. Adapted from: Sampson HA, *et al.* Second symposium on the definition and management of anaphylaxis: summary report – Second National Institute of Allergy and Infectious Disease/ Food Allergy and Anaphylaxis Network symposium. *J Allergy Clin Immunol*, 2006; 117(2): 391–7.

Anaphylaxis is highly likely when any one of the following is present:

1. Acute onset of illness (minutes to hours) with involvement of the skin, mucosal tissue, or both (pruritis, generalized urticaria, flushing, and/or swollen lips, tongue, or uvula) AND AT LEAST ONE OF THE FOLLOWING:
 - Respiratory involvement – dyspnea, wheezing, stridor, reduced peak-expiratory flow, hypoxemia
 - Hypotension or evidence of end-organ dysfunction – hypotonia, syncope, incontinence
2. Two or more of the following symptoms occurring rapidly (minutes to several hours) after exposure to a likely allergen for that patient:
 - Skin or mucosal involvement (pruritis, generalized urticaria, flushing, and/or swollen lips, tongue, or uvula)
 - Respiratory involvement – dyspnea, wheezing, stridor, reduced peak-expiratory flow, hypoxemia
 - Hypotension or evidence of end-organ dysfunction – hypotonia, syncope, incontinence
 - Gastrointestinal symptoms – crampy abdominal pain, vomiting
3. Hypotension after exposure to a known allergen for that patient within minutes to several hours:
 - Adults – systolic blood pressure less than 90 mmHg or 30% less than baseline
 - Infants and children – age-specific low systolic blood pressure or greater than 30% decrease in systolic blood pressure
 - 1 month to 1 year: less than 70 mmHg
 - 1–10 years: less than (70 mmHg + (2 × age))
 - 11–17 years: less than 90 mmHg

Table 4.6 Medications and anaphylaxis. Adapted from: Neugut AI, Ghatak AT, Miller RL. Anaphylaxis in the United States: an investigation into its epidemiology. *Arch Intern Med* 2001; 161(1): 15–21.

Antibiotics	Miscellaneous
Penicillin and derivatives	Aspirin
Cephalosporins	Non-steroidal anti-inflammatory drugs
Sulfonamides	Insulin
Tetracycline	Heparin
Ciprofloxacin	Protamine sulfate
Chloramphenicol	Immune globulin
Nitrofurantoin	Opiates
Vancomycin	Dextran
	Local anesthetics (esters)
	Glucocorticoids

for up to 27% of anaphylactic reactions occurring during general anesthesia.⁴³

Differential diagnosis

As the clinical presentation is variable, several disorders can share features with anaphylaxis. Vasodepressor syncope shares many features and should be considered in the differential diagnoses. However, with syncope, urticaria is absent, the patient is typically bradycardic rather than tachycardic, and respiratory symptoms are usually absent. Also, with syncope, the skin is typically cool and pale rather than flushed due to the vasodilation of anaphylaxis. Other diagnoses that can also cause abrupt and dramatic patient collapse include local anesthetic overdose, panic attack, cardiac arrest, foreign body aspiration, hypoglycemia, and seizure disorder.

Management

The initial signs and symptoms of allergy such as urticaria, erythema, pruritis, or wheezing may occur at any time after administration of a medication or local anesthetic. Allergic reactions occur as a continuum and can progress to the life-threatening respiratory or cardiovascular collapse very quickly.⁴⁴ The clinician should stop the procedure and be prepared for progression at any hint of allergy.

As with other emergencies, the airway, breathing, and circulation and level of consciousness should be assessed. In a suspected systemic allergy, however, administration of epinephrine should not be delayed. EMS should be alerted by a staff member. Every office emergency kit should include a preloaded syringe of epinephrine. It should be administered intramuscularly or subcutaneously as a 1:1000 dilution, 0.2–0.5 ml (0.2–0.5 mg) in adults, or 0.01 mg/kg in children usually into the lateral thigh, upper arm, or even intra- or sublingually.⁸ Gently massaging the site of administration may facilitate absorption. The dose may be repeated every 5 minutes as clinically indicated.

The patient should be placed in the Trendelenberg position to maximize cerebral blood flow. Establish and maintain an airway through head tilt, jaw thrust, or placement of an oral or nasal airway and provide supplemental oxygen. Laryngeal edema may also occur during an anaphylactic reaction and may occlude the airway, necessitating emergency cricothyroidotomy at any time during the management of the patient. Continuously monitor the patient's oxygen saturation, blood pressure and heart rate. Intravenous access should be obtained and 2 l of normal saline should be administered to adults and 20 ml/kg for children.

Once clinical improvement is seen after epinephrine and fluid are administered, other treatments can be administered. Antihistamines can dramatically improve symptoms of an allergic response but should

never be administered alone in anaphylaxis. Diphenhydramine can be administered intramuscularly or intravenously at 1–2 mg/kg up to 50 mg. Also, albuterol can be administered for continued bronchospasm. Corticosteroids are indicated to prevent recurrent or protracted anaphylaxis and can be administered by the surgeon or upon arrival at the emergency department. There is no established dose or dosage of choice, however hydrocortisone 5 mg/kg up to 250 mg has been recommended.⁴⁴

All patients with anaphylaxis should be taken to an emergency department for monitoring and observation as anaphylaxis can recur and present biphasically.⁴⁵

Seizures

Seizures are often perceived to the lay-person as a life-threatening emergency. However, seizures in isolation can be harmless as long as proper care is taken to prevent injury to the patient. They are the result of excessive activation of neurons in either the cerebral cortex or deep limbic system causing abnormal neurologic functioning.⁴⁶ Approximately 6% of the US population experience at least one afebrile seizure during their lifetime and this rate is considered to be higher in developing countries.⁴⁶

Seizures are classified into partial and generalized seizures. Partial seizures are restricted to discrete regions of the cerebral cortex. These are further classified into simple partial, if consciousness is maintained, and complex partial, if consciousness is lost. Generalized seizures result from activity in both cerebral hemispheres. These include absence, myoclonic, and the grand mal seizure or tonic-clonic seizure, the most common form of seizure disorder. Seizures can also be classified as primary or secondary. Secondary seizures are the result of an injury or illness affecting the central nervous system. Examples include traumatic brain injury, metabolic disturbances, tumors and infections of the nervous system.^{47,48} Seizures that have no known etiology are known as primary seizures.

Management

If a patient develops seizure-like activity in the office, the first thing to do is to confirm that it is actually a seizure. Syncope can be followed by abnormal movements resembling ictal activity. These movements, however, are usually brief and less forceful than tonic-clonic activity.

Patients who develop an aura or any sort of ictal activity should be reclined to the supine position or placed on the floor if not in the surgical chair, to minimize injury. Consideration to calling EMS should be made based on the history of the patient. As the patient is seizing, the surgeon should institute basic life support. The airway should be cleared of any

objects and a chin lift can help open the airway. The oral cavity can be suctioned with a soft suction catheter to prevent facial injury. A nasopharyngeal airway is preferred over an oropharyngeal airway as it can induce vomiting. A pulse should be checked to ensure that the convulsions are not secondary to cerebral hypoxia due to hypoperfusion. Oxygen should be administered and oxygen saturation monitored. Any objects near the patient should be moved by a member of the team to prevent injury. In most cases of tonic-clonic seizure activity, the duration is often less than 2 minutes and does not require administration of anticonvulsants. One must, however, continue to manage the generalized depression of the cardiovascular and respiratory systems in the postictal phase through close monitoring of vital signs and basic life support principles. A brief and limited seizure is easily managed and discharge home is based on whether this patient has a known seizure disorder or if this represents a new seizure. A first seizure should always be referred to the emergency department for consideration of inpatient work-up.

Status epilepticus

The true office life-threatening emergency is the patient who continues seizing and develops status epilepticus. Definitions vary, but it refers to seizures that last longer than 5–10 minutes or frequent seizures without return to baseline status between ictal states. The incidence in epileptic patients is around 5% and most likely results from failure of the patient to take anti-epileptic medications.⁴⁹ EMS should be called immediately if one suspects that the seizure is at all unremitting.

While continuing basic life support, one must rule out underlying, correctable causes of seizure activity. Attempts should be made to secure intravenous access if not already obtained. Hypoglycemia is a common cause of seizure activity and an ampule of 50% dextrose should be administered IV to rule out low blood glucose as a cause.

The first-line drugs for aborting seizure activity of all patients are benzodiazepines. Lorazepam 0.02–0.03 mg/kg should be administered IV.⁵⁰ One should allow a minute to assess effect. Diazepam 0.1 mg/kg IV or midazolam 0.05 mg/kg IV may be substituted if lorazepam is not available. If seizures continue after an initial dose, additional doses of lorazepam should be given at a maximum rate of 2 mg/min up to a total of 20 mg (0.1 mg/kg). If intravenous access cannot be achieved, diazepam can be given rectally (0.5–1.0 mg/kg), endotracheally, or intraosseously (0.2–0.5 mg/kg).⁵¹ Also, midazolam can be given intramuscularly (0.2 mg/kg).

If benzodiazepines do not terminate the seizure, the airway should be re-evaluated and intubation performed, particularly if the patient develops hypoxemia. Second-line agents should be administered, including phenytoin or its prodrug, phospho-

nytoin. Phenytoin is administered at 20 mg/kg as an infusion of 25–50 mg/min, and vitals signs should be monitored closely due to its hypotensive effects.⁵² Phosphenytoin causes much less hypotension, but is more expensive and should be administered at 15–20 mg/kg as an infusion of 100–150 mg/min. Both of these drugs can also be given intramuscularly.⁵³ Refractory status epilepticus after giving these drugs indicates a grave prognosis and any further management requires an intensive care setting. The practitioner must continue to closely monitor the patient and administer basic life support while awaiting EMS.

Seizures can and do happen in both epileptic and non-epileptic patients. All practicing oral and maxillofacial surgeons should be prepared to deal with them at any time.

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Chapter 5

Local Anesthesia

John Gerard Meechan

This chapter explains the current understanding of the mechanism of action of local anesthetics. It describes the techniques and materials that are used for local anesthesia in and around the jaws. In addition the localized and systemic adverse effects of local anesthesia in this region are considered.

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surgery. It may be the sole method of pain control, used in combination with sedation, or as a supplementary technique in conjunction with general anesthesia. A number of different techniques and drugs are available for use in and around the jaws and these are described below.

Mode of action of local anesthetics

Local anesthetic drugs achieve their action at the voltage-gated sodium channel. In simple terms they inhibit sodium entry into nerve cells. As the entry of sodium into the nerve cell during the firing cycle is the chief driver producing depolarization, blockade of sodium transfer causes inhibition of neural activity.

Knowledge of the structure of the sodium channel is essential for an understanding of the action of local anesthetics. The structure of the sodium channel has been well characterized.¹ It is composed of three subunits known as alpha, beta₁ and beta₂. The pore through which sodium enters is contained in the alpha subunit and a diagrammatic representation of this component is shown in Fig. 5.1. The alpha subunit is made up of four similar domains (I–IV) each of which contains six protein segments (S1–S6). At rest the positively charged S4 projects into the channel preventing sodium entry. During depolarization S4 moves away from the pore in a movement called the

Introduction

Local anesthesia is an important method of pain control for many procedures in oral and maxillofacial

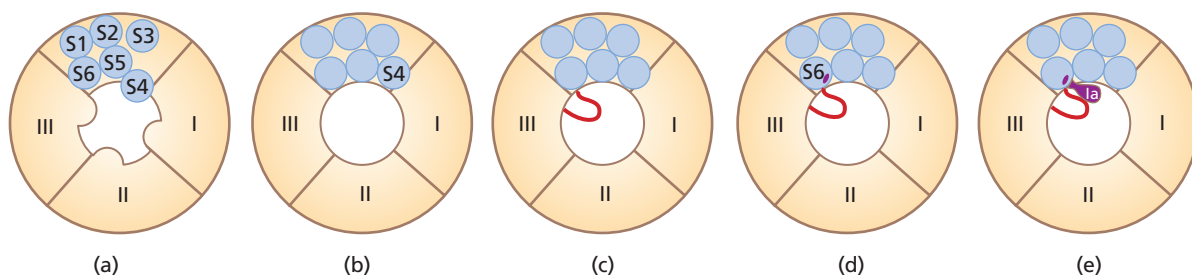


Fig. 5.1 The alpha subunit of the sodium channel: (a) in the resting state with S4 segments in the body of the channel; (b) in the firing configuration with S4 segments in wall of the channel; (c) in the refractory orientation with a protein loop extended into the channel; (d) exposure of binding site for local anesthetics in segment S6; (e) binding of local anesthetic maintaining refractory orientation.

sliding helix. This maneuver opens up the channel, permitting sodium entry. During the refractory period a protein link between domains III and IV projects into the channel producing a new blockade to sodium entry. When it projects into the channel this loop exposes a site on segment 6 of domain IV to which local anesthetics bind.² The binding of the local anesthetic then maintains the loop in its blocking position.

It should be pointed out that the sodium channel is not a singular structure and that at least nine different variations have been identified.³ In theory this means that very selective local anesthetics could be developed. At present the local anesthetics used clinically are not specific for peripheral sensory nerves and can affect transmission in any excitable tissue such as motor nerves, central nervous, and cardiovascular tissue. This means they are capable of producing unwanted effects and these are discussed later.

Techniques of local anesthesia for oral and maxillofacial surgery

There are a number of different techniques that can provide local anesthesia in and around the mouth and jaws. These include methods that are used elsewhere in the body such as topical anesthesia, infiltration, and regional block techniques; however there are methods that are unique to the mouth and jaws such as intraosseous, intraligamentary, and intrapulpal injections.

Topical anesthesia

Topical anesthesia can be useful when applied to the oral mucosa. It may be employed prior to local anesthetic injections in the mouth to lessen the discomfort of needle penetration. This can be effective prior to infiltration anesthesia when the topical agent has been placed on reflected mucosa; however application to keratinized tissue such as palatal mucosa is not so useful and there is no evidence that topical application lessens the discomfort of regional block techniques.

Topical anesthetics for intraoral use are available in a number of formulations including creams, ointments and sprays. The local anesthetic agents most commonly used as topical anesthetics in the mouth are lidocaine and benzocaine. Oraqix[®] is a combination of lidocaine and prilocaine specifically designed for intraoral topical anesthesia.

Advances in topical anesthesia (for example the development of Oraqix[®] and incorporation of drugs into liposomes) have meant that some intraoral soft tissue procedures can be performed using topical anesthesia as the sole agent; however anesthesia of the teeth and jaws is not at present possible by this method.

Infiltration anesthesia

Infiltration anesthesia is useful in providing localized skin and mucosal anesthesia and can also be used to provide anesthesia for some teeth and part of the jaws. It is the technique of choice in the maxilla for dental pulpal anesthesia and can also be used for this purpose in the mandible of children for anesthesia of the deciduous dentition. It can be successful in the incisor teeth in the adult mandible and there is increasing evidence that some formulations (see below) can be effective when infiltrated in the molar region of the mandible.

When used intraorally, access to the point of needle penetration is easiest when the patient has the mouth only partly open. The technique is performed by inserting the needle through reflected mucosa at the depth of the buccal sulcus; if bone is contacted the needle should be withdrawn slightly so that it is supraperiosteal. A supraperiosteal location is recommended as injection below the periosteum is painful at the time of injection and may cause postinjection discomfort. Following aspiration 1.0–2.0 ml of solution is deposited at a rate of 30 s/ml. This rate is slower than many practitioners use. Slow injection has a number of advantages. It reduces discomfort and increases success. In addition a slow rate of injection may lessen the effects of systemic problems (see below).

This method allows about 45 minutes of anesthesia of the dental pulps when a solution containing a vasoconstrictor (such as lidocaine with epinephrine) is used; soft tissue anesthesia is longer and the patient may have subjective anesthesia of the soft tissues for 1.5–2 hours.

Regional block anesthesia

Several regional block techniques are described to anesthetize structures in the jaws.

Mandibular anesthesia

There are a number of methods used to anesthetize the lower jaw, teeth and associated structures and these are discussed below. It should be remembered that midline structures (such as lower central incisor teeth) often receive bilateral supply; therefore these may not be satisfactorily anesthetized by a single regional nerve block.

Inferior alveolar nerve block (Halstead technique)

This method anesthetizes the teeth and bone on one side of the mandible along with the soft tissues on the buccal aspect anterior to the mental foramen. The Halstead method achieves its effect by deposition of the solution in the pterygo-temporal space on the medial aspect of the mandibular ramus, specifically in the region of the mandibular foramen. When successful this technique anesthetizes the inferior

alveolar nerve, which supplies the teeth on the same side of the mandible, the bone of the mandible to the midline and the soft tissues of the lower lip to the midline as well as the reflected and attached gingivae from the premolar teeth to the midline. In addition this injection usually anesthetizes the lingual nerve that supplies the anterior two thirds of the tongue on one side.

The technique is illustrated in Fig. 5.2. The patient has the mouth open wide and the operator places the thumb of the non-syringe hand in the mouth on the coronoid notch of the mandible; the index finger is extraoral at the same height on the posterior border of the ramus. The syringe is advanced to the point of needle penetration across the lower premolar teeth of the opposite side. The point of penetration of the needle is between the internal oblique ridge of the mandible (which was palpated by the operator's thumb before resting on the coronoid notch) and the pterygomandibular raphe (which is visible). The height of needle penetration is halfway up the operator's thumb nail. A long 35mm needle no narrower than 27 gauge is used and advanced until bone is contacted; this is usually around 25mm of needle insertion. After contacting bone the needle is withdrawn slightly, aspiration performed and 1.5–2.0ml of solution deposited slowly.

Two common problems with this method are either contacting bone too soon (after 5–10mm of insertion) or failure to touch bone. The former is corrected by withdrawing the needle away from the bone, but not completely out of mucosa, and swinging the barrel of the syringe over the mandibular teeth of the side being anesthetized. The needle is

then advanced about 25mm and swung back again over the opposite premolar teeth; the rest of the injection is completed as described above. This is a modification of the indirect method of performing an inferior alveolar nerve block. If bone is not contacted after about 30mm of insertion the needle should be withdrawn until about 15mm is still in tissue and the syringe swung over the molar teeth of the side not being injected and then advanced until bone is contacted. Failure to contact bone may lead to injection into the parotid gland, leading to hemifacial paresis (see below).

When a vasoconstrictor solution such as lidocaine with epinephrine is used this technique will anesthetize the hard tissues including the teeth for around 45 minutes; however subjective soft tissue numbness may be apparent for up to 3 hours. When the so-called long-acting solutions are employed anesthesia of the teeth can last for 6–8 hours.

Gow-Gates technique

In addition to the anesthesia of the inferior alveolar nerve this method⁴ may anesthetize the lingual, long buccal, mylohyoid and auriculotemporal nerves. This may be of value in countering accessory nerve supply to the teeth and jaw. This approach deposits solution more superiorly than the Halstead technique and that is the reason why more branches of the mandibular nerve are affected by this injection. The target is the mandibular condyle (Fig. 5.3). As with the Halstead approach the patient has the mouth open wide. The syringe is advanced in a plane parallel to a line visualized between the corner of the mouth and the inter-tragal notch. The syringe, fitted with a long needle, is introduced into the mouth across the maxillary canine tooth of the opposite side to that being injected and advanced across the palatal cusps of the maxillary second molar on the injected side. The needle is advanced in this direction until the bone of the condyle is contacted, withdrawn slightly and, after aspiration, 2.0ml of solution is deposited. The patient should maintain their mouth in the open position for as long as possible after injection. This method appears to be more successful in anesthetizing teeth than the Halstead approach. This may be the result of combating nerves other than the inferior alveolar nerve that might supply the teeth.



Fig. 5.2 The position of the needle during the Halstead approach to the inferior alveolar nerve. Reproduced with permission, © Quintessence Publishing Co Ltd.



Fig. 5.3 Syringe position for the Gow-Gates mandibular block. Reproduced with permission, © Quintessence Publishing Co Ltd.



Akinosi Vazirani technique

This technique⁵ also anesthetizes the lingual and mylohyoid nerves and on occasion the long buccal nerve. This technique (Fig. 5.4) differs from the methods described above in two respects. Firstly, it is administered with the patient's mouth closed and so is useful in individuals who cannot fully open their mouth and is helpful in individuals with large or uncontrollable tongues when it is difficult to get access to the insertion point of a Halstead block. Secondly, there is no bony end-point for needle insertion. As is normal for all of the methods used to anesthetize the mandibular nerve, a long needle no narrower than 27 gauge is required. The syringe is introduced intraorally in the buccal sulcus along a plane level with the mucogingival junction of the maxillary mucosa. It is advanced at this level towards the posterior aspect of the maxilla until the hub of the needle is adjacent to the distal surface of the maxillary second molar. At this point the needle will have entered the mucosa and reached the correct depth of insertion. Aspiration is performed and 2.0ml of solution injected slowly. If the anterior aspect of the mandibular ramus is contacted the barrel of the syringe can be swung more laterally or the patient instructed to move their lower jaw over to the side being injected. This method anesthetizes the inferior alveolar nerve, lingual nerve, nerve to mylohyoid, and occasionally the long buccal nerve.

Mental and incisive nerve block

This injection anesthetizes the teeth and jaw from the premolars anteriorly as well as the soft tissues of the lower lip and chin to the midline on one side. Anesthesia of the first molar tooth is achieved in some cases. It relies on the deposition of solution around the mental nerve as it exits the mental foramen and the entry of solution into the foramen to block the incisive branch that supplies the lower anterior teeth on the same side. The usual method is intraoral although an extraoral approach may also be used. The needle is inserted at the depth of the buccal sulcus between the premolar teeth and advanced to a zone below the premolar apices. Around 1.5ml of solution should be deposited and after injection the tissues should be massaged to encourage entry of solution into the mental foramen.



Fig. 5.4 The position of the syringe for the Akinosi-Vazirani mandibular block. Reproduced with permission, © Quintessence Publishing Co Ltd.

Long buccal nerve block

If the long buccal nerve has not been anesthetized by the methods used above (for example if the Halstead technique was used) then this nerve must be anesthetized separately if required. The area supplied is the buccal gingivae and mucosa and part of the cheek in the mandibular molar region. The nerve can be anesthetized by either buccal infiltration in the zone of interest or by a regional block. The regional block is achieved by depositing 0.5ml of solution in the region of the coronoid notch of the mandible (the point at which the thumb rests during the Halstead technique).

Extraoral approach to the mandibular nerve

The techniques described above rely upon intraoral approaches to the branches of the mandibular nerve, although it was mentioned that the mental nerve may be blocked from outside of the mouth. It is possible to anesthetize the mandibular nerve using an extraoral method.

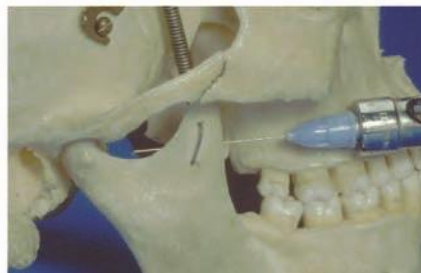
This technique approaches the foramen ovale via the sigmoid notch of the mandibular ramus. The site of insertion is the most concave point of the lower border of the zygomatic arch. A long needle is introduced at 90° to the skin and the sagittal plane and maintains contact with the lower border of the zygomatic arch. The needle is advanced until the bone of the lateral pterygoid plate is contacted. The depth of insertion of the needle at the point of bony contact is noted. This is important as it establishes the correct depth for the final injection. The needle is now withdrawn almost fully but leaving the tip under the skin. It is then redirected at an angle of 60° to the sagittal plane and advanced and this time will pass just behind the pterygoid plates. The final position for deposition of solution is no more than 4mm deeper than that established with the initial penetration to the pterygoid plates.

Maxillary anesthesia

Several regional block techniques are used in the maxilla. These are described below.

Maxillary nerve block

There are intraoral and extraoral approaches to the maxillary nerve block. The intraoral methods are the



tuberosity approach and the greater palatine foramen approach. The maxillary nerve block anesthetizes the teeth and bone of the maxilla on one side together with the buccal and palatal mucosa, the skin and mucosa of the upper lip, the lower eyelid, and the lateral aspect of the nose.

Tuberosity approach

This is administered by depositing solution high in the buccal sulcus in the plane of the distal surface of the maxillary second molar tooth. The needle is advanced at an angle of 45° superiorly, posteriorly and medially to a depth of 30mm, at which point 2.0ml of solution is deposited following aspiration.

Greater palatine foramen approach

This technique involves inserting a needle into the pterygopalatine fossa via the greater palatine foramen. The patient has the mouth open wide and the greater palatine foramen is identified as a depression medial to the distal surface of the second maxillary molar tooth. The needle is inserted into the greater palatine foramen and advanced at an angle of 45° superiorly and posteriorly to a depth of 30mm. At this point 2.0ml of solution is deposited.

Extraoral approach

This is similar to that described above for the extraoral approach to the foramen ovale. This time the target is the foramen rotundum. The point of insertion is identical to that described for the extraoral mandibular nerve block. Similarly, the needle is advanced until the lateral pterygoid plate is contacted. The depth of insertion is noted. The needle is withdrawn and redirected towards the pterygopalatine fossa. This is achieved by pointing the needle 10° more superiorly and 15° more anteriorly until the depth of insertion determined by the original needle insertion is reached. This is the point of delivery of solution.

Posterior superior alveolar nerve block

This injection anesthetizes the maxillary molar teeth, associated bone and buccal gingivae. In those individuals with a middle superior alveolar nerve the first molar will not be satisfactorily anesthetized with this method as the mesiobuccal root of this tooth is supplied by that nerve (see below). The technique is identical to that described for the tuberosity approach to the maxillary nerve except that needle penetration is only 20mm.

Maxillary molar nerve block

This is an adaptation of the posterior superior alveolar nerve block.⁶ It is designed to anesthetize the maxillary molar teeth by countering any accessory supply to the mesio-buccal pulp of the first molar from the middle superior alveolar nerve. During the injection the patient has the mouth half closed. The operator's finger palpates the zygomatic process of the maxilla intraorally and advances the finger poste-

riorly towards the maxillary tuberosity. The needle penetrates mucosa high in the buccal sulcus between the finger and the distal surface of the zygomatic process and advanced about 10mm into the space above the buccinator attachment. At this point aspiration is performed and 2.0ml of local anesthetic solution is injected. Finger pressure is maintained throughout the injection. Following injection the patient is asked to close the mouth slightly and the solution, which has created a swelling above the buccinator, is massaged in a superior, medial and distal direction towards the posterior superior alveolar foramen.

Middle superior alveolar nerve block

This injection anesthetizes the premolar pulps as well as the mesio-buccal pulp of the maxillary first permanent molar tooth. The technique is performed by delivering a buccal infiltration at the apex of the second premolar tooth.

Anterior superior alveolar nerve block

The anterior superior alveolar nerve supplies the maxillary incisor and canine teeth on one side of the jaw. It can be anesthetized either by an infraorbital nerve block (described below) or by a buccal infiltration in the region of the apex of the canine.

Infraorbital nerve block

The infraorbital nerve can be approached either from the intraoral or the extraoral sides. The intraoral approach (Fig. 5.5) involves inserting a long needle high into the buccal sulcus between the premolar teeth and advancing towards the infraorbital foramen, which is being palpated extraorally by the operator's non-syringe hand. At this point 1–1.5ml of solution is deposited following aspiration.

This injection anesthetizes the teeth and associated bone from the second premolar to the central incisor (although the latter may obtain some collateral supply from the opposite side). In addition the gingivae adjacent to these teeth and the mucosal and skin surfaces of one half of the upper lip and part of the skin on the lateral aspect of the nose are affected.

Greater palatine nerve block

This method anesthetizes the soft tissues of the palate from the foramen anteriorly to the canine region. The nerve can be anesthetized at any point by a palatal



Fig. 5.5 The intraoral approach to the infraorbital nerve block. Reproduced with permission, © Quintessence Publishing Co Ltd.

infiltration; however the true block involves injection of 0.5 ml of solution in the region of the greater palatine foramen. The soft tissues of the hard palate up to the canine region on one side will be anesthetized. In addition this technique can be effective in countering any palatal supply to the maxillary teeth. Duration of anesthesia is not as long as with mandibular blocks. Indeed both palatal infiltration and blocks provide a similar duration of around 45 minutes of soft tissue anesthesia.

Nasopalatine nerve block

This method anesthetizes the tissues of the hard palate adjacent to the incisor teeth bilaterally. It is performed by injecting 0.2–0.5 ml of solution adjacent to the incisive papilla.

Anterior middle superior alveolar nerve block

This technique relies on the slow delivery of solution and is best achieved using a computer-controlled local anesthetic delivery device.⁷ The method works by entry of local anesthetic solution into the cancellous bone of the maxilla via multiple foramina in the hard palate. It anesthetizes the soft tissues of the palate and the teeth from the premolars anteriorly on one side. The needle is inserted halfway between the palatal midline and the premolar palatal gingival margin and 1.0 ml of solution is injected very slowly.

Palatal anterior superior alveolar nerve block

This technique achieves anesthesia of the teeth and anterior hard palate mucosa by the deposition of around 1.0 ml of local anesthetic solution into the nasopalatine foramen and thence into the cancellous bone of the anterior maxilla.⁸ As was the case with the anterior middle superior alveolar nerve block injection, the technique is most readily achieved using a computerized local anesthetic delivery device as a slow rate of delivery is required for patient tolerance. This technique can achieve pulpal anesthesia of the canine and incisor teeth bilaterally and on occasion may also anesthetize the premolar teeth.

Supplementary intraoral techniques of local anesthesia

Intraosseous anesthesia

This method (Fig. 5.6) can be used in either jaw and, although it can be employed as a primary technique, it is normally reserved as a supplementary injection when conventional measures fail to produce adequate anesthesia of the pulps of teeth. The method is as follows. Firstly the buccal gingiva around the tooth of interest is anesthetized by a small-dose (0.2–0.5 ml) buccal infiltration. A perforation through soft tissue and cortical bone is made using a perforator matched to the needle (these are provided in customized systems) or with a small round dental bur. The point of penetration is 2 mm below the intersection of two

imaginary lines. These are: a line joining the lowest part of the buccal gingival margin of the tooth of interest and its posterior neighbour; and a line at 90° to the former line that bisects the interdental papilla. If the point 2 mm below the intersection of these two lines is in reflected mucosa a point more coronal in attached gingiva is chosen. Once the perforation has reached cancellous bone the perforator or bur is replaced with a needle and 1.0 ml of solution injected slowly intraosseously. As intraosseous deposition of solution is equivalent to intravenous entry, slow deposition and dose limitation are important with this technique.

Intraligamentary (periodontal ligament) anesthesia

This is regarded as a specific version of an intraosseous technique (Fig. 5.7). Like the method described above it can be used as a primary method of anesthesia but it is considered a supplementary method when tooth pulpal anesthesia is problematic. There are specialized syringes designed specifically for this method; however it is equally effective with conventional delivery systems. Normally a 30 gauge needle is used. It is inserted to the point of maximum pene-



Fig. 5.6 The intraosseous injection showing preparation of the needle site with a perforator and then needle insertion. Reproduced with permission, © Quintessence Publishing Co Ltd.



Fig. 5.7 The intraligamentary injection. Reproduced with permission, © Quintessence Publishing Co Ltd.

tration at the mesio-buccal aspect of the root of the tooth (each root for a multirooted tooth). The needle becomes wedged at the crest of the interdental bone and will not traverse far into the periodontal space. Delivery of solution (0.2ml per root) is performed slowly with controlled pressure. The method obtains anesthesia by entry of the local anesthetic solution into the cancellous space of the bone via the perforations in the cribriform plate of the socket. As the number of these perforations is low in anterior mandibular sockets the technique is not very successful in lower incisor teeth.

Intrapulpal anesthesia

This method (Fig. 5.8) has limited indications, as exposure of the tooth pulp is essential. It is unique among the techniques described here as the presence of a local anesthetic solution is not essential for success: saline has been shown to be as effective.⁹ The key to success is delivery of solution under pressure. This can be achieved with a needle that fits tightly into the pulpal exposure, by advancement of the needle into the pulp canal until it is wedged or by placing an obturator such as a cotton wool bud around the needle during injection. This is the most localized form of anesthesia described here and in theory could be used to anesthetize one pulp canal of a multirooted tooth.

Local anesthetic drugs

Various drugs are available for local anesthesia in and around the jaws. These can be classified in a number of ways. The main categorization is by chemical structure. Local anesthetics are divided into two groups by their composition. These are the esters and the amides. The local anesthetics in current use as injectable agents are amides. The ester procaine is only used in those patients who are proven to be allergic to amides and such cases are very rare. Topical anesthetics such as benzocaine and tetracaine (amethocaine) are esters. There are two major differences between esters and amides; these relate to metabolism and the production of allergy. Esters are



Fig. 5.8 An intrapulpal injection in a molar tooth. Reproduced with permission, © Quintessence Publishing Co Ltd.

metabolized in plasma. Most amides primarily undergo hepatic metabolism, although prilocaine also undergoes some breakdown in the lungs. Articaine is an exception: although it is an amide, its primary metabolism occurs in the plasma.

In addition to categorizing local anesthetics by their chemical structure they can also be classified with respect to their duration of action into conventional and long-acting agents. Conventional agents may provide better operative anesthesia; however long-acting agents may be preferred for postoperative pain control, especially in combination with general anesthesia. In order to achieve their long-acting effect such agents must be employed as regional blocks.

Lidocaine

Lidocaine is the gold standard drug to which all others are compared. When used as a plain solution in concentrations up to 2% it provides short-lasting soft tissue anesthesia. Such a formulation does not provide acceptable anesthesia of the dental pulps. When a vasoconstrictor is added to 2% lidocaine then satisfactory anesthesia is provided for the teeth. The vasoconstrictor most commonly employed is epinephrine (adrenaline) usually in the concentration range of 1:200 000 (5 µg/ml) to 1:80 000 (12.5 µg/ml).

Mepivacaine

Mepivacaine, when injected at a concentration of 2% in combination with 1:100 000 epinephrine, provides similar anesthesia to 2% lidocaine with epinephrine. It is also provided as a plain 3% solution and this provides better anesthesia than 2% lidocaine when a vasoconstrictor-free solution is required.

Prilocaine

Prilocaine is used as a plain 4% solution or as a 3% formulation in combination with the vasoconstrictor felypressin (a synthetic analog of vasopressin). The 3% formulation is a useful alternative to 2% lidocaine with epinephrine if an epinephrine-free solution is indicated.

Articaine

As mentioned earlier articaine is unique among the amides in that initial metabolism occurs in plasma and thus, having a shorter plasma half-life, it is safer systemically than the other amides. This means that it can be used in higher concentration and so most articaine formulations are 4% with epinephrine in either 1:100 000 or 1:200 000 concentrations. There are some concerns, however, that local anesthetic drugs used in high concentration may increase the chances of localized toxicity leading to long-lasting anesthesia, paraesthesia or dysaesthesia when used for regional

blocks (see later). An advantage of the 4% articaine solution is that it is superior to 2% lidocaine solutions in providing anesthesia of the mandibular teeth following infiltration in the adult mandible.¹⁰ There is some evidence that buccal infiltration with 4% articaine is as effective as inferior alveolar nerve blockade with 2% lidocaine in providing anesthesia of the adult mandibular teeth.¹¹

Etidocaine

Etidocaine in a concentration of 1.5% with 1:200 000 epinephrine has been used in oral surgery. It has a longer duration of action than 2% lidocaine with epinephrine 1:100 000 when used as a regional block but is not as effective as lidocaine with epinephrine when used for infiltration anesthesia.¹²

Bupivacaine

Bupivacaine is a long-lasting local anesthetic. When used as a supplementary intraoral injection during general anesthesia this drug has been shown to reduce the number of analgesics required for postoperative pain control following oral surgery.¹³ It is presented in a number of formulations ranging from 0.25–0.75% with and without epinephrine (usually 1:200 000).

Levobupivacaine

This is a single isomer of bupivacaine and has the advantage of being less cardiotoxic. It has been shown to be as effective a local anesthetic as bupivacaine and, like the latter drug, its use as an intraoral injection during general anesthesia can reduce postoperative analgesic requirements following oral surgery. It is available in concentrations ranging from 0.25–0.75%.

Ropivacaine

Like levobupivacaine this is a single isomer with reduced cardiotoxicity compared to bupivacaine. There is a suggestion that it may be as effective with and without a vasoconstrictor; when used intraorally, however, the presence of epinephrine increases efficacy.¹⁴ It is available in concentrations ranging from 0.2–1.0%

Complications of local anesthesia in the orofacial region

In addition to failure of local anesthesia there are a number of complications that can occur following the use of local anesthetics in the orofacial region. These can be separated into localized and systemic complications.

Localized complications

Localized complications can arise either as a result of physical damage from the needle or chemically as a result of the local anesthetic.

Nerve damage

Nerve damage can occur following regional block techniques. The nerve that is most commonly affected is the lingual nerve. This can be due to physical trauma from the needle, which can result in altered sensation lasting for a few weeks. Normal sensation should recover in most cases following such trauma. The deposition of local anesthetic into the nerve bundle should be avoided as this can cause damage, both as a result of the physical disruption and chemically.

As mentioned above, the concentration of the local anesthetic has been implicated in the production of long-lasting paresthesias. Certainly *in vitro* the survival of nerve cells decreases as the local anesthetic concentration rises.¹⁵ There is some circumstantial evidence from both North America¹⁶ and Europe¹⁷ that local anesthetics in the concentration of 4% are associated with more paresthesias (particularly of the lingual nerve) than 2% and 3% solutions, although other investigations have failed to confirm this.¹⁸

If nerve function does not return to normal within a few weeks the prognosis is poor. Unfortunately surgical repair is not as effective compared to the treatment of surgically damaged nerves.¹⁹

Motor nerve paralysis

Local anesthetics are not specific for peripheral sensory nerves and motor function can be affected. Facial nerve function may be affected if solution is injected into the substance of the parotid gland. Fortunately any paralysis produced is temporary. This may occur during the delivery of mandibular block techniques but can be avoided when using the Halstead method if bone is palpated before solution is delivered. This should ensure that the needle is not advanced beyond the posterior border of the mandibular ramus. The most serious consequence is loss of the blink reflex and thus the patient must have some form of eye protection until the effect wears off.

Trismus

Mandibular block techniques involve deep penetration of the needle and this may cause minor bleeding. If this occurs in the medial pterygoid muscle it can lead to muscle spasm and the inability to fully open the mouth. Although this problem does resolve it may take a few weeks to do so.

Intravascular injection

Intravascular injection can cause both localized and systemic (see below) complications. The use of an

aspirating technique should reduce the chances of intravascular injection. Localized problems may result from intra-arterial injection. It is more difficult to penetrate an artery compared to a vein, however it is not impossible and a variety of adverse effects may ensue. Entry or contact of an artery is painful and arteriospasm may produce localized vascular shut-down, which appears as blanching. Injecting local anesthetic into an artery that has an intracranial course can result in a number of spectacular sequelae. Vision may be affected. Both diplopia and loss of vision have been recorded following intraoral injections. Hearing loss has also been noted. The most dramatic effect reported is hemiparesis. This could result from the phenomenon known as reverse carotid flow. This occurs when local anesthetic is injected into a branch of the external carotid artery under pressure. This can send some of the agent to the carotid bifurcation resulting in transport of some anesthetic intracranially via the internal carotid. Such an effect has been demonstrated in animals following injection into the facial artery. This rare complication can be reduced by aspirating and injecting slowly.

Systemic complications

The following systemic complications caused by the injected agent can occur after the injection of local anesthetics: allergy, infection, toxicity, and drug interactions. These are discussed below. Other systemic complications such as syncope are unrelated to the injected solution.

Allergy

Allergic reaction to the amide group of local anesthetics is extremely rare. Many individuals who claim to be allergic are found not to be so after formal testing. Nevertheless any individual complaining of non-localized swelling, rash, or breathing difficulty following injection should be tested, as the full range of allergic reactions, including anaphylaxis, has been reported after intraoral injections. Ester allergy is more common. Some local anesthetic cartridges (carpules) contain latex in their plungers or diaphragms and these should be avoided in patients with severe latex allergy. Older local anesthetic formulations contained preservatives related to paraben and this could cause allergic reactions in susceptible patients; however most modern formulations are preservative-free.

Infection

Sensible precautions are essential during the administration of local anesthesia to prevent contaminating the patient and to avoid transfer of infection between operator and patient. The use of safety-type syringes, which eliminate resheathing of used needles, has

been shown to reduce the chances of needle-stick injury. The use of such equipment is recommended.

Toxicity

As many local anesthetics contain an anesthetic and a vasoconstrictor it is, in theory, possible to have a toxic reaction to either component; however as a result of their relative concentrations, a patient is more likely to suffer a toxic reaction to the anesthetic. The systems most susceptible to toxicity are the central nervous and cardiovascular systems. Most serious cases of overdose are the result of central nervous system effects. A toxic reaction to the anesthetic initially manifests as overexcitation, such as excessive talking and tremors. The later stages are signs of central nervous system depression ultimately leading to unconsciousness, and fatality can result from respiratory depression. The management is to stop the injection, monitor vital signs, and provide basic life support.

Overdose of epinephrine presents as anxiety, headache, and palpitations. If this occurs the patient should be sat upright and administered oxygen.

Systemic toxicity from local anesthetics can be the result of intravascular injection, use of excessive doses, or inability of the patient to metabolize the drug. Intravascular injection should be avoided by use of an aspirating technique. This is essential. Some workers report positive aspirates in over 20% of mandibular block injections.

Overdose of local anesthesia can occur, particularly in children. The maximum dose is related to the patient weight. This means that overdose is more likely to occur in children. Table 5.1 gives the recommended maximum doses for healthy individuals for the local anesthetics discussed in this chapter.

Table 5.1 Maximum recommended doses for healthy individuals for local anesthetics.

Drug	Maximum recommended dose (mg/kg)
Lidocaine	4.4
Mepivacaine	4.4
Prilocaine	6.0
Articaine	7.0
Etidocaine	8.0
Bupivacaine	1.3
Levobupivacaine	1.3
Ropivacaine	1.5

Hepatic disease will reduce the metabolism of local anesthetics and this must be considered when deciding maximum doses for patients with such conditions.

Drug interactions

Interactions may occur between concurrent medication and local anesthetics or vasoconstrictors. There is

no concurrent medication that absolutely contraindicates the use of local anesthetics or vasoconstrictors; however there are times when a dose reduction is required. The main group of drugs that necessitate dose reduction are the cardiovascular drugs. In theory all antihypertensive medications can interact with epinephrine, for example beta-adrenergic blocking drugs can result in unopposed rises in systolic blood pressure, and calcium channel blockers and diuretics may increase epinephrine-induced hypokalaemia. Similarly beta-adrenergic blocking drugs can increase the toxicity of local anesthetics by reducing hepatic blood flow, which inhibits metabolism.

As well as prescribed medication, drugs of abuse such as cocaine can interact with epinephrine and avoidance or dose reduction of epinephrine-containing anesthetics is wise for patients who have recently used this drug.

Although there are no absolute contraindications to the use of particular local anesthetics resulting from drug interactions there are some medical conditions where the use of an epinephrine-containing solution should be avoided. These include unstable angina, severe cardiac dysrhythmias, untreated pheochromocytoma and untreated hyperthyroidism.

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Chapter 6a

Sedation and General Anesthesia in Oral and Maxillofacial Surgery: A UK Perspective

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In the UK, the use of sedation and anesthesia in general practice (the dental office) has been severely restricted to light sedation with single-agent techniques on patients who are generally fit and well (ASA 1 and 2 only). All other patients are referred to a specialist setting, usually within a hospital environment, where they can be managed by trained sedationists or anesthesiologists. The technique of conscious sedation is defined as a state of depression of the central nervous system (CNS), allowing a cooperative patient with whom verbal communication can be maintained. Any drug used must carry a wide safety margin. The vast majority of UK sedation is focused on the benzodiazepines and, for inhalational sedation, on nitrous oxide. The use of other agents such as fentanyl and ketamine is rare.

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History

The discovery of nitrous oxide by Joseph Priestley in 1776 and its subsequent use by Humphry Davy to alleviate toothache began a golden era of pain control and relief which the dental profession has exploited ever since. Despite this, and nearly 250 years later, the fear of visiting the dentist is still quoted in the UK as the most common reason why patients fail to visit their dentist on regular occasions.

Some 75 years after Priestley's discovery, Horace Wells of Hartford, Connecticut, gave the first public demonstration of the practical use of nitrous oxide. It was 10 December 1844 and, despite some ridiculing by the elitist medical profession of the day, is generally accepted as the birthdate of modern anesthesia and sedation. In truth, 2 years previously, Crawford Long had anesthetized James Venable and excised two small neck tumors. Unfortunately, he failed to publish his results until 1849 and so has never had the prominence of William Morton (see below) or Horace Wells.

As for Wells, he committed suicide 4 years after discovering anesthesia, incensed that his discovery had been "stolen" by William Morton (Fig. 6a.1). In reality, it seems that Morton was a master at adapting the ideas of others and in 1846, Professor Jackson, a specialist of chemistry, physics and geology, suggested the use of sulfuric ether as a possible volatile anesthetic agent rather than nitrous oxide. On 16 October 1846, Morton successfully anesthetized



Fig. 6a.1 William Morton demonstrating ether anesthesia.

Gilbert Abbott thereby allowing surgeon John Warren to remove a tumor from his patient. Unlike Wells, Morton received wide acclaim for his technique but his life on earth ended some 22 years later. He was virtually bankrupt, having lined the pockets of several lawyers and even fought in Congress for the patents and rights to be known as the inventor of anesthesia.

Over the next 50 years the use of ether, chloroform, and triethylene (trilene) developed slowly on both sides of the Atlantic. In the UK, the use of chloroform on Queen Victoria also saw the first recorded use of the term “specialist in anaesthesia”, applied to John Snow.

It is difficult to trace the precise developments of sedation and anesthesia at the end of the 19th century, but the advent of “daycase”, “outpatient” or “ambulatory care” anesthesia can probably be attributed to JH Nicholl who in 1909 published a series of just under 9000 cases treated at the Glasgow Royal Hospital for Sick Children.¹ In the USA, Ralph Waters opened a clinic in Sioux City, Iowa in 1916 expanding his facilities into Kansas City in 1925. These clinics are generally regarded as the forerunners of modern, stand-alone daycase units.

However, the main differences in the early days of anesthesia related to the assumption of responsibilities: in the UK it remained the responsibility of the medical and dental profession; in the USA (and in much of Europe) anesthesia was largely regarded as an adjunct to surgery and became a designated function of the nurses. Even so, on both sides of the Atlantic Ocean, the period around the 1920s saw some major advances in anesthesia with the formation of societies, the development of anesthetic machinery, and the advent of intravenous anesthetics and effective local and regional anesthesia. One of the earliest accounts is that of Douglas MacDonald who used a naturally occurring alkaloid (Somniferine) to “lightly” anesthetize a patient prior to a regional abdominal block.² This concept of “light anesthesia” appears to coincide with the work of John Lundy of the Mayo clinic who described a stage of sedation preceding anesthesia.³ Lundy developed the use of compounds of barbituric acid (in particular pentobarbitone) which became the cornerstone of intravenous anesthesia for the next 50 years. Barbituric acid had been formulated in the mid-1860s (a synthesis of urea and malonic acid) but it was its derivative compounds which continued to fuel the development of modern anesthesia, in particular with the development of thiopentone (Pentothal) and methohexitone (Methohexital/Brevital/Brietal).

The latter was an ultra-short-acting barbiturate anesthetic agent which was synthesized in 1956 and which lacked the twitching effects of some of the earlier methylated compounds. It rapidly became popular on both sides of the Atlantic Ocean and is still used in the USA. In the UK, a simple technique of giving small increments to a loading bolus was

popularized by Stanley Drummond-Jackson. He was undoubtedly skilled at the incremental methohexitone technique and was an avid proponent of ultra-light anesthesia. Unfortunately some of those who tried to emulate him were less successful and a number of high-profile anesthetic accidents in the 1960s resulted in the technique being condemned by the increasingly vocal anesthetic profession.

This professional opposition coincided with the development of diazepam – the first commercially available injectable (although it can be administered in a variety of ways) benzodiazepine in 1963. Despite its highly addictive properties, diazepam rapidly became a panacea for just about every condition known to man before it gradually found a more sensible level of usage. The intravenous formulation was not water soluble and so was dissolved in propylene glycol, an irritant substance which undoubtedly resulted in several cases of allergy or anaphylaxis as well as the risk of thrombophlebitis. Despite these disadvantages, by the late 1960s, intravenous diazepam was beginning to be offered as a serious alternative to general anesthesia for simple surgical cases. In the UK, this growth continued in a largely uncontrolled way throughout the 1970s and, in an effort to ensure some order within the dental profession, the UK government established a working party under the chairmanship of Dr WR Wylie in 1978. In his report, the concept of deep levels of sedation was essentially ended by a new two-part definition.⁴ Its relative simplicity has ensured that it is still in use today and it states:

Conscious sedation is the use of a drug or drugs to depress the central nervous system in order to produce a relaxed and cooperative patient with whom verbal contact can be maintained throughout a procedure.

The drug or drugs used must carry a margin of safety wide enough to render the unintended loss of consciousness unlikely.

The two key factors (verbal communication with the patient throughout the procedure and a wide safety margin of the sedative agent) immediately rendered using barbiturates useless. Indeed, at the time, the only possible single intravenous drug available that could satisfactorily fulfil the definition was diazepam. The report, and the constant prominent headlines in the press whenever a dental death occurred (however rare) added to a body of opinion that was being “politically” driven by the increasingly powerful anesthetic lobby. Papers such as that by Muir *et al.*⁵ demonstrated the huge safety margin of local anesthetics compared to general anesthetics. Indeed, the rarity of deaths related to local anesthesia makes it difficult to give a figure for the risk involved but it is probably between 1 in fifty million and 1 in a hundred million. How much sedation increases that risk

is equally difficult to assess but there is little doubt that conscious sedation as defined and practiced in the UK is very safe.

In the USA, sedation has travelled a different pathway. Nils Jorgensen is generally regarded as the father of intravenous sedation and he used a cocktail of drugs (pentobarbitone/scopolamine/pethidine/hyoscine) before also giving local anesthesia. Whilst these multiple drug techniques have remained popular in the USA, the UK has almost entirely developed single-drug techniques and these are described in more detail later.

Although Wylie's report was the first, it was immediately followed by two supplemental reports looking at the practical application of his recommendations. One of these was that the sedation could be given by the operator providing there was "an appropriately trained second person" present whose sole purpose was to monitor the patient. This role was frequently fulfilled by a (supplementary) dental nurse who could be given the additional training at minimal expense compared with the cost of employing a second clinician. However, in the early days of sedation the extent of any additional training for either the practitioner or the appropriately trained second person was ill defined and, in many cases, probably grossly inadequate. This resulted in the UK government requesting a further report and establishing a working party chaired by the late Professor David Poswillo.⁶ On publication, its fundamental premise was that virtually all outpatient (office) dental anesthesia should stop and that it should be replaced by simple, single-drug sedatives wherever possible. (In reality, the reverse happened for a short period before the General Dental Council, the regulatory body for the whole of dentistry in the UK, reinforced that "advice".)

This occurred in 1998 and 2 years later the chief medical and dental officers for England issued yet another report on sedation entitled "A Conscious Decision".⁷ This really did seal the fate of office-based anesthesia as can be seen from Table 6a.1. In its heyday, it is estimated that over 3 million anesthetics were being administered by dentists annually. Although the figures in Table 6a.1 only refer to the number of anesthetics being offered by the National Health Service (NHS), there is good empirical evidence to show that very few anesthetics are being administered in the dental office in the UK.

Table 6a.1 The decline in provision of NHS dental anesthesia.

In the year to 31 March	Cases of dental anesthesia
1998	350 000
1999	127 000
2000	45 000
2001	35 000
2002	11 000
2003 onwards	Very few cases

The reports on sedation in the UK, produced around the turn of the millennium, seemed to fuel the desires of every organization and society in existence to offer advice and recommendations. It is not appropriate to list them all in this chapter but they have had the effect of deterring many able practitioners from offering any form of sedation in general practice when there is a huge demand for such services and there is a clear evidence base for the safety of sedation techniques as defined by Wylie. The next section of this chapter, therefore, considers the techniques of sedation currently used in the UK along with some of the potential developments.

The role and scope of sedation

It is important to realize that the management of all patients occupies a spectrum of behavioral management. Behavior is not an easy word to define but is commonly thought of as a response or series of responses to a given stimulus. So-called "normal" behavior is even harder to define since it usually involves a somewhat vague comparison with a majority. Thus if most people who attend a dentist can sit in a waiting room and appear relaxed, the person who sits there crying in fear of his or her appointment is deemed to be behaving "abnormally". In reality, the borderline between so-called normal and abnormal can be very difficult to delineate. What is more important is the ability of the dentist to "manage" behavior in such a way that he or she can complete a patient's treatment with the minimum of stress – for either party! Behavioral management is not taught to any great extent in many dental schools, yet having some understanding of the subject can considerably ease the treatment experience of both the patient and the dentist.

Influences on behavior are usually divided into intrinsic (e.g. the will, emotional state, expectation, prior experience, etc.) and extrinsic factors (e.g. environment, culture, time of day, dentist's attitude, etc.). The dentist can easily and directly influence and adjust many of the extrinsic factors. Understanding and modulating the patient's response to the intrinsic factors is more complex but can be improved by training and experience. At the extreme end, behavioral therapy of various types is available, some of which are more effective than others. The most evidence-based of these is cognitive behavioral therapy (CBT) but other methods, including systematic desensitization, transactional analysis (TA), and rational emotive therapy (RET), can be effective. The main problem with all the above techniques is the time (and often cost) involved and this is not something everybody can commit to.

A simple progression from behavioral therapy is the use of therapeutic hypnosis. Hypnosis is controversial in some circles but can be very effective in inducing relaxation – indeed, the sensation is rather

like that of a Sunday afternoon snooze: one is remotely aware and yet able to feign total unconsciousness! As an “altered” state of consciousness, hypnosis is not well understood but it can demonstrate five different features:

- ability enhancement – allowing the completion of tasks not previously thought possible;
- memory variation – a facility to apparently improve memory retention or to eliminate known memories;
- pain control/relaxation – a diminished response to painful stimuli;
- age regression – the capacity to revert to an earlier part of one’s life;
- a hidden observer – the feeling that half of the mind is observing what the other half is doing or that life is “happening” and you are just watching it remotely.

The use of therapeutic hypnosis has been summarized by Chaves⁸ and he presents ten different possible indications for its use. These are somewhat different from the techniques used for counseling or smoking cessation, for instance, and readers who may wish to deepen their interest on the subject should read the full textbook mentioned above. Interestingly, it is only a short step from hypnosis to the next easiest type of sedation – relative analgesia or inhalational sedation as it is more commonly known in the UK.

Inhalational sedation is almost certainly the safest form of drug-induced sedation for reasons that are outlined below. It has been used in all sorts of environments from childbirth to wartime situations but its use in dentistry has been particularly beneficial in pediatric patients. Inhalational sedation is based on stage 1 of anesthesia which was first described by Guedel in 1937. In practice, nitrous oxide sedation had been used before Arthur Guedel described his classic stages of anesthesia and before any attempt was made to sub-classify sedation into various “planes”. The reality of a “continuum of sedation” is evident to all clinicians who have had adequate experience in administering nitrous oxide and is further discussed later in the chapter. For those in whom nitrous oxide sedation is inadequate there are the additional options of oral sedation or intravenous sedation prior to consideration of a full anesthetic. Oral sedation is probably the oldest form of sedation with patients self-administering alcoholic beverages or even naturally occurring narcotics in attempts to overcome their fears. The more judicious use of oral agents, formally prescribed in correct dosage, has centered on a variety of hypno-sedatives of which the benzodiazepines are currently the most popular. This can be traced back into the 19th century and remains a popular form of premedication and sedation to this day.

Similarly, the use of intravenous benzodiazepines has been the mainstay of sedation in the UK for the

last 30 years, based principally in the early years on diazepam and, more recently, midazolam. These are discussed further in the section on intravenous sedation.

Inhalational sedation

Much of this work is attributed to the late Harry Langa⁹ who popularized the term “relative analgesia”. This was based on Guedel’s description of the first stage of anesthesia – the analgesic stage. Langa (and various others) noted that there were degrees of analgesia which increased prior to stage 2 of anesthesia – the excitatory stage. Within the early stages were clinical signs that occurred including relaxation, paresthesia, euphoria, dreaming, and limited amnesia. Whilst not entirely predictable, many patients (especially children) benefited from these experiences and found dental treatment much easier to cope with.

However, whilst Langa would use the deeper ends of sedation (relative analgesia), in the UK the trend has always been to limit the maximum exposure to nitrous oxide to about 25–30% and not to err towards the higher levels required for relative analgesia. For this reason the term “inhalational sedation” is almost exclusively used and practiced in the UK. The machines used are largely the same as those in the USA with several safety features, including automatic shutdown in the case of oxygen failure, a dual pressure valve system which limits the maximum nitrous oxide to oxygen ratio, a ventilation bag and emergency override, and a pin-indexed fitting system for the gas cylinders.

As a result, inhalational sedation with nitrous oxide has had an excellent safety record with no serious cases of morbidity recorded. The slight potential for causing diffusion hypoxia (a condition that ensues as a consequence of the extremely low solubility of nitrous oxide in the blood thereby resulting in nitrous oxide “pooling” in the alveoli followed by it being re-breathed) is averted by the 30% minimum oxygen levels and monitoring the patient carefully following termination of sedation.

The disadvantage of nitrous oxide could be considered to be its lack of potency. It is undoubtedly a weak agent and this has almost certainly enhanced its profile as the prime choice for inhalational sedation. Another disadvantage, however, is that there is growing evidence that chronic exposure to even small levels of nitrous oxide may result in toxic effects to the individual thereby putting both the clinician and his or her assistants at risk. To minimize this risk, the use of scavenging equipment should be employed to reduce the atmospheric concentration to as low a level as possible. Current UK recommendations are for an 8-hour, time-averaged level of no more than 100 parts per million of nitrous oxide in room air and this may be reduced to 50 ppm in the not-too-distant future. It is necessary to use closed breathing circuits

and active scavenging to get the background level down to this figure if inhalational sedation is being used on a regular or continuous basis.

The possibility of using other agents for inhalational sedation has also been tried. The early results with sevoflurane (a modern halogenated ether) have been excellent and, like nitrous oxide, it has extremely low blood solubility. The consequential rapid rise in gas partial pressure within the blood makes it very fast acting and also gives a very rapid recovery. However, unlike nitrous oxide, the potency of the agent itself is considerable and full anesthesia can too easily be induced. For this reason it is unlikely ever to find its way into mainstream usage within the UK.

Oral sedation

The history of prescribed oral sedation can probably be traced back to chloral hydrate although the use of some of the naturally occurring narcotics probably predates it. Morphine, for example, was first isolated from opium in 1803 by the German pharmacist FWA Sertürner, who named it after Morpheus, the god of dreams.

Chloral hydrate itself was introduced into therapeutics more than 125 years ago and became popular as a sleeping potion. It was the first ever synthetic CNS depressant and appears to work through its reduced metabolite, trichloroethanol – itself an anesthetic agent. In the middle of the last century other drugs, particularly of the phenothiazine group, were developed and found to be useful as mild hypnotic sedatives. The development of the benzodiazepines, especially in the 1960s and 1970s, transformed oral sedation with drugs that were more powerful as anxiolytics and yet which carried a wide safety margin; so wide that there were many reports of unsuccessful suicide attempts of people who had overdosed on benzodiazepines (BZDs) alone. Indeed the use of oral BZDs has been supplemented with transmucosal preparations including nasal drops, sublingual gels and rectal pessaries.

In practice, the use of a simple, relatively short-acting BZD such as temazepam given 1 hour preoperatively will relieve the apprehension of a significant majority of patients. The simplicity of administration, coupled with its dependable effectiveness and low cost, make it an ideal agent for those whose anxiety is moderate but who nonetheless suffer considerable stress when facing treatment. Patients who are medicated in this way tolerate the use of local anesthetics much more easily than non-sedated patients and exhibit lower levels of body stress responses (heart rate, blood pressure, ventilation rate, etc.). On the negative side, they will be unable to drive or operate machinery for the remainder of the day of administration and they will need to be accompanied home by a competent adult. There is also some evidence

that even a single dose of a BZD will affect routine sleep patterns for up to a week and some patients find this disturbing. The desire to develop a drug which does not have these negative effects, i.e. one which provides true anxiolysis without sedation, therefore continues.

Most recently interest has been focused on negative allosteric modulators (NAMs) of the metabotropic glutamate receptor 5 (mGluR5). An early example of this is fenobam (a product which has a similar mode of action to the NAMs) which demonstrated efficacy in patients with generalized anxiety disorders after repeated administration. The translation of this into a drug suitable for reducing anxiety in an acute dental setting has yet to be established and early results have only been partly successful. However, the ideal of a product which provides true anxiolysis (without the need for hypno-sedation) remains a goal for future development.

Intravenous sedation

The benzodiazepines (BZDs) are versatile drugs which have been used orally, intramuscularly, intravenously, rectally, and transmucosally. Pharmacologically, each one has at its base a fused dual ring of benzene and 1–4 diazepine. Whilst the benzene ring is basically a hexagon, the diazepine ring is a heptagon with positions 1 and 4 of the 7 corners being nitrogen rather than carbon atoms. (For those who enjoy the science, see Cooper, Bloom and Roth's book.¹⁰) For those who may be baffled by the science, what is important to understand is that by substituting or adding various radicals to the basic BZD ring, the pharmacokinetics and dynamics of the compounds change considerably. A range of activity varying from agonist through antagonist to inverse agonist has been described and these properties can be adapted to differing clinical requirements. In addition, the solubility of the compounds varies and, for intravenous usage, there are clear advantages to having a water-soluble drug. For orally consumed compounds, absorption is a more important factor and this is another marked variable. All the BZDs, however (whether agonist or inverse agonist), are thought to bind to a specific receptor, analogous to or part of the GABA_A receptor (see Fig. 6a.2) at the nerve synapses.

These receptors essentially control the flow of chlorine ions in and out of the nerve axons – inward flow (agonists) producing relaxation and anxiolysis and outward flow (inverse agonists) producing agitation and anxiety. Antagonists have the effect of displacing both agonists and inverse agonists thereby "neutralizing" the effect on the GABA receptor. Pharmaceutical companies have created thousands of benzodiazepines over the years since it is difficult to predict their exact properties along the activity spectrum without *in vivo* testing. Despite this, only three

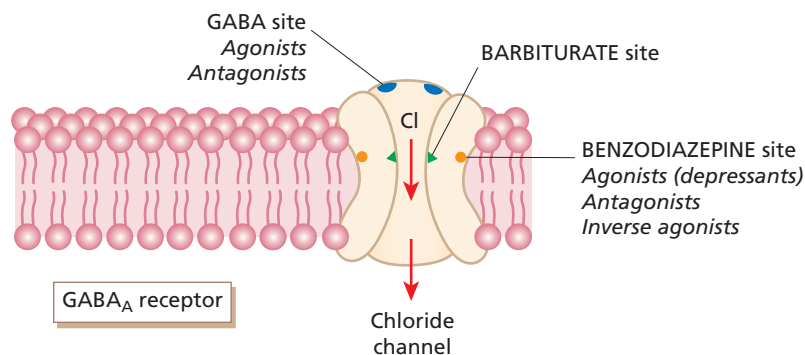


Fig. 6a.2 Functioning of the GABA_A receptor.

benzodiazepines are currently used in the UK for intravenous sedation:

- diazepam (now usually administered in an emulsified form to avoid the complications of thrombophlebitis);
- midazolam (a water-soluble, shorter-acting alternative);
- flumazenil (an antagonist, usually reserved for emergency use).

The similarity of the compounds is illustrated in Fig. 6a.3.

Intravenous BZDs are usually administered via an indwelling cannula inserted into either the dorsum of the hand or the antecubital fossa. They should be administered slowly in small increments, carefully observing the responses of the patient. With diazepam, the dose given was usually high enough to induce Verrill's sign. This consisted of a degree of partial ptosis that would bisect the pupil of the eye.

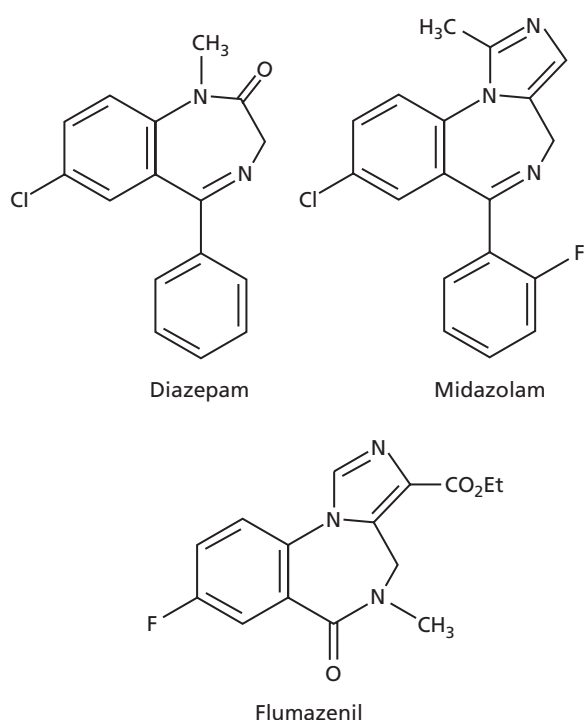


Fig. 6a.3 Midazolam, diazepam, and flumazenil.

However, it is now generally agreed that this level of sedation is too profound and the aim is to produce a more relaxed but less sedated patient. To this end, Eve's sign is used as a useful indicator of the early signs of loss of motor coordination. The sign is simply tested by asking the patient to touch his/her nose with one finger. As early relaxation occurs, the finger usually falls short – touching the top lip only. Many patients giggle when this occurs thereby adding a further indicator that they are becoming more relaxed.

In the general outpatient (office) setting, patients of ASA 1 and 2 are treated, while patients of ASA 3 or above are usually referred to a specialist unit. Preoperative assessments consisting of a full medical history, an assessment of ASA status, blood pressure, and body mass index (BMI) are usually undertaken. BMI is a ratio that calculates the degree of obesity of a patient and is the weight of the patient in kilograms divided by the height in metres squared. The ideal range is between 20–25, and patients who have a BMI of 35 or over begin to progressively demonstrate an increased risk of airway obstruction. While this can be monitored by the use of pulse oximetry, it is wise to use caution in treating patients who are significantly obese. The respiratory depression that is a side-effect of the BZDs together with the tendency of obese patients to obstruct their airways (or more seriously to suffer from sleep apnea) can prove a fatal combination and deaths from this have been reported by endoscopists. The advent of flumazenil has, to some extent, alleviated the concerns regarding BZD overdosage but it is unsafe to rely on an antidote to compensate for unsafe medical practice. Some practitioners use flumazenil as a reversal agent, in the same way that the paralyzing agents used during modern general anesthetics can be reversed using neostigmine. Others argue that the cost of the agent and the risk of resedation, however small, proscribe such use.

Despite the widespread use of the BZDs they do not fulfill all the requirements of an ideal sedative which should principally be aimed at reducing anxiety rather than causing hypno-sedation. At its simplest, the ideal drug would have no hypno-sedative effects but would simply remove the fear and anxiety commonly associated with dental treatment. Such a

drug would be best administered orally since many dental phobics often fear any form of injection including venipuncture. Although this can be ameliorated by using topical local anesthetic creams such as amethocaine or EMLA (a eutectic mixture of procaine and lidocaine), it still remains problematic for many patients. This has not stopped efforts to improve on the BZDs and, in the mid-1980s, a new class of drug was licenced.

Propofol is a 2,6-di-isopropylphenol with marked sedative-hypnotic and anesthetic properties. It is minimally soluble in water and so is formulated as an emulsion for clinical use. Propofol is highly lipophilic and distributes widely in the body. It has a three-phase model half-life of approximately 2–4 minutes in the alpha phase, 30–45 minutes in the beta phase and around 4–60 hours in the gamma phase. Propofol is rapidly and effectively metabolized by the liver prior to being eliminated through the renal system. Following administration, the clinical effect of sedation or hypnosis (depending on dose administered) occurs within a minute and usually lasts for 5–6 minutes.

Over the last 10 years, propofol has become the agent of choice for induction of general anesthesia although maintenance is more common with the modern volatile agents (see later). It has an excellent safety profile and, although it causes some decrease in both systolic and diastolic blood pressure (possibly by up to 25%), there is minimal change in heart rate. Cases of respiratory depression and apnea are also common. The cardiovascular and respiratory effects of propofol do not give cause for serious concern in otherwise healthy patients.

The use of propofol as a sedative in the UK has been restricted due to the risk of achieving unintentional anesthesia. However, the advent of patient-controlled sedation (PCS) (see Figs 6a.4 and 6a.5) has



Fig. 6a.4 The use of patient controlled sedation.



Fig. 6a.5 Microprocessor controlled pump.

allowed it to gain some popularity. PCS is an adaptation of patient-controlled analgesia (PCA). Drugs (whether analgesics or sedatives) are administered using a microprocessor-controlled pump which has two essential features. The first of these is the ability to adjust the dose administered; the second is the capacity to create a 'lock-out' period during which time the machine will not administer any agent even when prompted by the patient. Research into both the use of PCA and PCS systems has demonstrated that patients consistently administer less of the agents than would be the case if they were administered by a clinician.

General anesthesia

Despite significant advances in sedative techniques, there remains a number of patients who still require full anesthesia either as a result of their own anxiety or due to the complexity of the procedures. The consequence of effectively banning office anesthesia in the UK has been a marked increase in both the number of referrals to hospital units and the number of cases being seen as emergency admissions. All such patients who are accepted for anesthesia are treated by trained anesthesiologists – there being no concept of anesthetic nurses in the UK.

The last 20 years have seen a significant change in practice in the UK, with the laryngeal mask airway (LMA) becoming almost standard as a means of protecting the airway (Fig. 6a.6). Since its invention by Dr Archie Brain¹¹ in 1983 its use has grown exponentially both in the UK and around the world. It has been modified in various ways such that different types are now available. The inflatable diaphragm expands in the oropharynx, allowing the exchange of gases while at the same time protecting the larynx from any falling debris. After being placed by the anesthetist, the flexible LMA especially is easily

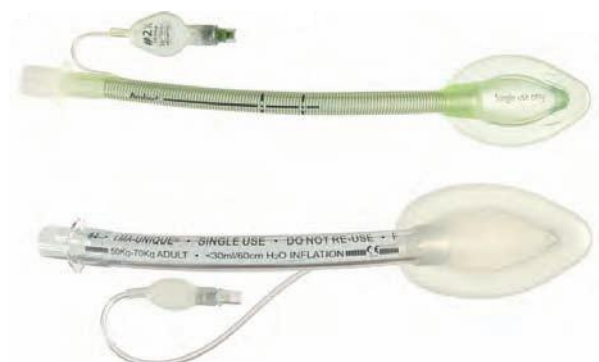


Fig. 6a.6 A rigid (lower) and flexible (upper) laryngeal mask side by side. The cuff is inflated and sits neatly in the oro-pharynx.

manipulated by the surgeon so that it does not interfere with the operation site.

In terms of induction and maintenance of anaesthesia, the use of propofol and sevoflurane is most common using oxygen and nitrous oxide as carrier gases within the breathing circuit (or system as it is more correctly known). The technical aspects of anaesthesia have, however, improved significantly, with electronic equipment capable of monitoring almost every aspect of everyday practice. While some argue that this has reduced the profession to little more than technocrats, there can be little doubt that improved monitoring equipment has contributed to an increased safety profile. On the negative side, it has added to the induction time required by the anaesthetist, as all the equipment has to be attached in some form to the patient. In a social health care system this could be considered to be less problematic than in an independently funded one but it still has to be considered in terms of cost and benefit. Since the potential benefit is so great, there can be little doubt that such monitoring can be justified in routine use.

Conclusion

Dentistry, surgery, and anaesthesia have been intricately linked from the earliest times of discovery. Most recently, sedation has had an increasingly prominent role within the dental profession and its increasing use should ensure that this continues to be the case for many years to come. However, this will be dependent on a demonstrable safety profile and an excellent therapeutic effect. In the UK, the controlled use of conscious sedation should ensure that this remains the case.

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Chapter 6b

Sedation and General Anesthesia in Oral and Maxillofacial Surgery: A US Perspective

Richard C. Robert

In the USA, office-based anesthesia for oral and maxillofacial surgery is considerably different from that which is practiced in the UK. In the US oral and maxillofacial surgeons train side-by-side with anesthesiology residents in teaching centers, as well as within their own departments during their 4–6 years of training. Once in private practice, largely using the operator–anesthetist model, they offer the full range of anesthesia services as defined by the American Society of Anesthesiologists. These include minimal sedation, moderate sedation, deep sedation, and general anesthesia. The anesthetic agents, monitoring and emergency preparedness for this office-based anesthesia are quite similar to those employed in free-standing and office-based outpatient surgery centers. The most frequently used anesthetic agents are propofol, midazolam, fentanyl, ketamine, and, in some cases, sevoflurane. Commonly a balanced approach with multiple agents is employed. Typical monitoring includes continuous electrocardiogram (EKG), blood pressure and pulse, pulse oximetry, auscultation with a pretracheal stethoscope, and, with increasing frequency, capnography. A requisite for membership in the American Association of Oral and Maxillofacial Surgeons is periodic office-anesthesia evaluations to assure patient safety. This process is reviewed by the American Society of Anesthesiologists to ensure consistency with ASA guidelines.

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Office anesthesia for oral and maxillofacial surgery in the US today is vastly different from that seen in the UK. A wide range of surgical procedures is performed in an office setting under sedation and light general anesthesia. The difference is largely due to the effectiveness of professional organizations in the US that have been formed by oral and maxillofacial surgeons, as well as those with which they have chosen to become affiliated.

Historical perspective

The 19th century after Wells and Morton

The year is 1883 and Sophie is scheduled to have her painful tooth extracted at the office of the Colton Dental Association, with Professor Colton himself (Fig. 6b.1) providing nitrous oxide anesthesia. Although Sophie would have normally dreaded such an appointment, she is now almost enthusiastic. At this point in his life Gardner Colton¹ is arguably the most famous anesthetist in the world. During the 20 years over which he has been delivering dental anesthesia, he has traveled to the Universal Exhibition and First International Congress of Medicine in Paris and impressed audiences in both Paris and London with nitrous oxide anesthesia. In the US he now has offices in St. Louis, Chicago, Brooklyn, Cincinnati, Boston, Baltimore, and Philadelphia and over 100 000 patients have been successfully treated.^{2,3}

From reports she has received from acquaintances who have also had extractions at the Colton Dental Association, Sophie feels that the anesthesia she will receive will be far more desirable than any of the other types of anesthesia about which she has heard. Ether, which has been used for major surgical procedures, sounds terrible. It takes a long time for the patient to fall asleep, and the smell is disgusting. Then, once the operation has been completed and the patient awakes, it is to bouts of uncontrollable retching and vomiting. Worse yet, she has heard of mysterious unexplained deaths associated with chloroform, the other popular anesthetic of the day. So drifting off



Fig. 6b.1 Gardner Quincy Colton 1814–1898. Reintroduced nitrous oxide for exodontia during the 1860s and popularized its use in the US and Europe. (Illustration courtesy of Yale Medical Historical Library.)

in a euphoric state under the influence of Professor Colton's nitrous oxide is in Sophie's opinion the best of all anesthetic alternatives.

As she drifts off into somnolence, Sophie is not aware of the true nature of the "anesthetic" to which she is about to subject her body. Colton will watch her breathe the gas until she exhibits color changes.³ She will pass through pallor to the shades of blue of cyanosis, and then the gas will be removed. At that point there will be about 2 minutes of working time before she once again "pinks up", and it is during these 2 minutes that her surgery will be performed. If she begins to awake before the procedure has been completed, she will again be given the nitrous oxide until she becomes cyanotic once more. This time when she "pinks up", she will be allowed to awake with her tooth extracted. She will be pleasantly surprised that she has been "asleep" through the entire procedure and felt no pain during her surgery. Sophie does not know that the "anesthetic" which causes her to lose consciousness is actually asphyxia from the hypoxic state into which she will be rendered by exclusion of oxygen from the gas that she breathes. She also does not appreciate the fact that Colton himself does not thoroughly understand his own technique and falsely believes that the oxygen to which the nitrogen molecules are bonded in nitrous oxide will maintain his patients' oxygenation during the surgical procedure.

Of course Sophie is not a real patient, rather she is a composite of some 200 000 patients who underwent nitrous oxide "anesthesia" performed by Gardner Colton from the 1860s to 1890s. In large part, due to Colton's efforts in its promotion, nitrous oxide was to remain the favorite anesthetic for exodontists and oral surgeons for three quarters of a century, both in the US and abroad. But how was it that nitrous oxide should assume such a position in the world of anesthesia after Wells' unsuccessful demonstration at Massachusetts General Hospital which had been described as pure "humbug"? The tale is an interesting one, worthy of repeating.

During the several years following Wells' failed attempt to demonstrate nitrous oxide anesthesia, Colton (who had administered the initial nitrous oxide anesthetic to Wells) became involved in other scientific pursuits, including the development of the telegraph and the electric engine. He moved to California during the Gold Rush, but was unsuccessful in his attempts at prospecting. On returning to San Francisco he was appointed as the first Justice of the Peace. In this capacity he was in charge of the sale of town lots in the city, which proved to be controversial. Colton ultimately left California a rich man, but with a tarnished reputation. He returned to Boston where he had begun his adult career.

During Colton's hiatus from the world of dental anesthesia, word of Morton's successful demonstration of ether at the "Ether Dome" spread to physicians and dentists throughout the world. This interest in

turn led to further experimentation. Notably, James Simpson,^{4,5} an Edinburgh obstetrician, became obsessed with the idea of finding another inhalation agent which lacked the disgusting smell of ether and its flammable properties. He and some fellow inquisitive scientists set out to test other agents on themselves. On Thursday evenings they met at Simpson's home and inhaled various chemicals to personally assess their anesthetic attributes. On 3 November 1847, one of the "chemicals de jour" was chloroform, which had been originally produced 16 years earlier, but for which no one had come up with a practical use. On sniffing chloroform, Simpson and his friends were transported to another world and finally into the "arms of Morpheus". Simpson found that chloroform had such desirable properties as a more rapid onset than ether, a less unpleasant smell, non-flammability, and low cost. The new agent was affectionately dubbed "Chlory", and it was to dominate the anesthetic world over the next several decades.

Chloroform became the favored anesthetic of American Civil War surgeons because of its more rapid onset of action accompanied by a decreased incidence of postoperative vomiting and nausea.⁶⁻⁹ There were periodic reports of deaths during anesthesia, presumably from cardiac dysrhythmias and postoperatively from liver failure. Nonetheless, to combat surgeons these were risks which they were willing to undergo for the rapidity of chloroform's induction. After the Civil War, ether regained prominence in the US, but chloroform continued to be the agent of choice in the UK for a number of years.

Neither ether nor chloroform were widely accepted by dentists for exodontia. The dental world awaited an agent which had a pleasant smell, relatively rapid onset and which had an "aura of safety" about it. And who could have been more qualified to bring this agent forth than the very man who had first demonstrated the use of inhalation anesthesia on Horace Wells, Gardner Colton himself.

After his return to the East Coast, Colton¹ fell on hard times when he lost the money he had made in California in an investment in the ill-fated Salt Works of Syracuse. This prompted his resuming his lectures and demonstrations of nitrous oxide. He ultimately associated himself with two eminent dentists in New York City which resulted in the formation of the Colton Dental Association. In 1898, a year before he died, Colton published a book summarizing his anesthetic accomplishments. He recorded that nitrous oxide had been administered to 193800 patients whose ages ranged from 3–90 years, reportedly without a fatality.

It is difficult to say why Colton held so firmly to his belief that he was providing adequate oxygenation for his patients with his 100% nitrous oxide approach. We do know that he never completed his medical studies which he had undertaken in 1842, 2 years before he administered nitrous oxide to Wells. He had entered the Crosby Street College of Physicians

and Surgeons in New York, but decided that he did not wish to pursue a medical career, stating that he had elected to "throw physic to the dogs".¹ Thus "Professor Colton" was not really a professor at all, but merely a popular lecturer with minimal medical background who lectured on chemistry with an emphasis on nitrous oxide. Unquestionably, however, he was endowed with certain perceptive qualities which enabled him to become past master at rendering patients unconscious with nitrous oxide hypoxia without killing them.

Other physicians and scientists studying nitrous oxide suspected that Colton's method did, in fact, induce a hypoxic state. Opposing views developed, and in 1868 Edward Andrews, a Chicago surgeon, advocated that oxygen be utilized with nitrous oxide to provide a safer anesthetic.¹⁰ However, with the equipment available at the time, a satisfactory combined delivery was not practical. Ultimately Thomas Evans, an American dentist living in Paris who had observed Colton's demonstrations there in 1867, introduced the concept of compressing the gas into a liquid form which could be more practically stored in containers.³ By 1881, the SS White Company had developed a storage cylinder for nitrous oxide which led to increased popularity of the agent for exodontia.¹¹

As attempts were being made to provide a safer inhalation anesthetic, other developments were taking place in New York which would ultimately impact dentistry and oral surgery just as profoundly as the discovery of surgical anesthesia. William Stewart Halsted,^{12,13} considered by many to be the "Father of American Surgery", began his landmark work in the development of effective local anesthesia. Halsted and a colleague, Richard J. Hall, administered nerve blocks with cocaine and actually even demonstrated systematically the application of local anesthetic for dentistry and oral surgery. Unfortunately, like so many of the early pioneers, Halsted and Hall administered the agent they were investigating to themselves and developed addiction to the drug which they were studying. Hall ultimately had to leave academia altogether and moved to California, where he remained addicted to cocaine until the end of his life. Halsted, in an attempt to overcome his addiction, entered the Butler Hospital in Providence Rhode Island for treatment. The "treatment" basically involved the transferral of his addiction from cocaine to morphine, to which he remained addicted until his death in 1922. Nonetheless, in his addicted state, he continued to make outstanding contributions to the development of surgery in the US and was ultimately appointed Surgeon in Chief and Professor of Surgery at Johns Hopkins in 1892.

As the 19th century came to a close, yet one more important event would take place that would ultimately enable anesthesia for oral and maxillofacial surgery to enjoy the success it holds today. German medical science, never to be overshadowed in the

19th century, produced the first of a line of drugs which would provide the cornerstone for outpatient anesthesia. In 1903, Emil Fischer, doyen of German organic chemists and Nobel laureate, joined pharmacology professor Joseph von Mering in the development of a hypnotic drug, diethyl barbituric acid, which was synthesized from malonic acid and urea. It was introduced on the market under the name Veronal® by their parent company Bayer as the first commercially available barbiturate.^{14–17} However, it would be several decades before a truly short-acting barbiturate would be developed that would enable patients to undergo anesthesia for their surgical procedures and yet go home at the end of the day.

The 20th century

Early decades

As the 19th century passed into the 20th, the “Great Transatlantic Debate” was finally coming to an end. For half a century the controversy had raged as to which was the better and safer anesthetic. Was it Morton’s “Yankee Dodge” as British surgeon Liston had coined ether,^{18,19} or was it Simpson’s chloroform which had dominated in the UK? In the 1880s, due to the frequent reports of deaths associated with chloroform anesthesia, a panel of experts was assembled to assess the agent’s safety. The panel was termed the “Hyderabad Commission” after the Nizam of Hyderabad, the wealthy Indian potentate who funded the study.⁸ It was chaired by a British medical officer, Dr Edward Lawrie, who had trained in Edinburgh, which had been “Chlory’s” birthplace. As one might anticipate, the commission’s output was somewhat tainted by Lawrie’s involvement.²⁰ Its report was rather inconclusive, which was held by Lawrie to substantiate the safety of chloroform. The prestigious British medical journal *The Lancet* published the conclusions of the Hyderabad Commission, but failed to accept the commission’s interpretation of the data. Instead, they encouraged Britain’s eminent physiologists to further study the problem, and the results of their studies left no doubt. Indeed, the fears regarding chloroform were justified – chloroform was not a safe anesthetic after all. Their conclusions were adopted in 1892 and confirmed again in 1901 by the British Medical Association’s Committee of Anaesthesia and published for all the world to see.⁸

Although ether had won the debate, its doing so was actually a mixed blessing. The difference in the attributes of ether and chloroform had actually been responsible for a dichotomy in the culture of anesthesia in the US and the UK. As we have seen, chloroform depressed respiration, was a myocardial depressant which induced ventricular dysrhythmias, and was responsible for hepatorenal toxicity.²¹ Consequently, it was necessary for those who administered chloroform to be physicians, not just technicians or nurses. Thus, in the UK, where chloroform was dominant,

the profession of anesthesiology developed early on. However, in the US, where ether reigned supreme, it appeared that there was no need for physicians to take on such a mundane task as periodically placing a drop or two of ether on the mask as the surgical patient slept. As opposed to chloroform, ether was incredibly safe. It neither caused respiratory depression nor induced arrhythmias. It was not histotoxic, and it was virtually impossible for even the inexperienced to give a life-threatening overdose of the anesthetic.¹⁸

So safe was ether that its administration could be relegated to technicians and nurses. These lesser-trained anesthetists often became more skillful than the surgeons for whom they worked, who only gave anesthetics infrequently themselves. The nurses were cheap, did not argue with the surgeon, and yet did an admirable job in providing anesthesia with ether. With nurses performing the task, there appeared to be no need for physicians to become involved with the process at all, and when they did so, they were poorly paid.²² Anesthesia was the “poor stepchild” of surgery and had no academic foundation in medicine and no journals. Eventually Francis H. McMechan, one of the few physicians to carry the torch for anesthesiology as a specialty of medicine, persuaded *The American Journal of Surgery* to provide some coverage for anesthesia which resulted in the *Quarterly Supplement of Anesthesia and Analgesia* in 1914. Not until 1922 did a periodical devoted solely to anesthesia appear, *Current Researches in Anesthesia and Analgesia*.²²

Although the very success of ether had thwarted the development of anesthesiology in the US, at the turn of the century pioneering physicians in the midwest were to finally plant the seeds for the establishment of anesthesiology as a profession. Principle among them were Ralph Waters at the University of Wisconsin and John Lundy at the Mayo Clinic.²³ In the same pioneering tradition which had resulted in the western expansion of the US, these physicians set out on a path of their own which enabled anesthesiology to become a profession in its own right, and assured that it was well founded in science and medicine. We will see more of John Lundy as we explore the evolution of “balanced anesthesia” on which anesthesia in oral and maxillofacial surgery is based to this day.

Although departments of anesthesiology were finally becoming established during the early decades of the 20th century, ether and nitrous oxide continued to be the only practical anesthetic agents available. Pharmacologic research got underway to identify better barbiturates and local anesthetic agents. Various other inhalation agents were explored, but none demonstrated clear-cut advantages. At least improved anesthesia machines were forthcoming, usually from the dental sector. In 1902 Charles Teter a dentist from Cleveland, designed and modified an improved nitrous oxide and oxygen anesthesia

machine. Six years later in 1908 another dentist, Jay Heidbrink, introduced flow meters to accurately control the delivery concentration of gases.¹⁰

Although Halsted and others had demonstrated the possibility of effective local anesthesia, cocaine did not prove to be an agent which would be applicable to the treatment of the masses of dental and medical patients. Once again, German medical chemistry came forth with a solution. In 1904, chemist Alfred Einhorn patented 18 para-aminobenzoic derivatives, one of which was given the name "Novocaine".^{24,25} Although Novocaine became the standard local anesthetic of the time, it had inherent drawbacks: many patients were highly allergic to para-benzoic acid from which it was derived and relatively high doses of epinephrine were required to maintain the anesthetic in the area of injection. The search for better agents continued until the advent of lidocaine, the first amide anesthetic, in 1949.¹³

The mid-20th century

Up until the early decades of the 20th century, efforts had been directed at finding the ideal mono-anesthetic agent which would promptly "put patients to sleep" and allow their rapid arousal at the end of the surgical procedure. However, one farsighted pioneer surmised that it was unlikely that a single chemical could provide all of the desired effects requisite to surgical anesthesia. In 1894, John Silas Lundy^{23,26} (Fig. 6b.2) was born in the Dakota territories. He originally practiced anesthesia in Seattle, but was ultimately recruited by Will Mayo to join him at the Mayo Clinic. At Mayo Lundy developed the concept of "balanced anesthesia" which relied on the combination of a number of different agents including premedication, regional anesthesia and light general anesthesia. Lundy's approach was based on the concept that the smaller doses of multiple agents in a balanced anesthetic would be associated with fewer complications and would provide a more desirable anesthetic experience for patients. He worked with pharmaceutical firms in clinical trials on various intravenous agents, and was one of the first to investigate the clinical use of thiopental (Pentothal Sodium) in 1934. In Lundy's technique small intermittent boluses were administered to maintain the patient at the desired level of anesthesia. Although his patients tended to awaken more rapidly with fewer after effects than with inhalation anesthesia, Lundy strongly felt that an overnight stay in the hospital was still necessary.¹¹

Just 3 years after Lundy's introduction of Pentothal Sodium to the anesthetic world, Adrian Hubbell^{3,11,27} (Fig. 6b.3), a newly graduated dental student from California, undertook his oral surgery training at the Mayo Clinic. Once he was introduced to intravenous anesthesia during his training, he became fascinated with the idea of adapting the technique to office oral surgery practice. Like so many other pioneers before



Fig. 6b.2 John Silas Lundy developed the concept of balanced anesthesia in the 1920s and published the first account of the clinical use of Pentothal Sodium. (Reproduced from Rabbitts JA, Bacon DR, Nuttall GA, Moore SB. Mayo Clinic and the origins of blood banking. *Mayo Clin Proc* 2007; 82(9): 1117–18 by permission. Copyright © 2007 Quadrant HealthCom.)



Fig. 6b.3 Adrian Hubbell. Completed his residency training at the Mayo Clinic in 1940 and developed the concept of office-based anesthesia in his oral surgery practice in Long Beach, California. (Photo courtesy of The American Association of Oral and Maxillofacial Surgeons.)

him, Hubbell decided to try the anesthetic himself and had his third molars removed under Pentothal anesthesia. On completing his training Hubbell returned to California and joined an established oral surgeon Berto Olsen in private practice. Olsen's practice had traditionally been a nitrous oxide practice, but he consented to change over to Pentothal Sodium under Hubbell's guidance. Over the next several years, Hubbell perfected his office anesthetic technique with Pentothal Sodium, and in 1945 he opened a 6000 sq. ft. oral surgery clinic in Long Beach, California. Over the next several decades more than 300000 anesthetics were to be administered in Hubbell's office without a single fatality.

Word of Hubbell's success in California was soon to spread throughout the country. He had frequent visitors to observe his office operation, and some of these entreated him to put on courses on intravenous Pentothal anesthesia. Consequently, in the mid-1950s



Fig. 6b.4 Harold Krough. Worked with Adrian Hubbell to establish courses in the use of Pentothal Sodium for office-based surgery during the 1950s. (Photo courtesy of The American Association of Oral and Maxillofacial Surgeons.)

from 1953–1958, Hubbell and an east coast colleague, Harold Krough (Fig. 6b.4), from Washington, DC fielded courses on the technique.

At the Mayo, Hubbell had learned Lundy's techniques but questioned one of his primary tenets, i.e. that balanced Pentothal anesthesia could only be performed with an overnight hospital stay. Hubbell amply demonstrated that this type of anesthesia could indeed be performed on an outpatient basis with safety and efficiency. His successful demonstration of office-based Pentothal anesthesia provided the foundation for the adoption of outpatient surgery as the primary form of surgical care delivery in virtually all surgical specialties by the end of the 20th century.

Another important mid-20th century pioneer in intravenous anesthesia was Niles B Jorgensen of Loma Linda University.^{2,3,27} Jorgensen also advocated a balanced approach, but preferred a lighter level of sedation. The "Jorgenson technique" entailed the administration of three agents: pentobarbital, meperidine, and scopolamine.^{28–30} His technique enjoyed an excellent safety record and laid the groundwork for the use of "conscious sedation" in all fields of dentistry.

By the end of the century's fifth decade, another ultra-short-acting barbiturate became available. Eli Lilly introduced sodium methohexital (Brevital®) which was initially field tested in Hubbell's private practice over a 2-year period.³¹ In 1960 it was released to the profession and rapidly replaced Pentothal Sodium as the anesthetic agent of choice in most oral surgery practices. It was to remain so until the end of the century.

Latter portion of the 20th century – the 1960s, 1970s, and 1980s

Barbiturate-based "balanced" anesthetic approaches with nitrous oxide gained wide acceptance in the profession, but the technique was not without its

shortcomings. Preoperative anxiety often translated into relatively larger doses of barbiturate. This tended to make laryngospasms more likely and prolong recovery as well. In addition, barbiturates lacked analgesic properties, and in some cases caused hyperalgesia. Consequently, during injections of local anesthesia, or when there was inadequate local anesthesia, the patient would often respond violently. Fortunately, advancements in pharmacologic chemistry made it possible to largely overcome these problems. Soon additional intravenous agents were added to the "balanced" combination of drugs.

An important new family of anesthetic drugs, the benzodiazepines, appeared in the late 1950s and early 1960s.³² Since that time the benzodiazepines have become an essential part of balanced anesthesia in outpatient surgery centers and oral and maxillofacial surgery offices as well. The first of these agents which enjoyed wide acceptance in dentistry³³ and oral and maxillofacial surgery was diazepam, introduced in 1963. Its anxiolytic and sedative properties were augmented by a profound amnestic quality as well. When it was administered in combination with the opioid meperidine prior to the short-acting barbiturate, a smoother anesthetic course could be achieved.

Another new family of anesthetic drugs which emerged during the 1970s was phencyclidine derivatives.³⁴ Phencyclidine itself was synthesized in the late 1950s, but was found to cause significant hallucinations and delirium. Experimental work continued with the investigation of a number of other derivatives. Finally, ketamine was synthesized in the mid-1960s and released in 1970. Unlike most other anesthetic drugs (with the exception of narcotics) ketamine had analgesic properties and tended to raise blood pressure as opposed to lowering it. Its CNS effects were largely centered in the thalamic relay centers which led to a state of anesthetic "dissociation". Although ketamine, like phencyclidine, also had the potential for causing dysphoria, it was found that the latter could be largely overcome by administration of benzodiazepines prior to ketamine.

Over the several decades at the close of the 20th century, health care costs would lead hospital administrators and insurance carriers to explore the idea of outpatient surgery, not unlike that which had been employed in oral and maxillofacial surgery offices throughout the country for decades. Indeed, between the 1980s and the first decade of the 21st century, there was a shift of approximately 75% of surgery procedures from the inpatient hospital setting to a variety of ambulatory venues. These included hospital outpatient surgery centers, free-standing outpatient surgery centers, and surgeons' offices.³⁵

An important element in safe delivery of outpatient surgery is rapid and complete recovery before the patient is discharged. Consequently, there was a need for shorter-acting drugs to ensure that the patients could be discharged without being overly

obtunded. Fortunately, pharmacologic research led to such agents. The first of these was fentanyl,³⁶ an anilidopiperidine related to meperidine, which was introduced in the 1970s. It was 100 more times more potent than morphine, had a shorter duration of action, and was less likely to cause nausea. Within the next decade midazolam, a shorter-acting benzodiazepine,³⁴ also became available. This new agent midazolam not only had a shorter duration of action than diazepam, but was soluble in blood and provided more profound amnesia.

During the latter decades of the 20th century, technologic advancements paralleled those in pharmaceutical research. Electronic monitoring devices gained popularity in all types of outpatient anesthesia, whether in freestanding surgery centers or in oral and maxillofacial surgery offices. Monitors included electrocardiogram (EKG), blood pressure, and pulse oximetry, which were usually accompanied by a pretracheal stethoscope.³⁷ Monitoring standards were introduced and were soon mandated for accreditation of facilities.

Although the barbiturate induction agents Pentothal Sodium and methohexital had been “workhorse” anesthetic agents for half a century, a new agent, propofol^{38,34} was to be introduced in the 1980s and 1990s which would ultimately replace the barbiturates. This new agent propofol was rapidly redistributed and metabolized like barbiturates, but had other desirable characteristics as well. It did not tend to predispose to laryngospasms, it had innate antiemetic properties and patients awoke with a feeling of well-being.

The 21st century

By the turn of the century propofol had largely replaced Pentothal Sodium for anesthetic induction in hospitals and outpatient surgery centers. However, the move to propofol took place much more slowly in oral and maxillofacial surgery offices. The incremental Brevital[®] technique which Hubbell had popularized in the 1960s was well entrenched and few saw the need to replace the technique with the expensive new agent propofol. However, within the first few years of the 21st century this was all to change. Due to a problem with contamination at the production plant, there was a hiatus in Brevital[®] (methohexital) availability.³⁹ This resulted in many oral surgeons having to convert to propofol as their primary intravenous anesthetic agent. When methohexital once again became available on the market, many practitioners elected to not return to methohexital because they had come to appreciate the more desirable anesthetic properties of propofol.

The first decade of the 21st century has also seen a broadened view of “balanced” anesthetic techniques. Many anesthesiologists and oral and maxillofacial surgeons noted the complementary effects of propofol and ketamine.^{40–43} Soon articles began to appear in

both the medical and the oral and maxillofacial surgery literature advocating the administration of low-dose ketamine with propofol. Another modification of the conventional “balanced cocktail” in some offices has been the replacement of fentanyl with remifentanyl.^{44,45} The latter is rapidly metabolized by esterases found in plasma, red blood cells (RBCs), and interstitial tissues, which enables rapid emergence to accompany an excellent level of intraoperative analgesia.

Training, professional organizations, and standards

During the early years training in surgery and anesthesia for oral surgeons was largely by preceptorship and no professional organizations existed. However, as the field of exodontia began to emerge from the parent dental profession, it became obvious to those practicing exodontia that they should establish an organization of their own. In 1918, after 3 years of preparation, the charter meeting of the American Society of Exodontists was held in Chicago. Over the years, the name of the organization evolved from the American Society of Exodontists to the American Society of Oral Surgeons and Exodontists and in 1943 the American Society of Oral Surgeons (ASOS). The latter name was to be kept until 1978 when the organization took its current name, the American Association of Oral and Maxillofacial Surgeons (AAOMS).⁴⁶

As discussed above, during the early part of the 20th century anesthesia was administered by nurse anesthetists rather than anesthesiologists.¹⁸ Consequently, oral surgeons obtained their training through anesthesia training programs which were run by nurse anesthetists. Gradually anesthesiology became an accepted specialty of medicine and university-based anesthesia departments began to appear in the late 1920s and 1930s.⁴⁷ By the 1940s formal oral surgery training programs had also become established, and oral surgeons were able to receive anesthesia training through the departments of anesthesiology.

Since it was apparent that anesthesia played such a critical role in dental practice, the American Dental Society of Anesthesiology (ADSA) was established in 1954.⁴⁸ Many of its initial members were prominent oral surgeons, and oral and maxillofacial surgeons continue to constitute the majority of the membership of the organization. The ADSA publishes its own periodical, *Anesthesia Progress*, and conducts continuing education programs on various aspects of anesthesia as it relates to both oral and maxillofacial surgery and dentistry in general.

Oral surgery educators and the leaders of the ASOS developed a close working relationship that led to the conference on Anesthesia for the Ambulatory Patient which was held in Chicago in 1966. This conference initiated discussion on the scope of training required for competent delivery of

anesthesia in an office setting.¹¹ The dialog culminated in an increase in the requirement for general anesthesia training from 3 to 6 months. The latter requirement was definitively recorded in the ASOS document “Essentials of An Advanced Educational Program in Oral Surgery”.

Fortunately for the members of the specialty, its leaders have often been farsighted individuals who have assured maintenance of high standards. Rather than wait for legislative mandates which might place unreasonable demands on its membership, the association and its component chapters have established anesthesia standards for the profession. The keystone to these efforts has been in the realm of self-regulation. In 1967 under the leadership of Dr Frank McCarthy and Dr Howard Davis, the Southern California Society of Oral Surgeons instituted a voluntary office anesthesia evaluation program.^{11,37} By 1970 the Office Anesthesia Evaluation became a requirement for membership in the society. In keeping with the concept of a competent anesthesia team for office-based surgery, these same surgeons in California developed a training program for surgical assistants. In 1971 the ASOS followed the lead of the California Society and added the office anesthesia evaluation to its national program. This was ultimately followed by an anesthesia assistant training program as well.

In 1970 a liaison committee consisting of the ASOS, the ADSA, the ADA (American Dental Association), and the ASA (American Society of Anesthesiologists) was formed.^{11,48} This assured continued training of dentists in anesthesia residency training programs. True to the path of training established by the liaison committee, current oral and maxillofacial surgery residents train side by side with anesthesiology residents in university and hospital training programs throughout the US.

An important affiliation in maintaining high standards in the delivery of anesthesia services by oral and maxillofacial surgeons has been the close relationship which exists between the AAOMS and the ASA. To ensure that guidelines for safe anesthesia practice are consistent, the ASA reviews both the *Office Anesthesia Evaluation Manual*⁴⁹ and the *Parameters of Care*⁵⁰ document published by AAOMS.

To ensure the effectiveness of the efforts of OMFS training programs, continuing education programs, and the office anesthesia evaluation program, AAOMS maintains an Outcomes Committee. In 2003 the committee conducted the most extensive study of office-based ambulatory anesthesia ever reported.⁵¹ The study found that “95% of the time the operating surgeon is the anesthetist and performs the operation with a support team of at least 2–3 individuals and monitoring techniques”. The study reported a safety record which was unexcelled and a high level of patient acceptance. Another recent study of nearly 50 000 anesthetics supported the concept of the opera-

tor-anesthetist model. The study concluded there was “no statistically significant difference in complication rates between anesthesia administered by an OMS functioning as an anesthetist–surgeon and anesthesia administered by an anesthesiologist”.⁵² Smaller studies from Massachusetts,⁵³ a residency training program,⁵⁴ and a private office setting⁵⁵ all point to equally low or lower complication rates in the operator-anesthetist setting.

Safety frequently goes hand-in-hand with quality and patient satisfaction.⁵⁶ In a study of over 34 000 OMFS patients Coyle⁵⁷ concluded that the patients were overwhelmingly satisfied with deep sedation and general anesthesia provided in the oral and maxillofacial surgery office-based ambulatory setting. Patient dissatisfaction was most closely related to anxiety, pain, vomiting, and awareness during surgery. The low incidence of these undesirable effects was responsible for the high levels of patient acceptance of office-based anesthesia in oral and maxillofacial surgery. These findings are consistent with those reported in other types of ambulatory anesthesia.⁵⁸

Goals and objectives in the administration of anesthesia

The initial intuitively derived concepts

Early on, both those who administered anesthesia and the patients who received it, established intuitively derived requisites for an anesthetic. The essential requisite of lack of toxicity was derived from experience with chloroform, while that of non-flammability was fostered by fires and explosions caused by ether. Other characteristics would ultimately find a place in the works of John Lundy when he published his papers on balanced anesthesia in the 1920s,^{23,59} three quarters of a century after the discovery of the anesthetic properties of nitrous oxide and ether. These essential characteristics included: (1) satisfactory analgesia, such that the patient experienced no pain associated with the surgical procedure; (2) a state of relaxation such that the patient would remain immobile during the procedure and exhibit relaxed muscle tone; (3) lack of awareness and amnesia to the surgical event; (4) loss of consciousness or hypnosis. When all of these requisites were not met, the anesthetic was considered a failure, as was the case in Wells’ demonstration of nitrous oxide at Massachusetts General Hospital in 1844.

Throughout the history of anesthesia patient acceptance has been a primary moving force in the development of improved and safer anesthetic techniques. On 28 January 1848, within weeks of Simpson’s successful demonstration of chloroform anesthesia, a young woman, Hannah Greener, died following chloroform anesthesia.^{60,61} Word soon spread round the world. This generated such fear

that some soldiers in the Northern army during the America Civil War declined anesthesia altogether rather than undergo anesthesia with chloroform.^{6,7} Similarly, patients were frightened of the flammability of ether, and dreaded its foul smell and the frequent nausea which patients experienced post-operatively. Consequently, the list of requisites for the ideal anesthetic began to grow almost immediately after the inception of anesthesia.

The initial approach

For the better part of the 150 years since the discovery of surgical anesthesia in the 19th century, a mono-anesthetic agent was sought. It was hoped that this agent would possess the full complement of the desirable characteristics discussed above. Several inhalation agents were investigated in the late 19th century and early 20th century in an attempt to find a single ideal agent which possessed the desired properties.² However, Lundy's work in the 1920s gave birth to a whole new concept: that there was in fact no ideal single agent and that multiple agents at lower doses could be administered to achieve the desired characteristics of the anesthetic.⁶² Furthermore, since the agents were given at lower doses, their side-effects could be minimized. As the 20th century progressed, the newer agents such as the benzodiazepines and the new narcotics were added to smooth the barbiturate-based intravenous anesthetic, providing validity to Lundy's concepts which were proffered well before their time.

Requisites for the ideal anesthetic in the modern era

In the modern practice of oral and maxillofacial surgery the original requisites for an anesthetic have been joined by a host of other attributes that are considered desirable in an anesthetic.^{58,63-65} Meeting these objectives not only provides a safe, but also a comfortable, anesthetic experience for patients as well. Many of these objectives have been derived from patient surveys to determine which outcomes are important to avoid.⁶⁶ These objectives include:

1. Maintenance of the body's homeostatic mechanisms.
2. Rapid onset and recovery.
3. Freedom from postoperative nausea and vomiting.
4. A feeling of well-being on emergence (postoperative euphoria).
5. A technique which can be used in medically compromised patients.
6. A technique which can be easily adapted for use in children and seniors.
7. A technique which retains sufficient pharyngeal and laryngeal reflexes to prevent aspiration, but not to predispose to laryngospasm.

8. Capable of being administered easily with familiar anesthetic equipment and supplies.
9. A technique well documented in the anesthesia literature and consistent with techniques currently in use by anesthesiologists.
10. A technique which is cost effective.

The essential role of the basic sciences in the advancement of anesthesia

Attempts to arrive at an ideal anesthetic in early years were thwarted by a lack of understanding of the areas of the CNS in which anesthetics exert their effects and how those effects are expressed. Initially anesthetic investigation was conducted on a purely empirical basis, i.e. subjects were given anesthetics, and the effects observed and recorded. As we have seen, often the first subject was, indeed, the investigator himself. Once an agent had been demonstrated to have efficacy, means were found to manufacture the agent, and it was then marketed. In the modern era research and development is often economically motivated. Large pharmaceutical corporations conduct research, or fund research at an academic institution, in order to identify agents which will ultimately provide a profit for the corporation. Regardless of the economic motivation, pharmaceutical research has led to a greater understanding of the nervous system, which in turn guides the search for new and improved anesthetic agents.

Areas of the brain affected by anesthetic agents

In the previous section we reviewed the extensive list of characteristics which are today considered essential in an anesthetic agent. An investigator attempting to identify such agents must have an appreciation of the multiple areas of the brain which are affected by anesthetics.⁶⁷ We will now examine some of the principal areas in which anesthetics exert their activity. The anesthetist must understand where these agents work to effectively provide a truly "balanced" anesthetic. Agents and dosages must be tailored to the individual case to provide an optimal anesthetic which is neither too light nor too deep. Too much of one or more agents can lead to a level of depression which can prolong recovery and compromise homeostatic mechanisms for respiration and cardiovascular stability. On the other hand, inappropriately chosen agents and low dosages may lead to an anesthetic that is so "light" that the patient is overly aware of the surgery.⁶⁸

The limbic system and the centers for memory

Emotion and memory are intimately related, and it is difficult to discuss one without consideration of the

other.^{69,70} Emotion exerts a powerful influence on what we notice and remember. We tend to recall more clearly those events which are related to strong feelings such as pain and unhappiness on the one hand, and joy and pleasure on the other. In providing anesthesia we must deal with the patient's emotionally imprinted memory "baggage" preoperatively and hope to imprint less unpleasant memories postoperatively.

The term "limbic" (Latin for rim) was originally given to the rim of brain tissue surrounding the brainstem (Fig. 6b.5) in the works of Willis in 1667.⁷¹ In the 1850s, Paul Broca defined the limbic lobe with two of its more prominent components, the cingulate gyrus and parahippocampal gyrus and a number of associated nuclei. Initially these areas of the brain were thought to be related to the sense of smell. However, in the 1930s Papez speculated that the function of these structures was related to emotion.⁷² He suggested various pathways amongst the nuclei which ultimately became termed the "Papez circuit". At about the same time that Papez was developing his theories, Heinrich Klüver and Paul Bucy removed the medial aspect of the temporal lobe in Rhesus monkeys, thereby destroying much of the limbic system. The animals developed hyperactivity and hyper-

sexuality and demonstrated marked changes in emotional behavior.⁷³ Over the years, investigators such as MacLean in the 1970s and Nauta and Domenick in the 1980s extended the areas thought to be involved in emotion to adjacent cortical and thalamic locations and the amygdala.^(71,74-76) The latter important structure was studied by John Downer at the University College in London in the 1950s and appears to control aggressive behavior and mediate the neuroprocesses which invest sensory experience with emotional significance.⁷⁷

In view of the various areas of the brain which are involved in emotion, some current authors feel that the term "limbic system" as a characterization for the brain's emotional processes is archaic. They feel that multiple systems are involved and that alternative terms should be developed.^{78,79} However, to date, no one has proposed a universally accepted term. Regardless of whether there is a single limbic system or multiple systems involved in emotion, human emotional response represents a spectrum ranging from calm and placidity at one end and anxiety, aggression, and rage at the other. From an anesthetic standpoint, the pharmacologic category of drugs which most profoundly affects this system (or systems) is the benzodiazepines.

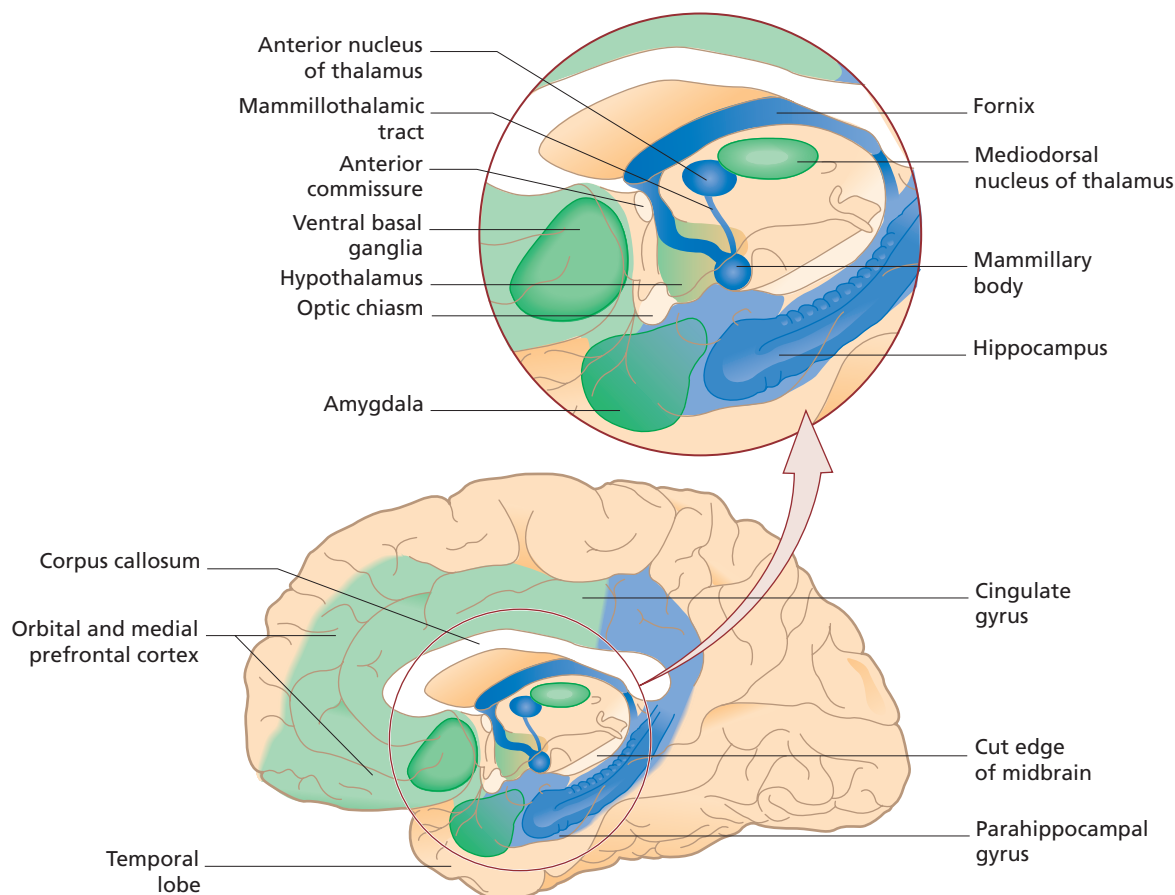


Fig. 6b.5 Modern conception of the limbic system. Especially important components of the limbic systems not emphasized in early anatomical accounts are the orbital and medial prefrontal cortex and the amygdala. These telencephalic regions, together with related structures in the thalamus, hypothalamus, and ventral striatum, are especially important in the experience and expression of emotion (green). Other parts of the limbic system, including the hippocampus and the mammillary bodies of the hypothalamus, are no longer considered important neural centers for processing emotion (blue). (Reproduced from *Neuroscience*, 4th edn. Sunderland, MA: Sinauer Associates, 2008; 741, Fig. 29.4 by permission. Copyright © 2008 Sinauer Associates Inc.)

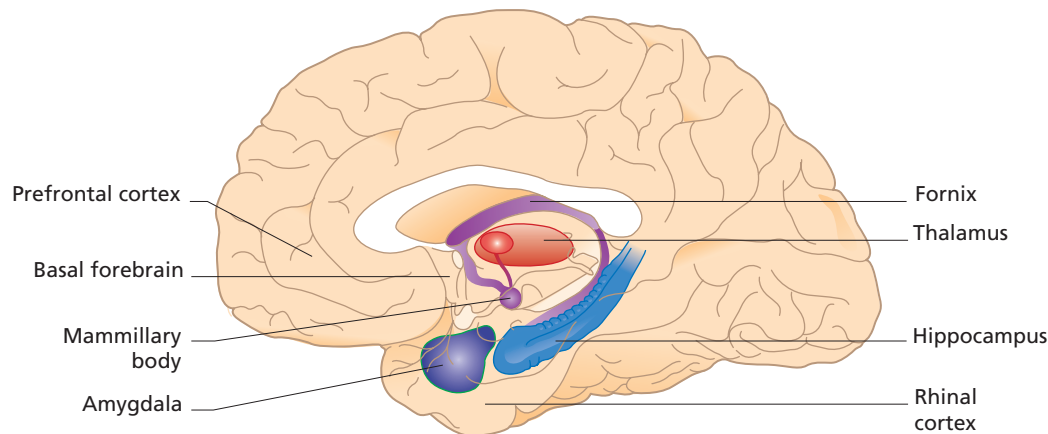


Fig. 6b.6 Brain areas which are thought to give rise to declarative memory. Of primary importance is the hippocampus which is in close approximation and has multiple connections with the amygdala. (Reproduced from *Neuroscience*, 4th edn. Sunderland, MA: Sinauer Associates, 2008; 800, Fig. 31.7 by permission. Copyright © 2008 Sinauer Associates Inc.)

Similar to emotion, memory involves multiple brain areas (Fig. 6b.6).⁸⁰ One of the primary amongst these is the hippocampus, which is in close approximation and has multiple connections with the amygdala. It has been recently demonstrated that extra synaptic alpha-5 GABA_A receptors in the hippocampus are vital to the amnesia-inducing effects of certain general anesthetics. It currently appears that the amnesic effects of anesthetic agents involve circuits in the hippocampus, amygdala and rostral areas of the cortex which have been implicated in learning and memory.⁸¹ As discussed above, emotion markedly influences memory, and it is not surprising that the pharmacologic agents which have greatest influence on emotion, also tend to affect memory, and therefore amnesia, during anesthesia for surgery.⁸²

The “reticular activating system” and thalamus

From the time of the initial demonstration of surgical anesthesia by Wells and Morton, loss of consciousness has been considered the primary goal of anesthesia. Thus the casual observer would assume that a considerable body of research would have been conducted to better understand loss of consciousness. However, with the exception of the studies of Moruzzi and Magoun in the 1940s discussed below, there was little investigation of consciousness and wakefulness during the entire 20th century. Both George Mashour,⁴ an anesthesiologist, and John Searle,⁸³ a philosopher, have commented on the paucity of studies on consciousness during the 20th century. In their reviews of the literature, both have found that, for inexplicable reasons, consciousness lost legitimacy as a topic of serious inquiry.⁴ However, within the last decade of the century a landmark conference consisting of physicians, neuroscientists, cognitive scientists, philosophers, and physicists was convened to discuss the possibility of an interdisciplinary approach to the study of consciousness. Soon numerous studies got under way, several of which are discussed below.

The term “reticular activating system” was proposed by Moruzzi and Magoun during their studies in the 1940s, and led to the concept that there might be a neural system in the midbrain and rostral pons that was responsible for wakefulness and its transition into sleep. The term has continued to be used since that time.^{84,85} However, once again, as in the case of emotion and memory, wakefulness does not appear to be controlled by a simple single “system”. Nonetheless, the reticular formation in the brainstem does appear to be a predominant component of “arousal”, presumably by its activation of various subcortical and cortical structures.^{86,87}

The reticular formation (Fig. 6b.7) is a somewhat loose group of cells that is spread from the brainstem to the thalamic area with a longitudinal orientation. Although originally described as being a “sparse network”, it contains a number of discreet nuclei and exhibits discreet neurochemical profiles.⁸⁶ Discharge rates within the neurons of the reticular formation have been correlated with arousal and active sleep by investigators such as Steriade *et al.*⁸⁸ However, the reticular formation also contains neurons which are responsible for muscle tone, nociception, and the control of breathing and the cardiovascular system. Consequently, the reticular formation is indeed complex, as opposed to the original concept of a “sparse network”.⁸⁵ Anesthetic agents which tend to exert their primary effects on the various centers for wakefulness are the barbiturates and propofol⁸⁹ amongst the intravenous agents and the halogenated hydrocarbons among the inhalation agents.

Consciousness is manifested in the cortex, and some studies, such as those by Velly *et al.*,^{90,91} suggest that the cortex itself may well be the main site of action of anesthetic agents in producing loss of consciousness. However, other investigators have proposed alternative mechanisms. An important structure in the path from the brainstem to the cerebral cortex is the thalamus⁸⁰ (see Fig. 6b.5). One theory as to the mechanism whereby anesthetics induce unconsciousness is the presence of a so-called “thalamocortical switch”

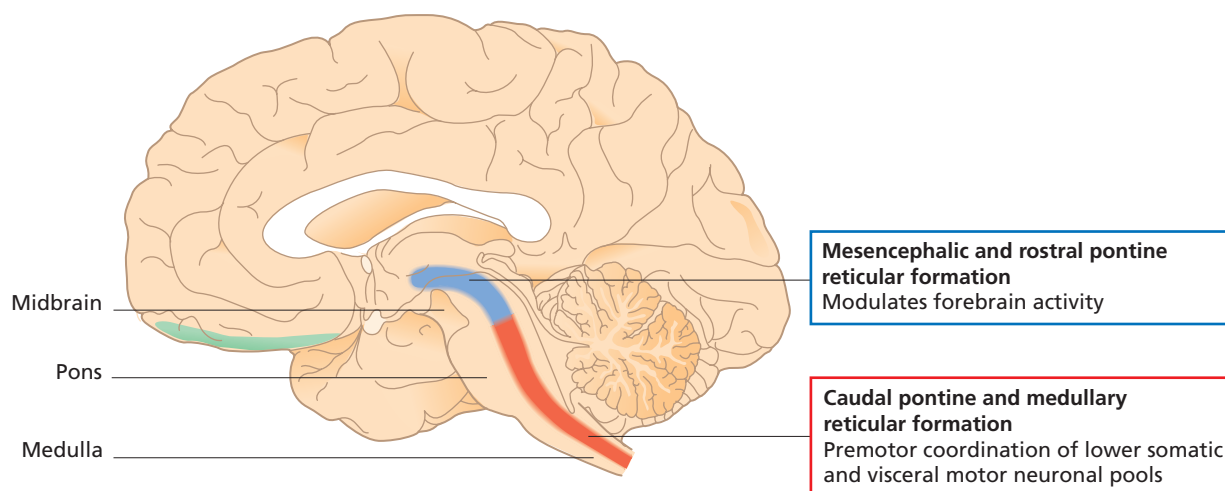


Fig. 6b.7 Brain areas which are thought to maintain consciousness. Consciousness appears to be dependent upon important interactions between the reticular formation and thalamocortical structures. (Reproduced from *Neuroscience*, 4th edn. Sunderland, MA: Sinauer Associates, 2008; 428, Fig. 17a by permission. Copyright © 2008 Sinauer Associates Inc.)

expounded by Alkire *et al.*⁹² Yet another study by John and Prichet⁹³ suggests that there may be an “anesthetic cascade” with a cascade of events leading to loss of consciousness. These include hyperpolarization of thalamic and cortical cell membranes, and suppression of arousal areas in the pons and midbrain. Regardless, it appears that there are important thalamic–cortical and brainstem interactions which are the most likely targets of anesthetic agents that render a patient unconscious.^{4,81,92,94–99}

Another important function of the thalamus is its relay function between sensory inputs and cortical perception.^{100,101} It is this relay which appears to be one of the primary targets of the dissociative anesthetic ketamine.⁶⁴ The patient enters a catatonic state in which he does not respond to sensory stimuli and remains motionless, apparently totally “dissociated” from his environment.³⁴

Central opioid receptor sites

Until the early 1970s, it was assumed that there was a single opioid receptor site in the CNS. However in 1973, radio ligand binding studies by three independent teams pointed to the presence of three types of opioid receptors. Each was given a Greek letter designation as follows: μ for the morphine type of receptor, κ for the ketocyclazocine type, and δ for the skf10047 (n-allylnormetazocine).¹⁰² The functions of the μ receptor are supraspinal and spinal analgesia, sedation, euphoria, slowing of gastrointestinal (GI) motility, inhibition of respiration, and modulation of hormone and neurotransmitter release.^{36,103} The δ subtype provides supraspinal and spinal anesthesia as well as modulation of hormone and transmitter release.¹⁰² Finally, the κ subtype, in addition to providing supraspinal and spinal analgesia, causes psychotomimetic affects and slows GI motility.¹⁰⁴

Until the late 1980s opioids were believed to exert their effects only centrally. However, we now know

that there are three levels at which they have activity: supraspinal sites, spinal sites, and peripheral sites.¹⁰⁵ In addition, there are two pathways along which there are sites at which opioids exert their affects. The first of these is the traditional afferent pain transmission pathway. Sites of action along this pathway include: (1) a direct action on inflamed or damaged peripheral tissues; (2) inhibition in the spinal cord;^{36,106} (3) probable central site in the ventrolateral aspect of the thalamus (Fig. 6b.8). The action of opioids at these spinal

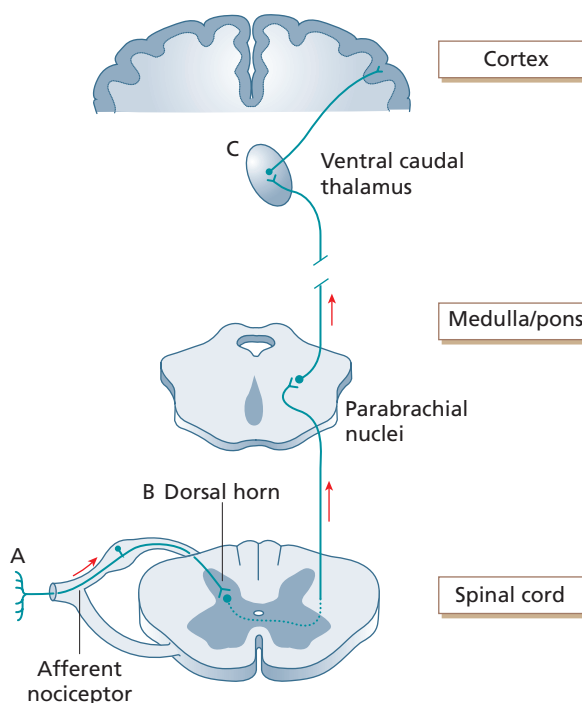


Fig. 6b.8 Putative sites of action of opioid analgesics on the afferent pain transmission pathway from the periphery to the higher centers. A: Direct action of opioids on inflamed or damaged peripheral tissues. B: Inhibition also occurs in the spinal cord. C: Possible sites of action in the thalamus. (Reproduced from *Basic and Clinical Pharmacology* (Katzung BG, ed.), 10th edn. New York: McGraw Hill, 2007. With permission of the McGraw-Hill Companies.)

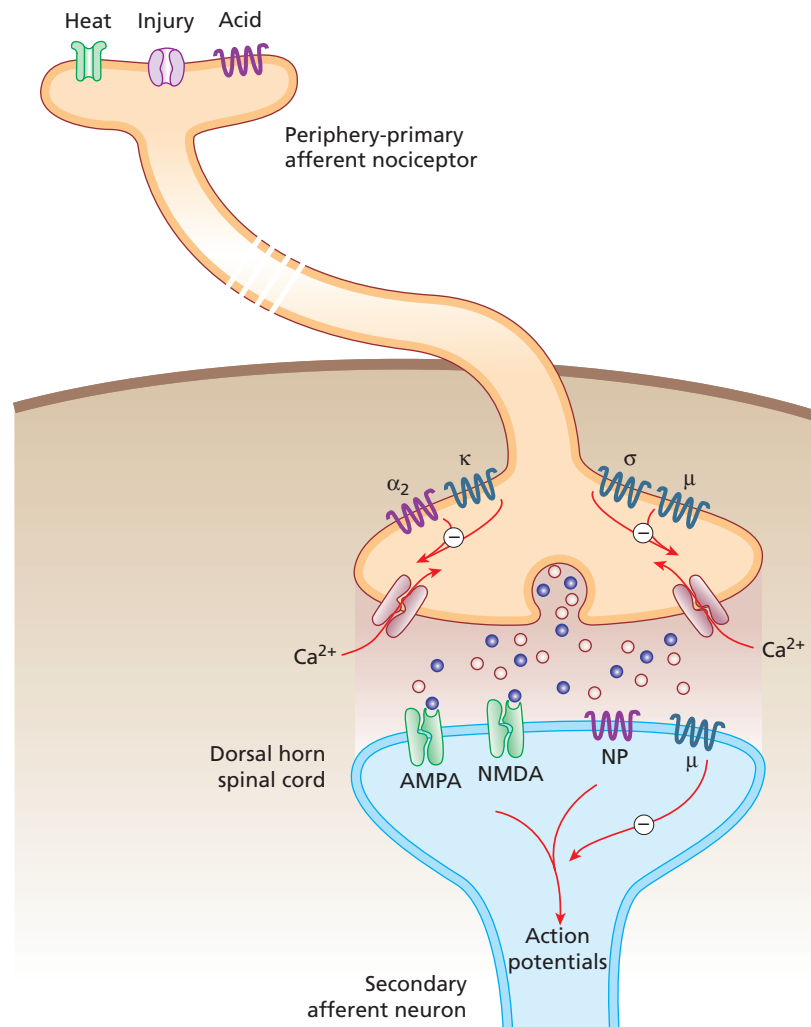


Fig. 6b.9 Action of opioids at spinal sites, mu: (μ), kappa (κ), and delta (δ) agonists reduce neurotransmitter release from the pre-synaptic terminals of nociceptive primary afferents. Mu agonists also hyperpolarize second-order pain transmission neurons by increasing K^+ conductance, evoking an inhibitory postsynaptic potential. (Reproduced from *Basic and Clinical Pharmacology* (Katzung BG, ed.), 10th edn. New York: McGraw Hill, 2007. With permission of the McGraw-Hill Companies.)

sites is illustrated in Fig. 6b.9. There is also a descending pathway (Fig. 6b.10) along which opioids exert their effects on pain-modulating neurons within the: (1) midbrain periaqueductal gray area;¹⁰² (2) the rostral ventral medulla; (3) the locus caeruleus. This indirectly controls pain transmission pathways by enhancing inhibition to the dorsal horn (Fig. 6b.11).¹⁰⁵

The vital centers and homeostatic receptor mechanisms

Most anesthetic agents and narcotics exert depressive effects on the body's normal homeostatic mechanisms for respiration and circulation. For instance, in the case of opioids there is dose-dependent depression of the ventilatory response to hypercapnia and hypoxia due to opioid action on the μ_2 receptors in the respiratory centers of the medulla.¹⁰⁵ In the case of propofol, there is a 50% reduction in ventilatory response to carbon dioxide and decrease in the acute ventilatory response to isocapnic hypoxia.¹⁰⁷ In addition, the

autonomic cardiovascular centers in the medulla are depressed by most anesthetics, leading to a loss of sympathetic vascular tone. The resulting vasodilation tends to cause hypotension.¹⁰⁶ This is true for almost all anesthetic agents except ketamine, which tends to cause a sympathetic response that leads to hypertension.³⁴

The critical realization that on a molecular basis all anesthetic drugs share a common mechanism of action

At the beginning of the 20th century Hans Meyer and Charles Ernest Overton^{108–110} made the observation that a wide spectrum of chemically dissimilar agents could produce general anesthesia. Furthermore, they determined that the potencies of the general anesthetics available at that time correlated well with their water/oil partition coefficient. These "lipid theories"

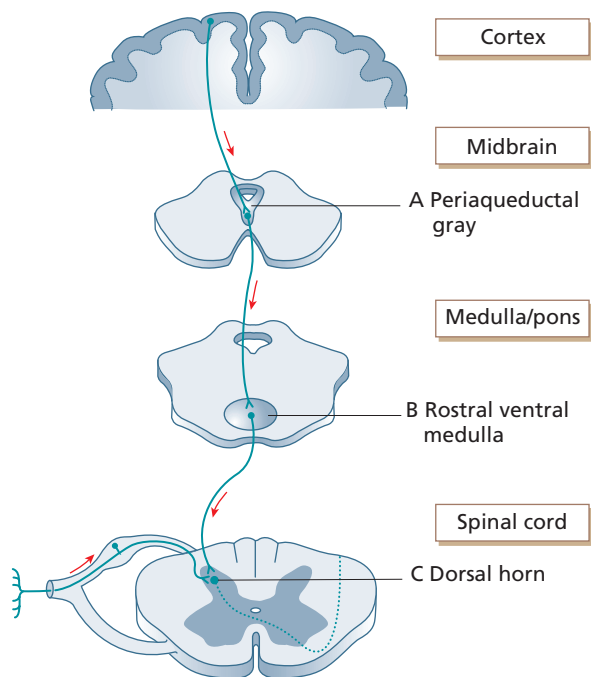


Fig. 6b.10 Opioid analgesic action on the descending inhibitory pathways. Sites of action on pain-modulated neurons in the mid-brain and medulla including the midbrain periaqueductal gray area (A), rostral ventral medulla (B), and the locus ceruleus indirectly control pain transmission pathways by enhancing descending inhibition to the dorsal horn (C). (Reproduced from *Basic and Clinical Pharmacology* (Katzung BG, ed.), 10th edn. New York: McGraw Hill, 2007. With permission of the McGraw-Hill Companies.)

were developed into the concept that anesthetic agents had a common non-specific mechanism. They theorized that the agents dissolve in the membrane of neurons and bring about a physical change such as thickening of the membrane. These changes were felt to alter membrane permeability to ions which then led to the anesthetic effect. The idea presented by Meyer and Overton was so convincing that it was looked upon as the primary mechanism of anesthesia for three quarters of a century. It was not until the closing decades of the century that Franks and Lieb¹¹¹⁻¹¹³ convincingly demonstrated that the actual mechanism behind ion flux was the action of general anesthetics on proteins that leads to opening and closing (or blocking) of ion channels in the nerve membrane. Thus the basic sciences have led to a critical realization in that it appears that on a molecular basis all inhalation and intravenous anesthetic agents share a common mechanism of action: they affect the movement of ions through ion channels in neuronal membranes.^{81,108,115-116} Consequently, one's understanding of the mechanism of action of anesthetics is dependent upon an appreciation of the elegant mechanism by which nerve impulses are transmitted through the nervous system.

Nerve impulse transmission is based on the movement of ions such as Na^+ , Cl^- and K^+ through channels in the neuronal membrane. Control of this movement takes place in one of four ways (see Fig. 6b.12).^{98,117}

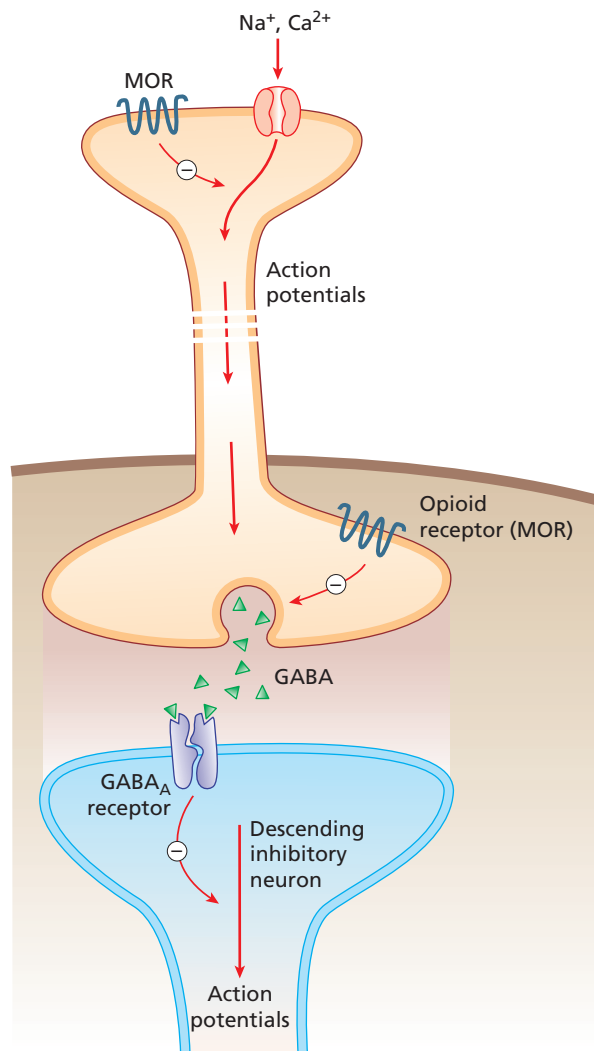


Fig. 6b.11 Modulating affect of descending pathways by μ opioid receptor mediated analgesia. Opioids inhibit an inhibitory GABAergic interneuron resulting in enhanced inhibition of nociceptive processing in the dorsal horn. (Reproduced from *Basic and Clinical Pharmacology* (Katzung BG, ed.), 10th edn. New York: McGraw Hill, 2007. With permission of the McGraw-Hill Companies.)

1. In voltage-gated channels, control is through a voltage sensor component of the protein.
2. In ligand-gated channels, control is through the binding of a neurotransmitter substance to the channel receptor (ionotropic receptors).
3. In G protein-coupled (metabotropic) channels a neurotransmitter activates an intermediary protein, the G protein, which in turn interacts directly with the ion channel.
4. In diffusible second-messenger metabotropic ions channels the neurotransmitter activates a G protein which then activates an enzyme. The activated enzyme generates a diffusible second messenger which interacts with ion channels.

It was originally thought that the action of anesthetics was due to their effects on ligand-gated channels. However, it has now been demonstrated that voltage-

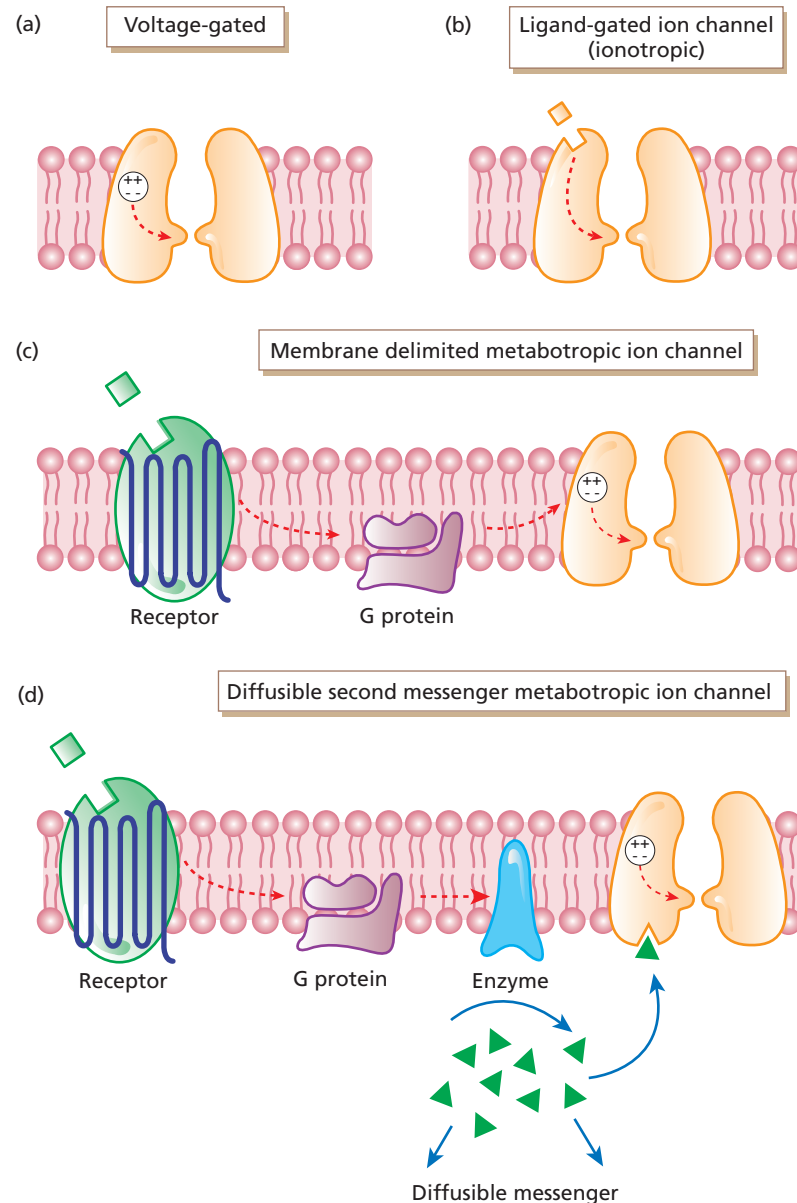


Fig. 6b.12 Types of ion channels and neurotransmitter receptors in the CNS. (a) Voltage-gated channel in which a voltage sensor component of the protein controls the gating (broken arrow) of the channel. (b) Ligand-gated channel in which binding of the neurotransmitter to the ionotropic channel receptor controls the gating (broken arrow) of the channel. (c) A G protein-coupled (metabotropic) receptor, which when bound, activates a G protein that in turn interacts directly with the ion channel. (d) A G protein-coupled receptor, which when bound, activates a G protein that activates an enzyme. The activated enzyme generates a diffusible second messenger such as cAMP, which interacts with the ion channel. (Reproduced from *Basic and Clinical Pharmacology* (Katzung BG, ed.), 10th edn. New York: McGraw Hill, 2007. With permission of the McGraw-Hill Companies.)

gated sodium channels, as well as calcium channels, are also affected by anesthetic agents.

Transmission of the action potential along the axon

The mechanism of action potential propagation is illustrated in Fig. 6b.13. A depolarizing stimulus on the axon opens the voltage-sensitive sodium channels in the region.¹¹⁷ The open channels allow passage of ions into the neurons.^{98,118} This results in depolarization of the membrane potential that generates an action potential. The local current flow depolarizes the membrane potential in the adjacent region that leads to opening of the sodium channels in the neigh-

boring membrane. This local depolarization triggers an action potential in this region as well, which then spreads in a continuing cycle to the end of the axon.^{118–120}

Impulse transmission through the synapse

When the action potential reaches the synapse, voltage-sensitive calcium channels are activated in the membrane of the terminal bouton (Fig. 6b.14). The calcium flowing into the terminal promotes the fusion of synaptic vesicles with the presynaptic membrane.¹²¹ Neurotransmitter substances contained in the vesicles are released into the synaptic cleft and diffuse to the receptors on the postsynaptic mem-

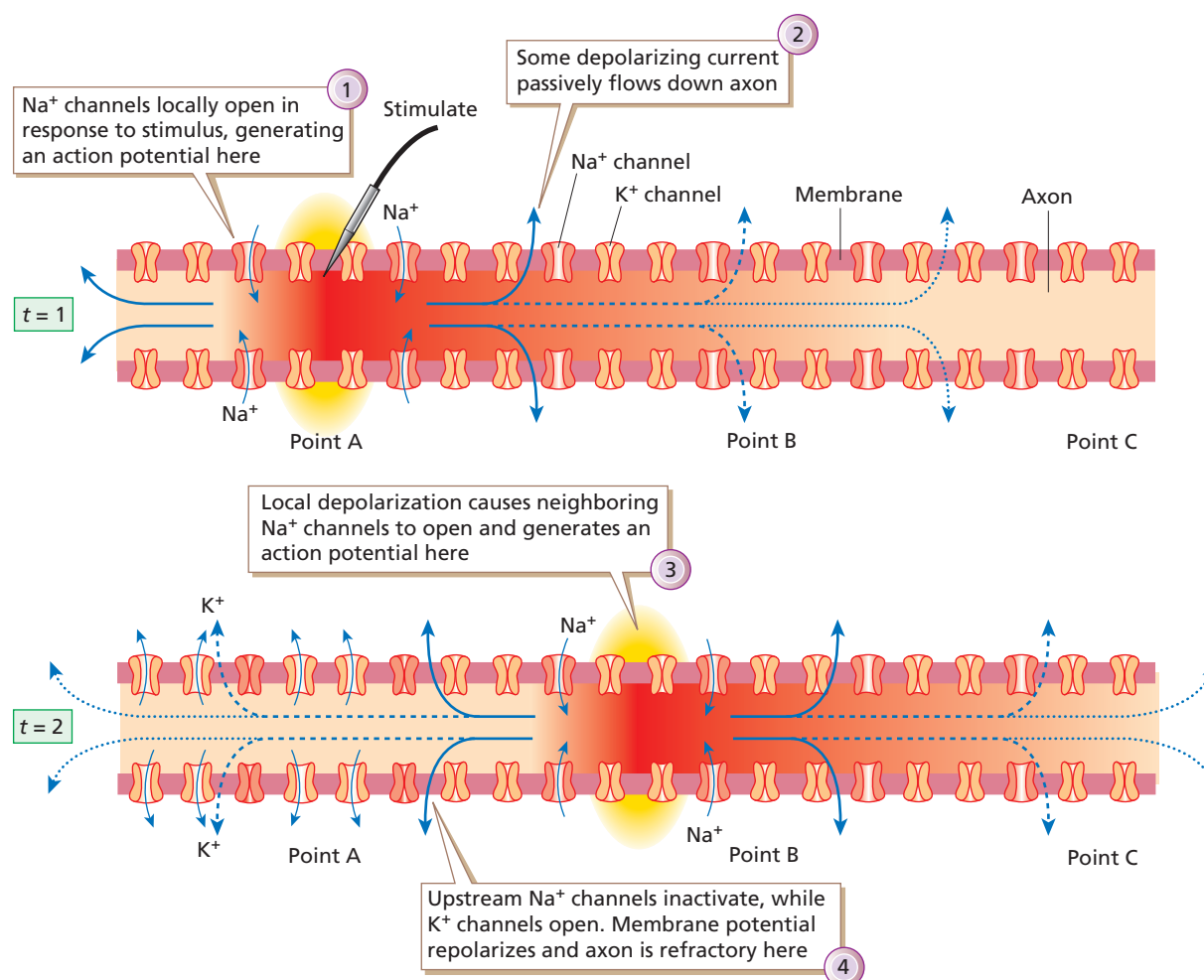


Fig. 6b.13 Action potential conduction requires both active and passive current flow. Depolarization opens Na⁺ channels locally and produces an action potential at point A of the axon (time $t = 1$ above). The resulting inward current flows passively along the axon, depolarizing the adjacent region (point B) of the axon. At time 2 ($t = 2$) the depolarization of the adjacent membrane has opened Na⁺ channels at point B. This results in the initiation of the action potential at this site and additional inward current that again spreads passively to an adjacent point (point C) farther along the axon. The cycle continues along the full length of the axon. As the action potential spreads, the membrane potential repolarizes due to K⁺ channel opening and Na⁺ channel inactivation. This creates a "wake" behind the action potential during which the axon is temporarily refractory to depolarization. The latter prevents backward propagation of the impulse (time 4 above). (Reproduced from *Neuroscience*, 4th edn. Sunderland, MA: Sinauer Associates, 2008; 55, Fig. 3.12 by permission. Copyright © 2008 Sinauer Associates Inc.)

brane.¹¹⁷ When the transmitters bind to their receptive sites, there is a brief change of membrane conduction in the postsynaptic cell. This leads to an increase in sodium and potassium permeability which enables impulse conduction through the postsynaptic neuron. Virtually all of the drugs that act in the CNS produce their effects by modifying one of the steps in chemical synaptic transmission.¹¹⁷ In the postsynaptic region the transmitter receptor provides the primary site of drug action. However, it now appears that there are extra-synaptic receptors as well.^{81,122}

Neurotransmitter substances which enable transmission of the impulse from one neuron to the next

GABA_A receptor

As we have seen, virtually all anesthetic agents exert their action on ligand-gated ion channels. Gamma

amino butyric acid (GABA_A) is the most important inhibitory neurotransmitter in the mammalian CNS.¹²³ As many as one third of all synapses in the CNS are GABAergic.¹²⁴ The GABA_A receptor (Fig. 6b.15) is a member of a super family of ligand-gated ion channels that include the nicotinic acetylcholine receptor, the 5-hydroxytryptamine type 3 receptor (5HT₃), and the glycine receptor.¹²⁵ The GABA_A receptor has a pentameric structure assembled from five subunits (Fig 6b.15).¹²⁶ The most common combination of subunits is two α sub-units, two β subunits, and one γ subunit. Activation of GABA receptors has been shown to initiate an influx of chloride ions which leads to hyperpolarization of the nerve membrane. This blunts excitatory input and reduces the excitability of the affected neurons.¹²⁴

Neurons which contain GABA receptors are found throughout the brain, including the cortex, the basal forebrain, the thalamus, the hypothalamus, and the brainstem. During non-rapid eye movement (NREM)

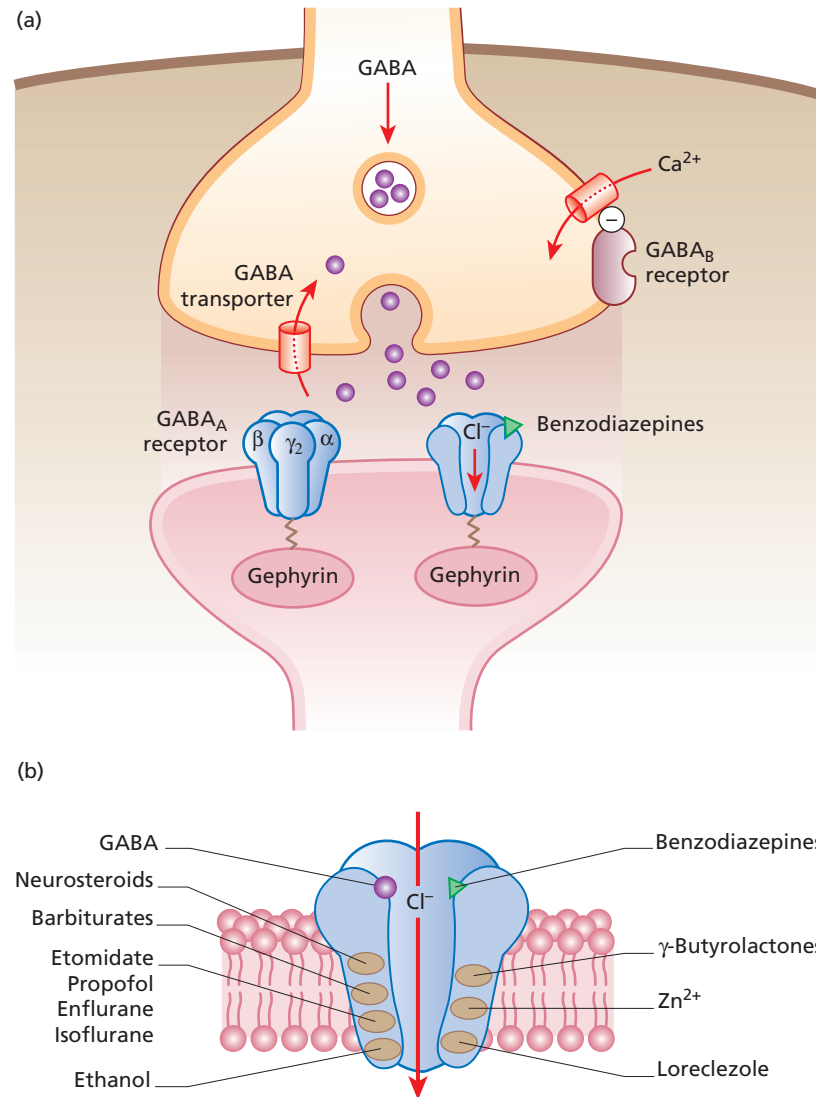


Fig. 6b.14 (a) A GABAergic synapse. Calcium flowing into the terminal promotes fusion of the synaptic vesicles with the presynaptic membrane. Neurotransmitter substances are released from the vesicles into the synaptic cleft and diffuse to the receptors on the postsynaptic membrane. When the transmitters bind to the receptor sites, there is a brief change of membrane conduction in the postsynaptic cell. On the cytoplasmic side these channels are directly linked to the anchoring protein gephyrin. (b) GABA_A binding sites of the GABA_A receptor. In addition to the binding sites for the physiologic transmitter GABA, the receptor has modulatory binding sites for a number of anesthetic agents such as the benzodiazepines, barbiturates, propofol and inhalation agents, as well as halogenated inhalation agents. (Reproduced from *Anesthetic Pharmacology: Physiologic Principles and Clinical Practice* (Evers SA, Maze M, eds.). Philadelphia, PA: Churchill Livingstone, 2004; 418, Fig. 25-2 by permission.)

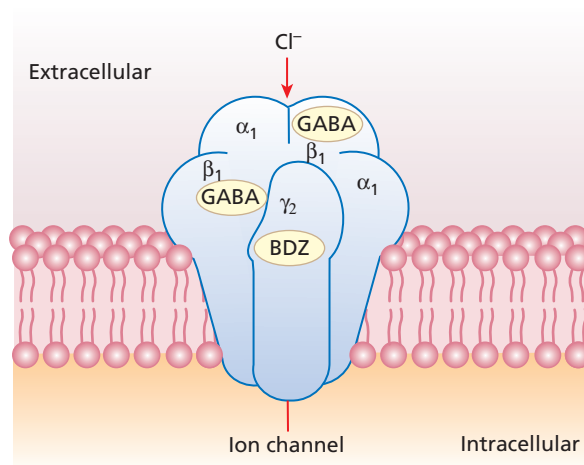


Fig. 6b.15 The GABA_A receptor has a pentameric structure assembled from five subunits: two α subunits, two β subunits and one γ subunit. Activation of the receptor initiates influx of chloride ion which leads to hyperpolarization of the nerve membrane. (Reproduced from *Miller's Anesthesia* (Miller RD, ed.), 6th edn. New York: Elsevier/Churchill Livingstone, 2005; 337, Fig. 10-14 by permission. Copyright © 2005 Elsevier.)

sleep GABA is released into the cerebral cortex in the highest concentration.¹²⁷

A large number of agents target the GABA_A receptor. These include the benzodiazepines, propofol, barbiturates, inhalation anesthetics, and ethanol (Fig. 6b.14b).¹²⁸ The most extensively studied of the drugs which interact with the GABA_A receptor are the benzodiazepines.^{127,129} The classic bioaffinity benzodiazepine binding site is located between the α_1 and γ_2 subunits.¹³⁰ However, there are a number of subtypes of α , β and γ receptor subunits. The various actions of the benzodiazepines appear to be mediated by different receptor subtypes. For instance, the sedative action appears to be mediated by the α_1 receptor while the anxiolytic action is mediated by α_2 , α_4 , and γ_5 receptors.¹²⁷ Anterograde amnesia and anticonvulsant properties seem to be mediated through α_1 receptors and anxiolysis and muscle relaxation by α_2 receptors.¹³¹

Propofol can be used for either sedation or for general anesthesia. For intravenous anesthetic agents it appears that loss of consciousness is mediated purely through β_3 -containing receptors while β_2 subunit receptors mediate the sedative properties of these anesthetics.¹³² Several studies suggest the β subunit M₃ segment is the binding site for propofol's direct anesthetic action.¹³³

It was initially thought that all GABA_A receptors were located in synapses. However, more recent evidence indicates that there may be extra-synaptic GABA_A receptors as well.¹²⁴ These appear to be sensitive to low concentrations of anesthetics and contain an $\alpha 5$ subunit which most synaptic receptors lack.¹²²

The NMDA receptor and glutamate

Glutamate is the main excitatory transmitter in the brain and the spinal cord. Approximately 75% of the excitatory transmission of the brain is due to glutamate.¹¹⁸ One of its ionotropic receptors is the NMDA or N-methyl-D-aspartate receptor (Fig. 6b.16).^{134,117} The ion channel is permeable to calcium, as well as sodium and potassium to a lesser extent.¹³⁴ Like the GABA_A receptor the NMDA receptor is a pentameric structure for which six subunits have been identified. Simple binding of glutamate does not open the channel. Glycine is a co-transmitter and the channel is normally blocked by Mg²⁺ ion.^{118,135} Consequently, the neuron must be strongly depolarized so that the Mg²⁺ ion is expelled before the channel opens.^{117,118}

The NMDA receptor contains several binding sites. Glutamate and glycine have separate binding sites, while the recreational hallucinogen phencyclidine and the dissociative anesthetic ketamine, have yet another binding site inside the channel.¹¹⁸

Other pertinent receptors

Other neurotransmitters modulate ligand-gated ion channels that are affected by anesthetic agents, presumably to a lesser extent than do the GABA_A and NMDA receptors. For instance, ketamine appears to

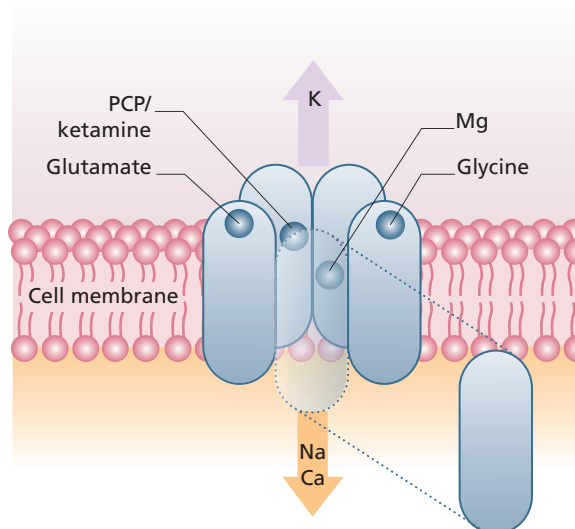


Fig. 6b.16 The NMDA receptor is a pentameric structure for which six subunits have been identified. Channel opening also depends upon the co-transmitter glycine as well as expulsion of Mg²⁺ ion by depolarization of the neuron. (Reproduced from Kohrs R, Durieux ME. Ketamine: teaching an old drug new tricks. *Anesth Analg* 1998; 87: 1186–93 by permission. Copyright © 1998 Lippincott Williams & Wilkins.)

be a potent inhibitor of the central nicotinic acetylcholine receptor (nAChR).¹³⁶ Agents like ketamine which inhibit nAChR have analgesic effects at subanesthetic concentrations, while those which do not, such as etomidate and propofol, possess no analgesic activity. Since nicotinic acetylcholine receptors are sensitive to ketamine, this interaction may be in part responsible for the dissociative and analgesic properties of ketamine. However, overall the contribution of acetylcholine activity to general anesthesia is less clearly defined than that of GABA_A and NMDA receptors.¹³⁷

Yet another neurotransmitter which may be affected by anesthetics is the glycine receptor.¹²³ Compared to the GABA_A receptor the glycine receptor has a very modest pharmacologic profile. Currently there are no therapeutically useful ligands that act at the glycine receptor site, despite its pivotal role in providing innervation in the brainstem and spinal cord.

Classification of anesthetics on the basis of molecular targets

As we have seen, all general anesthetic and sedative agents do not have the same molecular target. Solt and Forman¹³⁸ have proposed a classification of anesthetic agents based on their molecular targets. Their classification is illustrated in Table 6b.1.

Multiple sites of action and receptors in the CNS dictate a balanced approach

Because multiple receptors in the CNS are involved in anesthesia, there is no currently available single agent which interacts with all of the appropriate

Table 6b.1 Anesthetic classification based on clinical features and molecular targets. (Modified from: Solt K, Forman SA. Correlating the clinical actions and molecular mechanisms of general anesthetics. *Curr Opin Anaesthesiol* 2007; 20(4): 300–6, by permission. Copyright © 2007 Lippincott Williams & Wilkins.)

	Group 1	Group 2	Group 3
Anesthetic agents	Propofol, etomidate, barbiturates	Nitrous oxide, ketamine	Halogenated agents
Clinical features	Strong hypnotics, strong amnestics	Weak hypnotics, potent analgesics	Strong hypnotics, strong amnestics
Molecular targets	GABA _A receptor	NMDA, nCAhR receptors	GABA _A , glycine, glutamate, and nAChR receptors

receptors. In addition, it is highly unlikely on a statistical basis that a monoanesthetic agent could ever be developed that would provide the interaction with all the desired receptors. Interestingly the empirically derived approach of using a balanced anesthetic is consistent with what is currently known about anesthetic agents from a scientific standpoint. Consequently, when anesthesiologists and anesthesiologists seek to develop an anesthetic approach for outpatient surgery, multiple agents must be used with a balance of their various characteristics. In many cases, it is not sufficient to merely balance such primary characteristics as hypnosis and dissociation. Often the so-called “side-effects” must be balanced as well. This will often negate the untoward influence of those side effects.¹³⁹

The current status of office anesthesia in the oral and maxillofacial surgery practice

The maintenance of high anesthesia standards for the administration of anesthesia in oral and maxillofacial surgery has enabled oral and maxillofacial surgeons to provide a wide range of outpatient anesthesia services for their patients. The vast majority of procedures performed by members of the specialty can be treated with anesthesia administered by the operator–anesthetist.⁵² This includes anesthesia for ASA I, II, and low-level ASA III patients. For high-level ASA III patients and ASA IV patients, treatment is best performed in an outpatient surgery center or hospital setting with a separate anesthesiologist or anesthetist.¹⁴⁰

What enables the delivery of safe and comfortable anesthesia for patients in a modern office setting is the maturing perspective on the nature of the anesthesia (as discussed above). During the late decades of the 20th century and the first decade of the 21st century advances in basic science have provided a far more complete understanding of where and how anesthetics work in the CNS. This has in turn led to the development of anesthetic techniques combining multiple agents that make it possible for surgeons to perform procedures in their offices which heretofore could only be performed in a hospital. Historically an empirical approach has been used to select those agents which appeared to have more desirable characteristics than their predecessors. In this section we will explore the currently preferred agents. Although

the majority of surgeons prefer using the agents listed, the list is not intended to be exhaustive. There are surgeons who continue to use some of the “older” agents and techniques to provide a safe and comfortable anesthetic experience for their patients. We will examine the primary anesthetic agents propofol and ketamine, as well as the various ancillary agents which are used, including anxiolytics, narcotics, and anticholinergics.

Primary anesthetic agents

Propofol

Until the waning decades of the 20th century, ultra-short-acting barbiturate anesthetics such as thiopental and methohexital reigned supreme as the intravenous agents of choice in hospital operating rooms, outpatient surgery centers and oral and maxillofacial surgery offices. However, during the 1970s work began on a new family of anesthetic agents that in a relatively short period of time would surpass the daunting popularity of the barbiturates. By the end of the 1970s and the beginning of the early 1980s one of these agents was ready for release and reports began to appear in the literature. In 1977 Kay and Rolly¹⁴¹ reported on the first clinical trials, and in 1980 James and Glen³⁸ reviewed the research that had resulted in the development of an intriguing new agent, 2,6-diisopropylphenol, or propofol. It provided rapid onset and recovery, a “smooth” intraoperative course with stable operating conditions, as well as antiemetic and postanesthetic euphoric qualities.¹⁴² Over the next 25 years propofol would not only replace thiopental and methohexital, it would revolutionize the practice of outpatient anesthesia as well.

Propofol formulation

Propofol is a substituted phenol (2,6-diisopropylphenol) which belongs to a family termed “hindered phenols” (Fig. 6b.17).³⁸ It is insoluble in water and must be mixed with other agents to make it suitable for injection into the bloodstream. The initial propofol formulation was with Cremophor EL, but it had to be abandoned because of anaphylactoid reactions.^{34,143,144} Cremophor was replaced with an intralipid mixture similar to that which had been used for total parenteral nutrition. This resulted in a 1% aqueous emulsion in a vehicle that includes 10% soybean oil, 2.25% glycerol and 1.25% purified egg lecithin. This mixture

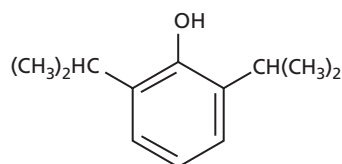


Fig. 6b.17 Propofol (2,6-diisopropylphenol). (Reproduced from *Clinical Anesthesiology* (Morgan GE, Mikhail M, Murray M, eds.), 4th edn. New York: McGraw-Hill, 2006. With permission of the McGraw-Hill Companies.)

is perishable, which necessitated the inclusion of preservatives.^{143,145} The first of these was EDTA employed by AstraZeneca. A less expensive formulation utilizing sulfites was introduced by Baxter, but sulfite allergy proved to be a problem with this formulation.¹⁴⁶ Ultimately benzyl alcohol, which had a time-honored track record as a preservative in other pharmaceuticals, was added by several manufacturers of generic products.¹⁴⁷ The expense of the initial propofol formulations limited its acceptance to some extent, but this problem has been overcome with the newer, far less expensive formulations.

Although improvements in the formulation have reduced the problem of water insolubility, they have not totally eliminated it. Consequently, considerable pharmacologic research has centered on developing water-soluble propofol-like drugs.¹⁴⁸ These have now become a reality and are discussed in the final section of the chapter, Future trends and advancements – on the horizon.

Mechanism of action

Like most intravenous anesthetic agents, propofol exerts its effects by positive modulation of the inhibitory function of GABA on the GABA_A receptor.³⁴ At lower concentrations, such as those used for sedation, propofol appears to facilitate the action of GABA in enhancing chloride conductance and hyperpolarization of the postsynaptic membrane. However, at higher concentrations, such as those used for general anesthesia, propofol directly activates chloride-channel opening (see Fig. 6b.14). Like barbiturates, propofol has a different receptor site from the benzodiazepine receptor site.¹⁴⁹ In addition to its primary activity on the GABA receptor, propofol is active at glycine activated chloride channels, nicotinic acetylcholine receptors, and some excitatory glutamate receptors.¹⁴⁵

Propofol exerts its activity in several areas within the brain, including the traditional reticular activating system (or “systems” as previously discussed). Some of its sedative effects are due to its action on GABA receptors in the hippocampus which results in inhibition of acetylcholine release within both the hippocampus and prefrontal cortex.¹⁵⁰ There is also widespread inhibition of NMDA glutamate receptors with modulation of sodium channel gating.¹⁵¹ In addition to its effects within the brain, there are also direct depressant effects on the neurons of the spinal cord due to action at GABA_A and glycine receptors.^{151,152}

Two interesting CNS actions of propofol which have led to its widespread popularity are a postoperative sense of well-being and its antiemetic effects. The former may be related to the increase in dopamine concentration in the nucleus accumbens which follows propofol administration.¹⁵³ This is a phenomenon similar to that which has been found to play a role in the euphoric effects of recreational drugs and drugs of abuse. Propofol’s antiemetic action may be related to its diminution of serotonin levels in the area postrema.¹⁵⁴

Distribution

Propofol is highly lipid-soluble and is rapidly redistributed to peripheral compartments, including muscle, fat, and skin. When viewed from the standpoint of a three-compartment model, it has an initial distribution half-life of 1–8 minutes, a slow distribution half-life of 30–70 minutes, and an elimination half-life of 4–24 hours.^{34,145} Although there are several factors which affect redistribution, the major ones are age, gender, and body weight. These effects tend to create different distribution groups:

1. Children – the drug is very rapidly redistributed in younger patients who may require at least a 50% increase in drug dose on a weight-for-weight basis.^{155,156}
2. Older patients – in the latter decades of life there is an increasing impairment of redistribution with a commensurate increase in sensitivity to propofol.^{157,158}
3. Gender – the drug is more rapidly redistributed in women who may require 10–15% larger doses of the drug on a weight-for-weight basis.^{159–161}
4. The obese – redistribution is altered in obesity and dosing should be based on lean body mass as opposed to total body weight.^{162,163}

Biotransformation and elimination

Propofol is rapidly metabolized through conjugation in the liver at a rate approximately 10 times faster than thiopental.¹⁶⁴ Interestingly, the pharmacokinetics of the drug are not affected by moderate cirrhosis.¹⁰⁶ There is extrahepatic metabolism, some of which takes place in the lung.¹⁶⁵

Hepatic conjugation of propofol results in inactive metabolites which are rapidly eliminated by renal clearance and less than 1% of the drug is excreted unchanged in the urine.¹⁶⁴ Renal insufficiency, even chronic renal failure, does not appear to affect the clearance of the parent drug.^{106,166}

Effects on organ systems

The primary effect of propofol on the cardiovascular system is a decrease in arterial blood pressure.¹⁴⁵ Propofol does not appear to impair myocardial contractility,^{144,145} but appears to exert its effects by decreasing vascular resistance secondary to inhibition of sympathetic vasoconstrictor activity.¹⁶⁷

Vasodilation may be in part due to a direct effect on intracellular smooth muscle calcium flux and increased release of nitric oxide by the vascular endothelium.¹⁶⁸ With age, the problem is accentuated by blunting of the baroreceptor response.¹⁶⁹

Relatively large induction doses of 2–2.5 mg/kg can produce a reduction in systolic blood pressure of up to 25–40%.³⁴ However, far less dramatic decreases are seen at the lower induction doses which are usually used in oral and maxillofacial surgery offices.¹⁷⁰ In addition, the hypotensive effect is largely overcome by stimulation,⁶² such as injections of local anesthetic solutions. Overall, the factors that tend to exacerbate hypotension are excessively large bolus doses, rapid administration of boluses and advanced age of the patient.¹⁰⁶ Heart rate does not undergo a significant change after induction with propofol. Several studies have suggested that propofol resets the baroreflex and thus reduces the tachycardic response to hypotension.¹⁷¹ Mild bradycardia may be noted within the first 10 minutes of induction.¹⁶⁶

Like barbiturate intravenous anesthetics, propofol is a respiratory depressant which causes apnea after an inductive dose.^{106,144,172} It affects central chemoreceptor sensitivity, largely by reducing the ventilatory response to hypercapnia.³⁴ Usually the ventilatory response returns to normal within approximately 30 minutes following discontinuation of a propofol infusion.¹⁴⁵

Unlike intravenous barbiturate anesthetics, propofol induces bronchodilation which can decrease the incidence of intraoperative wheezing in patients with asthma and COPD.^{106,145,173} There is also diminution of upper airway reflexes^{174,175} sufficient to allow pharyngeal suctioning of secretions. This lack of hyperactive airway reflexes may also be responsible for the markedly low tendency for laryngospasms in patients undergoing propofol anesthesia,^{174,176} especially compared to anesthesia with methohexital.⁵²

In addition to its hypnotic effects, propofol has antiemetic^{172,177–182} and euphoric^{153,183} properties. Other CNS effects include a decrease in both intracranial pressure and cerebral blood flow.¹⁴⁵ Propofol appears to have anticonvulsive properties as well.¹⁴⁵ Ironically, a few cases of long-term postoperative seizures have been reported in patients anesthetized with propofol, but the mechanism has not been elucidated.^{166,184}

Several of the various attributes discussed above would suggest that propofol would be associated with a rapid and smooth recovery. This has been substantiated by a number of clinical series.^{143,185–189}

Complications and side-effects

Although propofol possesses many highly desirable characteristics as an anesthetic agent, it is associated with untoward reactions as well. Propofol-associated hypotension¹⁴⁶ was discussed above and can largely be overcome by control of dosing and stimulation.

Another common problem is pain on injection. However, a pretreatment dose of lidocaine as low as 0.1 mg/kg is usually effective in reducing the incidence of pain on injection.⁶⁵ It has also been demonstrated that 1 ml of ketamine (10 mg/ml), 30 seconds before administration of propofol will markedly reduce the incidence of pain during propofol injection.¹⁹⁰

The propofol emulsion vehicle can lead to potential complications due to the inclusion of food products in the formulation, namely soy bean oil and egg lecithin.¹⁹¹ Allergic responses to these components have been reported, albeit rarely. Patients who are allergic to eggs are usually allergic to the albumen in the egg white as opposed to the lecithin from the yolk which is used in the propofol emulsion.¹⁰⁶ Approximately two thirds of infants with egg allergy will become tolerant to eggs by 7 years of age.^{192,193} Although soy allergy is fairly common in infants, it is usually transient. The prevalence of soy allergy is estimated at 0.3–0.4%. Reactions are usually mild, although deaths due to soy allergy have been reported. Soy oil, which is the product used in the propofol emulsion, appears to not be allergenic in soybean-sensitive individuals.¹⁹⁴

As mentioned above, the original formulation of propofol with Cremophor was abandoned due to the high incidence of allergic reactions.^{34,143,144} With the current propofol emulsion, hypersensitivity reactions have been dramatically reduced and have been estimated to be approximately 1:80 000–100 000. This compares to 1:1600 to 1:7000 for methohexital and 1:2500 to 1:10 000 for penicillin.¹⁰⁷

The use of a sulfite preservative in propofol formulations can incorporate an additional risk for allergic response.^{146,195} The incidence of true sulfite allergy in the general population is estimated at 1:1000.¹⁹⁶ Even non-sulfite-sensitive patients with asthma have a high incidence of bronchospasm following a metabisulfite challenge. In addition, 5–10% of chronic asthmatics are hypersensitive to sulfites.^{146,197} A recent study by Rieschke *et al.* demonstrated increased intubation-induced bronchoconstriction in patients with a long smoking history when sulfite-containing formulations of propofol were utilized.¹⁹⁶

As discussed above, the various allergic problems and bronchospastic reactions which attend the propofol emulsion and preservatives have led to a search for propofol modifications which would be water-soluble. These are now available and will be discussed at the end of the chapter under “Future trends and advancements – on the horizon”.¹⁴⁸

The inclusion of a perishable emulsion carries with it the potential for contamination and postinfusion infection. Periodic outbreaks of bloodstream infections have been reported and appear to be attributable to a breakdown in sterile technique. This underscores the significance of a high level of training of all members of the anesthetic team and diligence in administration of the drug.¹⁰⁷

Other side-effects and complications range from very minor to life-threatening. An interesting side-effect of no clinical importance is the occasional production of green urine following propofol infusions.¹⁹⁸ More severe reactions include the rare case of post-infusion pancreatitis¹⁹⁹ and propofol infusion syndrome.¹⁰⁷ The latter has usually been reported in high infusion rates of 5 mg/kg/h or greater for 48 hours or longer. Consequently, the likelihood of encountering such a complication in an oral and maxillofacial surgery is exceedingly remote.

The impact of propofol on clinical practice

Unquestionably the advent of propofol has been one of the pivotal events in the practice of anesthesia in hospitals, outpatient surgery centers and oral and maxillofacial surgery offices as well. In 2008, Paul F White MD, Chairman of the Department of Anesthesiology at the University of Texas Southwestern Medical Center penned an article in *Anesthesiology* entitled 'Propofol – its role in changing the practice of anesthesia'. When Dr White began his academic career at Stanford University in the early 1980s he was intrigued by the potential of this novel new anesthetic agent. Throughout his career he watched it exert a "revolutionary impact" on the practice of anesthesia.²⁰⁰ In oral and maxillofacial surgery the transition from methohexital to the propofol was somewhat slower than within the practice of anesthesiologists in hospitals and outpatient surgery centers.^{201–203} However, as was discussed in the history section above, the "Brevital crisis" in the early part of the first decade of the 21st century prompted many surgeons to convert to propofol.²⁰⁴ In 2008, Lee *et al.* reported a study on nearly 48 000 patients who underwent ambulatory anesthesia procedures in oral and maxillofacial surgery offices. The study concluded that the lowest risk for adverse events was with propofol as compared to methohexital or benzodiazepines/narcotic approaches.⁵²

Ketamine and dissociative anesthesia

In the late 1950s work began on a new class of anesthetic drugs based on phencyclidine.²⁰⁵ Phencyclidine itself was found to produce unacceptably high levels of dysphoric effects such as hallucinations and deliri-

um. However, because of several of the other desirable characteristics of this type of anesthetic, pharmaceutical research continued with exploration of other agents. In 1970, a phencyclidine congener, ketamine, was released for clinical use,²⁰⁶ and has continued to be the only dissociative anesthetic on the market. The term "dissociative" aptly describes the appearance of the patient under ketamine anesthesia. The patient appears to be in catatonic state, dissociated from the sensory inputs from the environment.³⁴ Corneal, cough, and swallow reflexes are still present and the patient's eyes are still open, but with a distant gaze.¹⁶⁴ Ketamine provides profound analgesia, even at subanesthetic doses.¹⁰⁷ It also causes amnesia, although the latter is not as great as is noted with benzodiazepines. A primary difference between ketamine and other anesthetics is that it is not associated with either hypotension or significant respiratory depression.

Chemical structure

Ketamine is a highly lipophilic arylcyclohexylamine related to phencyclidine which exists in a racemic mixture (Fig. 6b.18) consisting of an S(+) and R(–) enantiomer.²⁰⁷ Only the racemic mixture is available in the US. However, in Europe the S(+) isomer has become clinically available.^{107,208} It is twice as potent as the racemic mixture, it appears to have fewer adverse side-effects, and it is associated with more rapid recovery.

Mechanism of action

Dissociative anesthesia is due to a reduction of activation of thalamocortical structures, but with increased activity in the limbic system²⁰⁹ and hippocampus.²⁰⁵ The major excitatory synaptic receptors in the CNS are mediated by L-glutamate and are termed NMDA (N-methyl-D-aspartate). As discussed above, the receptor ion channel (see Fig. 6b.16) is permeable to calcium as well as sodium and potassium to a lesser extent. The channel requires glycine as an obligatory co-agonist and is inhibited by magnesium (Mg^{2+}) in a voltage-dependent manner. Ketamine binds to the phencyclidine receptor in the NMDA channel and inhibits glutamate activation in a non-competitive fashion. At resting membrane potential the NMDA receptor is inactive because of the

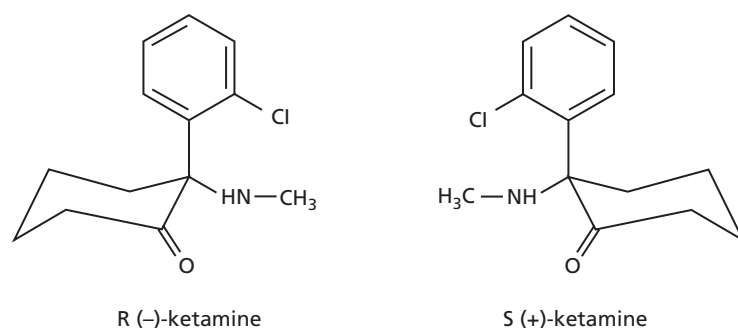


Fig. 6b.18 Structural formulas of the two enantiomers of ketamine. (Reproduced from Kohrs R, Durieux ME. Ketamine: teaching an old drug new tricks. *Anesth Analg* 1998; 87: 1186–93 by permission. Copyright © 1998 Lippincott Williams & Wilkins.)

voltage-dependent block of the channel pore by magnesium ions. Then, as the cell membrane becomes depolarized, the negative field weakens and magnesium ions are released. Consequently, the NMDA channel is both a ligand-gated, as well as voltage-gated, ion channel. By binding at the PCP site, ketamine blocks the open channel and reduces its mean open time.²⁰⁵

Ketamine has effects at several other receptors as well. The analgesic effects of ketamine appear to be due in part to its action on μ and κ opioid receptor sites. It is likely that the psychotomimetic effects are due to action at the κ rather than the μ receptor.²⁰⁵ Muscarinic acetylcholine receptors are inhibited by ketamine which may explain such effects as bronchodilation. This may also explain the increase in bronchial and mucus secretions, and perhaps ketamine's amnesic effect. Interestingly, ketamine tends to affect sodium channels as well, in a manner similar to that of local anesthetics.¹³⁴

The L-type calcium channel is susceptible to the effects of ketamine. Ketamine blocks the calcium channel in a manner somewhat similar to calcium channel blockers such as verapamil. In part this action may help explain the vasodilation, bronchodilation, and negative inotropic effects of ketamine.²⁰⁵

Distribution

Ketamine is highly lipid-soluble, and its effects are noted within seconds of intravenous administration and reach a peak at 1 minute. Its distribution half-life is approximately 10–15 minutes and patients become fully oriented to person, place, and time within approximately 15–30 minutes.³⁴ However, the effects of ketamine may be somewhat prolonged by the co-administration of benzodiazepines, which are usually administered to attenuate the psychotomimetic actions of ketamine.⁶⁴

Biotransformation and excretion

Ketamine undergoes biotransformation in the liver, primarily by N-demethylation.²⁰⁷ The N-demethylated metabolite is active, but has only one sixth of the potency of ketamine.²⁰⁵ Ketamine and its metabolites are hydroxylated and conjugated with water-soluble conjugates that are excreted in the urine. The elimination half-life is approximately 2.5–3 hours.³⁴ Only a small percentage (approximately 4%) of unchanged ketamine is excreted.²⁰⁵ Decreased renal function does not prolong the drug's action.²⁰⁹ It has been found that the elimination in children is approximately twice as rapid as that in adults.

Effects on organ systems

Unlike most other anesthetic agents, ketamine tends to stimulate the cardiovascular system and it is usually associated with increases in blood pressure, heart rate, and cardiac output.²¹⁰ These effects are due to central stimulation of the sympathetic nervous system and inhibition of the reuptake of norepineph-

rine.¹⁰⁶ They are noted with both the S(+) enantiomer and the racemic mixture. The hemodynamic changes are not related to the dose of ketamine, and a follow-up dose of ketamine may actually even diminish the effects of the initial dose.²¹¹ Ketamine appears to inhibit intraneuronal uptake of catecholamines, much like cocaine.³⁴ Obviously, cardiovascular system stimulation is not always desirable, especially in patients with tachycardia or systemic hypertension. The effects can usually be largely overcome with other agents, especially the benzodiazepines. Even in modest doses diazepam and midazolam tend to attenuate the hemodynamic effects of low-dose ketamine.^{209,212}

As compared to most other anesthetic agents, ketamine exhibits an unaltered response to carbon dioxide and has minimal affect on central respiratory drive. Only at very high induction doses is there a transient decrease in minute volume or apnea. Ketamine is a bronchial smooth muscle relaxant, probably as a result of sympathomimetic effects.^{207,213} This activity makes the agent particularly useful in asthmatic patients and it has even been used to treat status asthmaticus in cases which were unresponsive to conventional treatments.^{34,214,219} Ketamine reduces bronchoconstriction via antagonistic effects of the vagus nerve and attenuates opioid-induced hyperventilation.²¹⁵ On the negative side, ketamine tends to increase salivation which can be a contributing factor in the development of laryngospasms.³⁴ However, the hypersalivation affect can be attenuated with anticholinergics such as glycopyrrolate or atropine.^{64,106,164}

Ketamine crosses the blood-brain barrier rapidly with an onset of action within 30 seconds and maximal effect within 1 minute. It is rapidly redistributed from the brain and blood to other tissue. When administered alone, it can produce emergence reactions such as vivid dreaming, extracorporeal expressions, and illusions.²⁰⁶ However, when ketamine is given at low doses, these psychotomimetic effects can be largely overridden by benzodiazepines,^{134,209} especially midazolam 0.025–0.050 mg/kg.¹⁶⁴ Emergence reactions are rarely noted in pediatric patients and are less commonly seen in males as compared to females. In addition, low doses at slower rates tend to minimize them³⁴ and they are often not remembered by the patient.²¹⁶

Ketamine increases cerebral oxygen consumption, cerebral blood flow and intracranial pressure. Interestingly, under controlled ventilation ketamine does not increase intracranial pressure. Yet another CNS effect of ketamine is its neuroprotective action¹⁰⁷ which has been demonstrated in numerous *in vitro* studies. However, this will need further clinical studies to assess whether there are any true benefits of ketamine in the treatment of patients with neuronal damage.

Complications and side-effects

Overall, ketamine is considered to be a relatively safe anesthetic with a wide therapeutic range. Even in

cases in which patients have been given ten times the normal dose of ketamine, there has been uneventful recovery. As discussed above, some of its less desirable effects such as hypersalivation and cardiovascular stimulation can be easily minimized with antisialogogues¹⁰⁷ and benzodiazepines respectively. In addition, when ketamine is used at relatively low doses, its psychotomimetic tendencies can also be attenuated with benzodiazepines, especially midazolam.¹⁰⁶

When there are significant advanced comorbidities, some of the above problems can constitute contraindications. These include severe cardiovascular disease such as angina,²⁰⁹ severe hypertension with multiple antihypertensive agents, glaucoma which is poorly controlled,¹³⁴ severe psychotic illnesses with suboptimal control with psychiatric medications³⁴ and pregnancy, especially in the first trimester.

Effect on clinical practice

In 1998 an article entitled ‘Ketamine: teaching an old drug new tricks’ appeared in *Anesthesia and Analgesia*, the journal of the International Anesthesia Research Society.¹³⁴ It followed the course of ketamine from the initial enthusiasm for the drug, its fall into disfavor, and finally the resurgence of interest which occurred at the close of the 20th century. The drug’s beneficial effects far outweigh its detrimental effects, and the latter can be largely overcome when ketamine is used in combination with other agents as part of a balanced technique. During the latter years of the 20th century and the first decade of the 21st century, numerous reports began to appear demonstrating the successful use of low-dose ketamine as a part of a balanced anesthetic, especially in combination with propofol. Consequently, it does appear that, indeed, it is possible to “teach an old drug new tricks”.

The complementary effects of propofol and ketamine

As is apparent from the description of the primary agents propofol and ketamine discussed above, these agents tend to have many complementary effects. Some of the principle characteristics are compared and contrasted in Table 6b.2.

The complementary effects of the two agents have not gone unnoticed. These complementary effects can be capitalized upon in the development of a balanced anesthetic approach. Anesthesiologists and oral and maxillofacial surgeons throughout the US and abroad have used the agents in various combinations in a number of different clinical settings. These include: pediatric oncology,²¹⁷ pediatric burn dressing changes,⁴³ anorectal surgery,⁴² cosmetic surgery,^{41,218,219} emergency department,^{220,221} traumatic brain surgery,²²² as well as oral and maxillofacial surgery.⁴⁰ Although the vast majority of these reports suggest a definite benefit to the combination of propofol and ketamine, some have not.^{223–225} As yet there are no

Table 6b.2 Complementary effects of propofol and ketamine.

Complementary effect	Propofol	Ketamine
Analgesia	Pain on injection	↑↑↑↑
Relaxation	↑↑	↑↑
Amnesia	↑↑	↑↑
Heart rate	0	↑↑
Blood pressure	←←←	↑↑
Ventilation	←←←	0/←
Bronchodilation	0/↑	↑↑↑↑
Cerebral blood flow	←←←	↑↑↑↑
Intracranial pressure	←←←	↑↑↑↑
Antiemetic	↑↑	←
Pharyngeal reflexes	←	0/↑
Emergence euphoria	↑↑	←←

large studies under controlled conditions which provide a definitive comparison between propofol anesthesia and anesthesia with the combination of propofol with ketamine. Albeit truly definitive studies are not yet available, the author’s experience with well over 8000 oral and maxillofacial surgery cases has been decidedly positive, as will be discussed below.

The studies which have found the combination to be beneficial allude to the diminution of marked fluctuations in cardiovascular and respiratory parameters, better intraoperative and postoperative analgesia and rapid recovery with little in the way of dysphoria. Other benefits include diminished pain on propofol injection,²²⁶ earlier recovery of cognition,²²⁷ and improvement of postoperative depressive state in depressed patients.²²⁸ Virtually all of the studies point to a lessened requirement of postoperative opioid analgesics.^{229,230}

Ancillary agents

Historically it was always hoped that pharmacologic research would unveil a monoanesthetic agent that would have all of the characteristics desirable in an anesthetic agent.^{64,206} The agent could literally be “turned on” and “turned off” and would exhibit virtually no untoward effects. Then, when an agent failed to meet expectations (as in the case of ketamine), interest would wane and the search would continue for yet another “perfect agent”. Ultimately the agent which has come closest to expectations has been propofol. However, it too has shortcomings and the use of other agents along with propofol is necessary to obtain the multiple characteristics which are today considered essential in an anesthetic. Out of the wide range of agents that have been used over the years, this chapter will now discuss some of those which currently enjoy the widest popularity in hospital operating rooms, outpatient surgery centers, and oral and maxillofacial offices.

Anxiolytics

Preoperative anxiety has been shown to increase the demand for principal anesthetic agents²³¹ and can be responsible for patient dissatisfaction with anesthesia.²³² Consequently, it behoves the anesthetist to deal directly with patient anxiety rather than attempt to overcome it with large doses of primary anesthetic agents. The agents which have been found to be of greatest benefit in dealing with patient anxiety include the benzodiazepines and nitrous oxide.

Midazolam

Diazepam, introduced in the 1960s, initially enjoyed wide popularity as a component of methohexital-based anesthesia in oral and maxillofacial surgery.³⁷ However, its protracted biotransformation and elimination and vein irritation prompted a search for a water-soluble agent with shorter action.^{233,234} This investigation led to the development of midazolam, which began to overtake diazepam as the preferred anxiolytic by the turn of the 21st century. Midazolam is water-soluble and rarely leads to thrombophlebitis.²³⁴ It provides greater sedation and more profound anterograde amnesia, yet is shorter-acting than diazepam.²³⁵ Like other benzodiazepines, it possesses anticonvulsant and muscle relaxant properties. It has relatively little cardiovascular effect except for diminution of sympathetically induced hypertension.²³⁵ In large, rapidly introduced boluses it can induce respiratory depression, and the patient's respiratory status must be diligently monitored. When the agent was initially introduced, its markedly greater potency^{34,236} than diazepam (especially in conjunction with fentanyl) went unrecognized by some practitioners, and respiratory depression,^{149,237} and even deaths, were recorded.²³⁸ However, it became apparent that the problem was inadequate respiratory monitoring rather than a fault of the drug itself. Today midazolam is the most widely used benzodiazepine in anesthesia practice.³⁴ It is used intravenously as a preinduction agent, as a component of moderate ("conscious") sedation and as oral premedication in pediatric patients.²³⁴

Other benzodiazepines

Several different benzodiazepines have been used as oral premedication in oral and maxillofacial surgery. These include diazepam, lorazepam, and triazolam. However, trials comparing different benzodiazepines have failed to demonstrate consistent differences in efficacy among the different agents.²³⁹ Of the various agents available, the one which appears to have the most desirable pharmacokinetic and pharmacodynamic properties is triazolam.^{240,241} It reaches peak blood level at approximately 1 hour and has an elimination half-life of only 2–3 hours. This is in large part due to the absence of active metabolites. Triazolam is usually used in a dose of 0.25–0.5 mg for adult patients, except the elderly. The latter are quite sensi-

tive to benzodiazepines and a dose of 0.125 mg or less is appropriate for patients of advanced age.²⁴² Although its pharmacokinetic and pharmacodynamic profile makes triazolam an excellent agent for anesthesia premedication, in the late 1980s and early 1990s the drug's acceptance suffered from a number of sensational media reports. The latter suggested that triazolam might be associated with extreme excitation and even violence.²⁴³ Subsequent scientific investigation disclosed that triazolam shared the same exceedingly safe drug profile of the other benzodiazepines and that the immediate concern was unjustified.^{244,245} Today the medication is used widely for both anesthesia premedication and oral sedation in dentistry.²⁴⁶

Although, as stated above, benzodiazepines as a group are relatively safe medications, respiratory depression can be significant, and even life threatening. Consequently, efforts were directed at developing a specific benzodiazepine antagonist. These efforts resulted in the development of flumazenil in 1979.³⁴ However, it did not become available clinically in the US until the early 1990s.²⁴⁷ As a typical competitive antagonist, it has minimal intrinsic effects of its own and replaces benzodiazepine agonists at the receptor site. However, its short duration of action (plasma half-life of approximately 1 hour) can create a false sense of security. The reversal may be far more short-lived than the respiratory depression associated with the original agonist. Consequently, the patient should be observed following administration of flumazenil to assure that re sedation does not take place.²⁴⁸ The initial reversal dose is 0.1–0.2 mg. Repeat doses can be given up to a total dose of 3 mg.³⁴

Nitrous oxide

Nitrous oxide has been the "workhorse" inhalation agent in anesthesia for over 150 years.²⁴⁹ It tends to relieve anxiety, reduces the incidence of intraoperative awareness,²⁵⁰ and provides a synergistic effect with sedative and hypnotic agents, thereby reducing the dose of the primary anesthetic agent required. It also has analgesic effects which appear to be related to activation of opioid receptors²⁵¹ in the periaqueductal gray matter and noradrenergic neurons in the locus ceruleus.²⁵² Nonetheless, within recent years there have been some concerns regarding possible nitrous oxide toxicity.²⁵³ It appears to affect methionine synthase function which could potentially lead to genetic and protein aberrations.²⁵¹ An extensive review of potential adverse effects of nitrous oxide was presented in an article entitled 'Biologic effects of nitrous oxide' in *Anesthesiology* in 2008.²⁵⁴ It concluded that it would be prudent to avoid nitrous oxide in certain vulnerable populations, but abandonment of the agent is not indicated based on current scientific studies. Similar conclusions were drawn by Ian Smith in his review in 2006²⁵⁵ and by Philip Hopkins in another review in 2005.²⁵⁰ Absolute contraindications²⁵⁴ would include patients of known deficiency

of the enzyme or substrate in the methionine synthase pathway, patients with raised intracranial pressure, and patients with gas-filled space such as those with emphysema, pneumothorax or those undergoing middle ear surgery. Relative contraindications would include patients with pulmonary hypertension, patients in their first trimester of pregnancy, and patients who are at a high risk of postoperative nausea and vomiting.

In addition to the toxic effects of nitrous oxide which can be experienced by patients, there is also a risk to office personnel. To minimize these risks, scavenging systems have been in place in dental offices for over half a century. Scavenging is intended to limit the concentration of nitrous oxide in the operator to meet standards established by governmental agencies. The safe limit established by the National Institute for Occupational Safety and Health (NIOSH) is 50 ppm (parts per million) while that for the American Conference of Governmental Industrial Hygienists (ACGIH) is 25 ppm.²⁵⁶ A study reported in the *Journal of American Dental Association* in February 2009²⁵⁷ compares the levels achievable for a conventional scavenging mask and a newly designed mask with enhanced features (the Safe Sedate dental mask, Airgas, Rednor, PA), which is shown in Fig. 6b.19. The new mask delivers the anesthetic gas directly into each naris via an adjustable cannula which passes through the mask. The wasted anesthetic gas is scavenged from the chamber which houses the mask via a single hose that is attached to the vacuum system. The balloon of the mask is designed to provide excellent adaptation, and the mask is held in place with straps which go around the patient's head.

The comparison used infrared thermography, digital videography, and real time nitrous oxide air concentration levels with infrared spectrophotometry. It was found that the advanced features of the new mask provided better reduction of nitrous oxide, especially in conjunction with key work practices which included proper mask sizing and fitting, activating the exhaust before administration of nitrous oxide, and discouraging the patient from talking and mouth breathing. Statistically significant results were obtained when these practices accompanied use of the new mask. Fig. 6b.20 demonstrates the difference in mask leakage disclosed by infrared thermography.

Another key issue in limiting staff exposure to nitrous oxide is design of the office ventilation system. Since nitrous oxide is heavier than nitrogen and the other components of air, there is stratification with nitrous oxide settling in the lower part of the room. Many offices have exhaust vents in the ceiling which tend to exhaust predominantly air in the top of the room, leaving much of the nitrous oxide around the operative team. Evacuation of the residual nitrous oxide which has been inadequately scavenged requires placement of exhaust vents on the floor or



Fig. 6b.19 The Safe Sedate dental mask by Airgas. The nitrous oxide is delivered directly into the nares and evacuated from the mask chamber into the vacuum exhaust. Note the close adaptation of the mask to the face and the securing straps to assure a satisfactory seal. (Photos courtesy of John Moenning, DDS, Fishers, IN.)

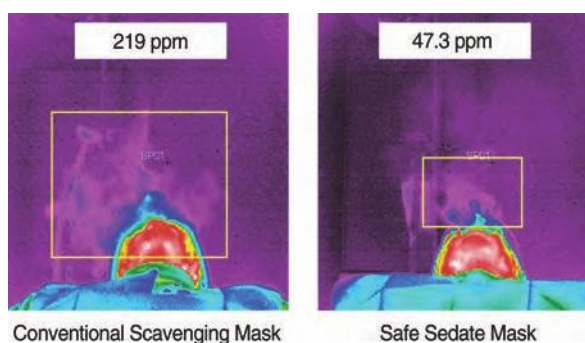


Fig. 6b.20 Infrared thermography demonstrating the difference in leakage of nitrous oxide by a conventional scavenging mask on the left and the Safe Sedate dental mask on the right. (Photos courtesy of John Moenning, DDS, Fishers, IN.)

lower portion of room walls rather than the ceiling.²⁵⁸

Opioid analgesics

Opioid analgesics were an essential component of most balanced anesthetic approaches for the better part of the last century. In modern practice the older agents such as morphine sulfate and meperidine have been replaced by the newer agents, fentanyl and remifentanyl of the anilidopiperidine series. Although

fentanyl is currently the most frequently used of these drugs,¹⁰⁴ remifentanyl is gaining popularity.

Fentanyl

Fentanyl is the original anilidopiperadine opioid and has gained wide popularity in anesthesia. It is a μ receptor agonist which is 100 times as potent as morphine and has a rapid onset of less than 1 minute.³⁶ Its short duration of 30–60 minutes²⁵⁹ makes it an ideal narcotic for outpatient surgery in general and oral and maxillofacial surgical anesthetics in particular.²⁶⁰ There is less histamine release than with meperidine, which makes the agent more appropriate for asthmatic patients than its predecessor.²⁶¹ There appears to be a similar incidence of nausea and vomiting as was noted with most of the other opioid agents used previously.³⁶ The cardiovascular system remains relatively stable, but the patient may develop modest bradycardia.¹⁰² Chest wall rigidity has been reported, but appears to be related to relatively large doses¹⁰⁶ and has been infrequently encountered in oral and maxillofacial surgery practice.

Remifentanyl

The newest drug in the anilidopiperadine family is remifentanyl,²⁶² a μ agonist introduced in 1996,^{221,263} which differs from the previous opioid agents in that it is metabolized by esterases throughout the body (Fig. 6b.21).²⁶⁴ Consequently, there is very rapid dissipation of the clinical effects of the drug once an infusion has been discontinued. It also has a pK_a that is less than physiologic pH, which allows circulation of the drug in its non-ionized moiety. This in turn enables rapid penetration of the blood–brain barrier.^{102,265} Although it has an analgesic potency similar to that of fentanyl,²⁶⁶ the pharmacokinetic properties of remifentanyl lead to an onset of action of 1 minute^{36,267–269} and duration of action of only 5–10 minutes following discontinuation of the infusion.²⁵⁹ Because its metabolism is based on non-specific esterases, remifentanyl's pharmacokinetics profile is independent of end-organ failure.^{261,270} Since the elderly have a smaller volume of distribution and slower clearance,²⁶⁵ patients over the age of 65 should have

the dosage of remifentanyl decreased by at least 50%.²⁶⁸ Dosing of obese patients should be based on lean body mass rather than total body weight.^{262,271,272} Studies in the use of remifentanyl in oral and maxillofacial surgery point to rapid recovery, decreased propofol requirements,⁴⁵ and more stable cardiovascular parameters.⁴⁴ Remifentanyl can be administered as a separate infusion, with incremental boluses or as a combined infusion with propofol.

Anticholinergics

Anticholinergic agents are frequently indicated for the control of salivary and bronchial secretions.²⁷³ In particular, they can be of benefit in the management of the hypersalivation which accompanies administration of ketamine. Pharmacologic control of secretions helps to prevent laryngospasms, and anticholinergic medications have been the primary agents used. The older belladonna alkaloids, scopolamine and atropine, are tertiary amines and cross the blood–brain barrier where they can have untoward psychological affects.²⁷⁴ In addition, the tachycardia produced by the vagolytic effect of these agents may be undesirable in older, medically compromised patients. Consequently, atropine and scopolamine have been largely replaced in anesthesia by a potent quaternary anticholinergic, glycopyrrolate.²⁷⁵ Although the latter may cause mild tachycardia, it is far less prominent and of shorter duration. As a quaternary ammonium agent, it does not cross the blood–brain barrier and carries with it no untoward mood affects or psychotomimetic reactions.²⁷⁶ As an antisialogogue it is far more potent and the effect may last two to five times longer (up to 8 hours) than that of atropine.²⁷⁷

Antiemetics

In surveys of patient acceptance, lack of postoperative nausea and vomiting is considered a highly desirable trait of anesthetics.^{66,278,279} Postoperative nausea and vomiting (PONV) is the most frequent side-effect after anesthesia. Consequently, over the years many antiemetic medications have been used to help control or eliminate emesis and nausea following anesthesia. Historically the more effective agents tended to be dopamine (D_2) receptor antagonists such as the phenothiazines and metoclopramide. However, in the 1990s a new category of exceedingly effective antiemetic medications was developed, the 5-HT₃ receptor antagonists.^{56,280} The prototypic drug in this class is ondansetron which now enjoys wide use in the treatment of chemotherapy-induced emesis as well as anesthesia-associated nausea and emesis.²⁸¹ Combination therapy is more effective than monotherapy.^{280,282–286} An especially effective combination is a 5-HT₃ receptor antagonist accompanied by a potent corticosteroid such as dexamethasone.^{279,281,286–288}

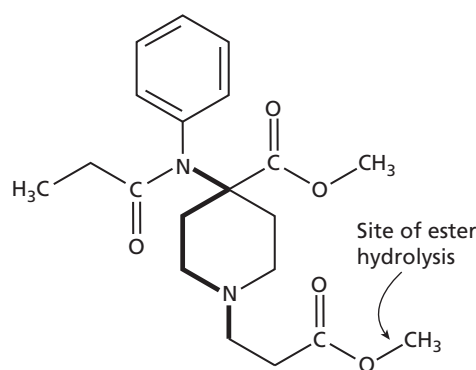


Fig. 6b.21 The structure of a remifentanyl. Note the site of ester hydrolysis. (Reproduced from *Clinical Anesthesiology* (Morgan GE, Mikhail M, Murray M, eds.), 4th edn. New York: McGraw-Hill, 2006. With permission of the McGraw-Hill Companies.)

Inhalation agents – sevoflurane

Historically, oral and maxillofacial surgeons have tended to prefer the total intravenous anesthetic approach as opposed to inhalation anesthesia. However, with the introduction of sevoflurane in the 1990s,²⁸⁹ an agent became available which possessed far more desirable characteristics than its predecessors.⁶³ Sevoflurane has a number of appealing attributes which have encouraged greater use of inhalation anesthesia in some oral and maxillofacial surgery practices.³⁷ Sevoflurane anesthesia is characterized by rapid onset and emergence,^{290,291} a pleasant smell,²⁹² lack of airway irritation,²⁸⁹ and lack of myocardial irritability.^{63,291} Nonetheless, like all inhalation anesthetics, it has the significant liability of being a potential triggering agent for malignant hyperthermia.²⁹⁰ Consequently, offices using the agent must be prepared with dantrolene and training in the treatment of malignant hyperthermia.⁴⁹

Balanced anesthetic approaches currently used in oral and maxillofacial surgery practices

Sedation and anesthesia in oral and maxillofacial surgery today cover a wide range of drugs and techniques. They can produce a depth of sedation which ranges from minimal sedation to general anesthesia. In recognition of this continuum, the ASA published its *Practice Guidelines for Sedation and Analgesia by Non-anesthesiologists* in 2002.^{293,294} In that article it was stated, "Because it is not always possible to predict how a specific patient will respond to sedative and analgesic medications, practitioners intending to produce a given level of sedation should be able to rescue patients whose level of sedation becomes deeper than initially intended." It is important that the oral and maxillofacial surgeon remains constantly aware of this continuum in choos-

ing and monitoring the anesthetic chosen for a given patient. In a healthy teenager or young adult, a patient may pass through each the four levels of the continuum during his time in the office. The four levels are as follows:

1. Minimal sedation (anxiolysis) – there is normal response to verbal stimulation with the airway, ventilation, and cardiovascular function unaffected.
2. Moderate sedation ("conscious sedation") – purposeful response to verbal or tactile stimulation with no compromise of the airway and ventilation and cardiovascular function usually maintained.
3. Deep sedation/analgesia – purposeful response after repeated or painful stimulation with possible need for maintenance of airway and ventilation and cardiovascular function usually maintained.
4. General anesthesia – patient unarousable, even with painful stimulation, airway intervention required with possible support of ventilation and possible impairment of cardiovascular function.

Fig. 6b.22 graphically illustrates the levels of sedation through which a typical patient passes during routine dentoalveolar surgery such as third molar removal. The agents which provide the appropriate level of sedation for the various stages of treatment are as follows:

1. Preoperative anxiety – oral premedication with an anxiolytic can induce a state of minimal sedation and anxiolysis which reduces the demand for the primary anesthetic agent.
2. Nitrous oxide can be administered prior to the venipuncture to reduce anxiety regarding the needle stick as well as preoxygenate the patient.
3. Preinduction agents including a benzodiazepine and narcotic establish a state of moderate sedation

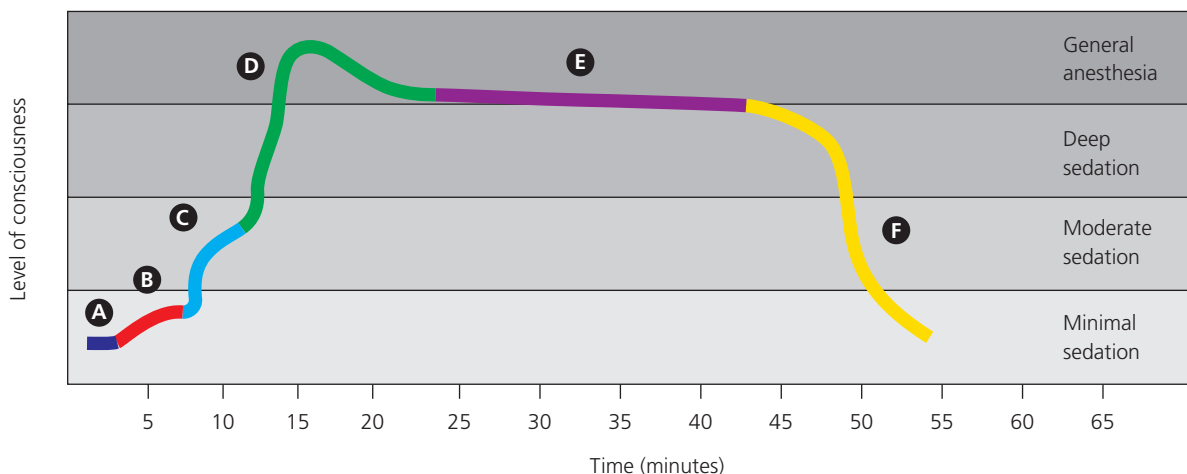


Fig. 6b.22 The levels of sedation during intravenous anesthetic. Appropriate agents transport the patient through several levels of sedation to a plane of surgical anesthesia during which the procedure is performed. These include: A. Reduction of preoperative anxiety with oral premedication. B. Anxiolysis and preoxygenation with N_2O/O_2 . C. Preinduction medication with preinduction agents such as a benzodiazepine and narcotic. D. Induction with propofol (and possibly ketamine). E. Maintenance with a propofol infusion. F. Discontinuation of infusion with emergence.

which provides amnesia and analgesia and “balancing” of the anesthetic.

4. Induction with a potent hypnotic such as propofol and possible dissociative anesthesia with ketamine. Just after induction while the patient is in a state of deep sedation/general anesthesia, local anesthesia is administered, which subsequently reduces the anesthetic requirement.
5. The patient is maintained on a continuous infusion of propofol to maintain a level of consciousness bordering deep sedation and general anesthesia.

Method of delivery

Historically the primary barbiturate anesthetics thiopental and methohexital were delivered in small incremental boluses (the “bump” technique). As most oral and maxillofacial surgeons made the transition from methohexital to propofol, they tended to continue with the same method¹⁴⁰ but used smaller boluses at more frequent intervals.²⁹⁵ Some anesthesiologists have found the technique to be satisfactory, but many others have desired to capitalize on the “smoothness” of a constant infusion via infusion pump.^{163,296,297} Fortunately, simple, syringe-based infusion pumps have been developed which enable ease of administration of the anesthetic and elimination of the need to constantly administer boluses.²⁹⁸ Surgeons who have made the transition to an infusion pump find that there is another advantage beyond the greater smoothness of the anesthetic. There is less likelihood of patient awareness during the procedure when there is no interruption of the infusion, as often takes place during manual bolus administration.²⁹⁹ A typical pump which has been a “workhorse” delivery system in many operating rooms and outpatient surgery centers is pictured in Fig. 6b.23. Newer, totally digital pumps are appearing in the marketplace. However, a number of anesthesiologists have found them to have “too many bells and whistles” and, in some cases, to be less reliable.³⁰⁰ Consequently, many prefer to continue to use the partial analog device described here. Details on the use of the pump will be provided below in the discussion of the author’s technique for total intravenous anesthesia.

Total intravenous anesthesia (TIVA)

In the US most oral and maxillofacial surgeons currently use a total intravenous anesthetic approach based on propofol, and in some cases, propofol with ketamine. The choice of ancillary agents and dosages is highly variable from surgeon to surgeon.¹⁴⁰ Attempting to address this wide range of variables is beyond the scope of this chapter; the author’s technique will be described as an example. The author co-directs the outpatient anesthesia curriculum within the Oral and Maxillofacial Surgery Department at the University of California at San Francisco. The technique has been used for over 8000 patients in the

author’s private practice, and it has been taught to the oral and maxillofacial surgery residents who now also use it in the various outpatient surgery clinics in which they perform dentoalveolar and implant surgery.

The technique is based on a combination approach with propofol and ketamine, with the propofol administered via an infusion pump. The technique was developed with contributions from the literature as well as from anesthesiology colleagues of the author. As such, it illustrates several important points that have been made in the anesthesia literature over the last several years:

1. The components of balanced intravenous anesthesia do not always act in a synergistic fashion. For example, the addition of ketamine to propofol anesthesia adds dissociation but little in the way of enhancement of hypnosis.^{301,302}
2. Drug interactions with intravenous anesthetics are not always detrimental. For instance, the hypotensive effects of propofol are frequently largely overcome by the hypertensive affects of ketamine when it is added to the mix.³⁰³
3. The “short-acting” agents from merely a few years ago are now “long-acting” in comparison to intravenous propofol and ketamine and remifentanyl. For instance, midazolam and fentanyl were shorter acting than their predecessor benzodiazepines and narcotics. However, they are considerably longer acting than propofol and ketamine. Consequently, the smallest effective dose possible of these agents should be used to promote more rapid recovery. In most cases, 2mg of midazolam and 50µg of fentanyl provide the desired characteristics to the anesthetic without significantly prolonging recovery.
4. A propofol-based anesthetic has attributes which distinguish it from virtually all other forms of intravenous or inhalation agents. For instance, the inherent antiemetic effects and postoperative feeling of well-being which accompany propofol anesthesia lead to a high level of patient acceptance.^{144,200}



Fig. 6b.23 The Baxter Infus O.R. partial-analog anesthesia infusion pump.

5. Propofol can be used in either its sedative or anesthetic mode, and is hence very flexible for use in different patient populations. Often sedation is the more appropriate anesthetic approach for elderly and/or medically compromised patients. Sedation is also a more appropriate means of managing long implant cases. In both these settings, propofol can be used very effectively for sedation as opposed to deep sedation or surgical anesthesia.³⁴

The author's office-based anesthesia technique

The author's technique embraces the various principles outlined above and is described below.

Oral and anesthetic premedication

Sedation and anxiolysis is provided by triazolam 0.25–0.5mg PO. Dosages are reduced commensurately for elderly patients and given on a weight basis to pediatric patients. For children who have difficulty swallowing tablets, the medications are crushed into a powder and given with a concentrated, pulpless fruit punch. Since ketamine is a component of most of the intravenous anesthetics, glycopyrrolate is given as an antisialagogue with a usual dose of 2mg PO with scaled doses for children. For middle-aged and older males with potential benign prostatic hypertrophy, no glycopyrrolate is dispensed. To counteract gastric hyperacidity the H₂ antagonist ranitidine is given in two doses. The first dose of 150mg is given to be taken at bedtime on the night before the procedure and the second with the other anesthetic premedications 1 hour prior to the procedure.

Preparation for venipuncture and the preinduction agents

Prior to establishment of intravenous access, nitrous oxide and oxygen 40–60% are administered via face-mask. For adults, the venipuncture site is prepared with a spray of ethyl chloride. For children, EMLA cream is placed 1–1½ hours prior to venipuncture. These agents are discussed in greater detail in the perioperative management section below.

The intravenous route is established with a catheter-over-needle device and a continuous infusion of normal saline administered throughout the procedure. The first agent introduced is midazolam 0.02–0.03mg/kg of lean body mass. This provides satisfactory amnesia, relaxation, and anxiolysis without prolonging recovery. The preinduction narcotic consists of either fentanyl or remifentanyl. In the case of fentanyl, the usual initial dose for a 70kg patient is 50µg and it is rarely repeated. When remifentanyl is used, it is added to the propofol infusion (50µg per 20ml propofol) as discussed below. If it appears that there are still excessive secretions in the face of the oral glycopyrrolate, an additional 0.2–0.25mg of glycopyrrolate is administered.

Induction and maintenance

The dosages for induction and maintenance of propofol and ketamine are based on distribution groups as outlined in Box 6b.1. Note that the induction dose of ketamine is half of the induction dose of propofol as determined by the table. For most cases the low-dose ketamine bolus given at the beginning of the procedure is adequate for completion of the surgery. In patients who are resistant to medication, incremental ketamine doses of 5–10mg are given every 10–15 minutes during the procedure. In children the incremental doses are given routinely, usually in increments of 2.5–5mg. For patients over the age of 60, no ketamine is administered.

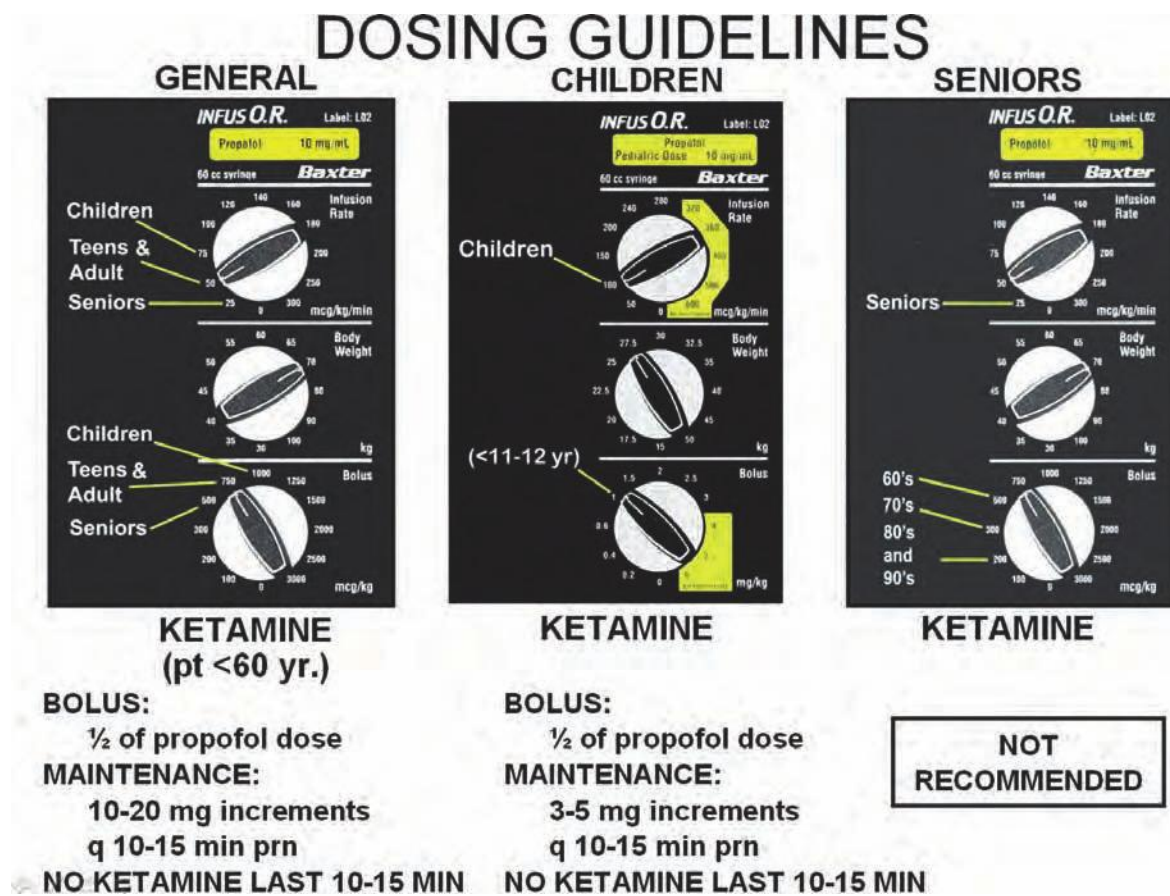
In most cases remifentanyl is used in conjunction with propofol. It is reconstituted and diluted 1:20, 1ml of a 50µg/ml remifentanyl solution is added to each 20ml multiple-dose vial of propofol. In keeping with other series reported in the oral and maxillofacial surgery literature, the author has found that use of remifentanyl as a component of balanced anesthesia has shortened recovery and allowed less fluctuation in cardiovascular parameters.^{44,45}

Intravenous sedation

For certain patient groups intravenous sedation is preferable to total intravenous anesthesia. Examples would include elderly and/or medically compromised individuals and those undergoing implant surgery. Traditionally cases such as these have been managed with a combination of a benzodiazepine (usually midazolam) and a narcotic (usually fentanyl). However, a number of anesthesiologists, as well as the author, have found that use of propofol in its sedation mode will often provide a more desirable anesthetic course. In this setting a small dose of midazolam and fentanyl is given at the beginning of the procedure and followed with a low-dose propofol infusion. The latter provides excellent sedation for the procedure, but more rapid recovery than that which is usually seen with multiple incremental doses of benzodiazepine and narcotic.

Local anesthesia

A consistent cornerstone in any balanced anesthetic is satisfactory analgesia. In oral and maxillofacial surgery a large component of an anesthetic's analgesia is provided by local anesthesia, as opposed to heavy doses of intravenous agents.^{304–306} Thus it behoves the operator-anesthetist to provide the best possible local anesthesia to obviate the need for large doses of narcotic and/or ketamine to obtain adequate relief from pain. A thorough knowledge of the anatomy of local anesthesia and local anesthetic technique are essential in the delivery of satisfactory outpatient anesthesia. In addition, judicious use of the agents themselves can provide a more comfortable anesthetic course for the patient.



Box 6b.1 The author's dosing guidelines for administration of propofol, ketamine, and remifentanyl for office-based anesthesia. The propofol and remifentanyl are delivered via the Baxter Infus O.R. Anesthesia Infusion Pump. A separate syringe is used to administer the induction bolus of ketamine. Subsequent incremental boluses of ketamine are used routinely for pediatric patients, but only on a prn basis for teens and adults. At the top of each column is a graphic depiction of the pump settings used for the administration of propofol for each age group of patients. Beneath the graphic depiction of the pump are the guidelines for the dosing of ketamine which is used in conjunction with propofol. When remifentanyl is administered in combination with propofol, 1 ml of a 50 µg/ml solution is added to each 20 ml vial of propofol.

Anatomic considerations

The greatest incidence of failure in providing adequate local anesthesia is in what is traditionally considered the distribution of the mandibular division of the trigeminal nerve (V_3).³⁰⁷ This may be due to poor technique, lack of appreciation of cross-innervation, and anatomic variability. Both the traditional "cross arch" mandibular block procedure technique with a straight needle^{308,309} or the Gow-Gates³¹⁰ approach are technique-sensitive.

Another approach used by the author, as well as a number of other surgeons, is based on an appreciation of the anatomy of the medial aspect of the mandibular ramus obtained through experience performing the sagittal ramal split osteotomy. Surgeons experienced in the technique are aware of the high degree of variability in the prominence of the medial buttress of the coronoid process which begins at the base of the process and descends down the medial aspect of the anterior portion of the mandibular ramus. The prominence of the buttress and marked flaring of the ramus will at times make visualization of the neurovascular bundle rather difficult as it enters the mandibular canal inferior to the lingula.

This same buttress often presents an impediment to the delivery of local anesthetic to the appropriate location using the traditional straight-needle approach. The author's technique calls for incorporating a gentle curvature in the needle (Fig. 6b.24) to enable it to pass over and posterior to the buttress directly to the mandibular groove (Fig. 6b.25). Traditionally students have been admonished to "never bend a needle" because of needle fractures that have occurred when sharp, angular bends have been incorporated into local anesthetic needles, especially at the hub.³¹¹ However, the gentle curvature as described and illustrated in Fig. 6b.24 does not substantially weaken the needle and allows deposition of the local anesthetic in the appropriate location. As a matter of fact, with a slight curvature, the needle can be used as a "sound" to verify that the tip is indeed in the mandibular groove.

A frequent cause of inadequate local anesthesia in the mandible is the failure of the operator to appreciate the complexity of the innervation of the mandible and perimandibular soft tissues. In recent years the problem has become more pronounced because of new procedures which are performed at the periphery of the areas in which traditional mandibular out-



Fig. 6b.24 Incorporation of a gentle curvature in a 25 gauge 38 mm (1½ inch) needle in preparation for a mandibular block.



Fig. 6b.25 The slight curvature allows passage of the needle around the medial coronoid buttress to the mandibular groove.

patient procedures were performed. In particular, monocortical ramus and symphyseal grafts used in implant surgery require dissection in areas not adequately anesthetized by conventional local anesthetic approaches in the mandible.

Although the third division of the trigeminal nerve provides innervation to much of the mandible and the perimandibular soft tissues, a significant contribution is provided by the greater auricular and transverse cervical branches of the cervical plexus (see Fig 6b.26).³¹² Consequently, local anesthetic must be administered along the lateral aspect of the mandibular ramus and posterior mandibular body to take into account the innervation by greater auricular nerve. Anteriorly the branches of the transverse cervical nerve provide innervation along the inferior border of the mandible and beneath it. Thus local anesthetic must be given in these areas in order to achieve adequate anesthesia.

Another failure of mandibular anesthesia can be attributed to the anastomoses which exist between branches of the facial nerve (CN VII) and the trigeminal nerve and cervical plexus branches previously described (Fig. 6b.27).³¹³ The periosteum enveloping the mandible contains nociceptors from all three sources and wide infiltration in the areas of monocortical graft harvest is usually required.^{314–316}

Sensory branches from the mylohyoid nerve can also provide innervation to the lingual aspect of the

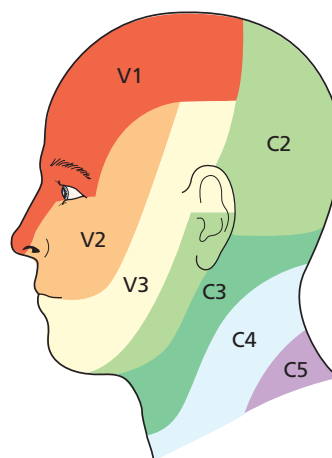


Fig. 6b.26 Innervation of the face and submandibular region. An important portion of the lower portion of the face along the lower and posterior borders of the mandible receives its innervation from the greater auricular and transverse cervical branches of the cervical plexus. (Reproduced from *Miller's Anesthesia* (Miller RD, ed.), 6th edn. New York: Elsevier/Churchill Livingstone, 2005; 1709, Fig. 1, by permission. Copyright © 2005 Elsevier.)

mandible in the face of an otherwise profound mandibular block. This may be in part due to individual patient variability in the level at which the nerve takes its origin from the third division of the trigeminal nerve.^{317,318} Once again, wide infiltration in the area of the distribution of the mylohyoid nerve on the medial aspect of the mandible is required to overcome this inadequacy in the delivery of profound mandibular anesthesia.

In the maxilla, the posterior portion of the hard palate receives innervation from the lesser palatine nerves as well as the greater palatine nerve (Fig. 6b.28).³¹⁹ Inadequate anesthesia in this area can be troublesome unless this anatomic relationship is appreciated. In the removal of maxillary third molar teeth, infiltrations of the palatal aspect of the posterior alveolus and tuberosity region are often necessary to adequately anesthetize the area for third molar removal.

There is extensive collateral innervation of the hard palate involving the nasopalatine nerves and the greater palatine nerves bilaterally (Fig. 6b.28).³¹⁹ Adequate anesthesia for surgery in the anterior palate will often require a greater palatine nerve block (or bilateral blocks) in addition to a nasopalatine nerve block.

The operator-anesthetist must be cognizant of these complexities of local anesthetic administration. When the patient begins to respond to surgical stimulation, reassessment of the adequacy of local anesthesia should be addressed, as opposed to merely administering large doses of intravenous anesthetic agents.

Local anesthetic agents

In the latter decades of the 20th century and first decade of the 21st century, new amide local anesthetics prilocaine, bupivacaine, and articaine became

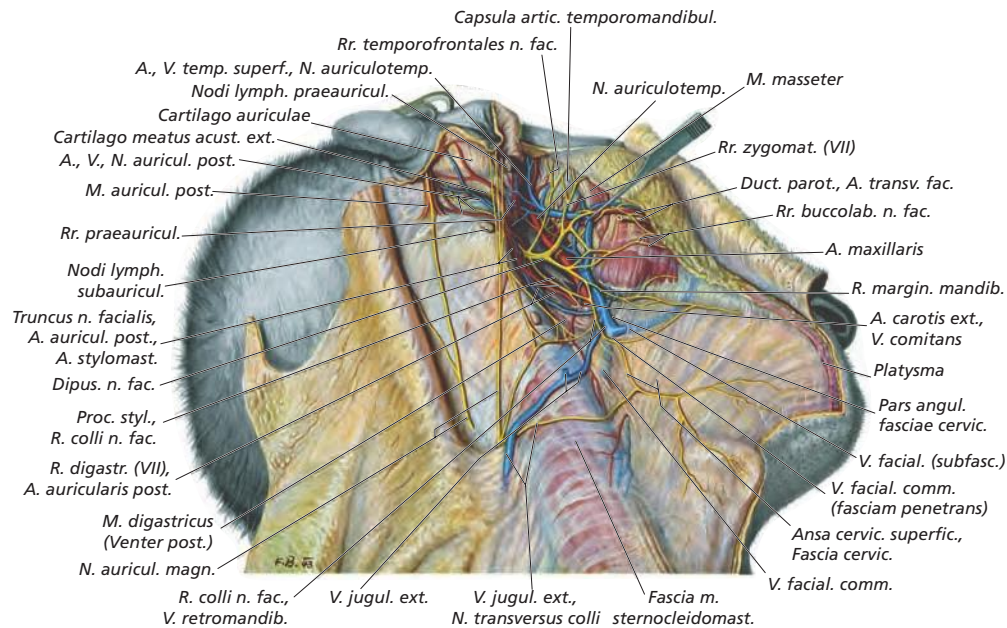


Fig. 6b.27 Anastomoses between branches of the cervical plexus and CN VII. These sensory fibers contribute to the sensory innervation of the lower portion of the face and the medial aspect of the mandible. (Reproduced from Pernkopf: *Atlas der topographischen und angewandten Anatomie des Menschen* © Urban & Schwarzenberg Verlag München, 238, Fig. 253 by permission.)

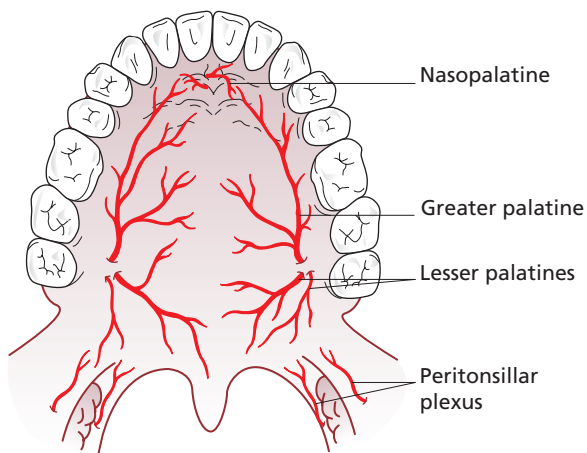


Fig. 6b.28 Sensory innervation of the palate. Reproduced with permission from *Anatomy for Surgeons: The Head and Neck* (Hollinshead WH, ed.), 2nd edn. New York: Hoeber Medical Division, 1968. Copyright © Lippincott Williams & Wilkins.)

available. These agents have properties which enable them to address needs not met by previous local anesthetics. Prilocaine, introduced in the mid-1960s, is potent and is available in a 4% concentration, which often enables the administration of adequate local anesthetic without epinephrine. This can be of benefit in patients who are sensitive to epinephrine or have severe cardiovascular disease which precludes its use. However, prilocaine can induce methemoglobinemia in higher doses,³²⁰ and it appears to be associated with more frequent reports of paresthesia than other local anesthetics.³²¹

In 1963 another new amide local anesthetic, bupivacaine, became available in the US.²⁴ It is considerably longer acting than traditional amide local anesthetics such as lidocaine and mepivacaine. Surgical patients who have received bupivacaine

often do not require postoperative pain medication until several hours after their anesthetics. By the time narcotic pain medication is necessary, the dizziness and sedation associated with the anesthetic have dissipated considerably, and it has been possible for the patient to take in oral fluids or a liquid diet. Thus, when narcotic pain medication does become necessary, it is less likely to cause nausea. Since some patients find the protracted period of anesthesia and hypesthesia unpleasant, it is best to advise the patient and/or his escort of the rationale for use of the long-acting anesthetic.

Within the last decade a final amide local anesthetic, articaine, has become available in the US.³²² It initially appeared that the agent might have greater potency than other anesthetics such as lidocaine, and it became quite popular. However, subsequent double-blind studies suggest that the efficacy of the agent is comparable to the more traditional lidocaine.³²³ The apparent potency of the agent appears to be in part due to the 4% solution in which it is formulated. In the 1990s reports from Haas in Canada suggested that articaine might be associated with a greater incidence of nerve toxicity leading to postoperative paresthesia.³²⁴ However, subsequent reports by Pogrel³²¹ in California in the US did not confirm any greater incidence of paresthesia with articaine as opposed to lidocaine.

Perioperative management

Preoperative preparation

Today the modern oral and maxillofacial surgery office has the appearance of, and functions as, a small

outpatient surgery center. As a consequence, perioperative management of patients treated in these offices is increasingly similar to that which a patient would encounter in an outpatient surgery center. By the end of the 20th century, the majority of surgical procedures were being performed in an outpatient setting.³⁵ Considerable data have been generated within the anesthesia literature regarding the enhancement of the anesthetic experience which accompanies careful attention to perioperative management. The astute oral and maxillofacial surgeon stands to learn much from these studies of perioperative management in the anesthesia literature. Some of those aspects which have particular application in an oral and maxillofacial surgery practice will be examined here.

Management of preoperative anxiety

There is a large body of data within the anesthesia literature examining the profound effect of high levels of preoperative anxiety on anesthetic outcomes.²³¹ If this anxiety is not appropriately managed, intraoperative anesthetic requirements increase, patient's satisfaction with the anesthetic diminishes, and in children there is an increase in emergence delirium and postoperative maladaptive behavior.³²⁵ Having a preoperative consultation appointment to discuss both surgical and anesthetic issues tends to diminish this anxiety.³²⁶ At the appointment it can also be determined whether one or more forms of preoperative medication would be appropriate for the given patient. In both adults and children benzodiazepines have been found to be the most efficacious pharmacologic agents for dealing with preoperative anxiety.^{327–329} Short-acting agents such as triazolam in teens and adults and midazolam in children provide anxiolysis and sedation which tend to reduce intraoperative anesthetic requirements, but do not appreciably lengthen the in-office recovery period.^{330,331}

It has also been reported in some studies that parental presence during induction of anesthesia (PPIA) can decrease preoperative anxiety in children.³³² In the oral and maxillofacial surgery office a practical approach to this practice is to allow parental presence until venipuncture has been accomplished and midazolam has been administered. If the patient does not appear to be adequately sedated to enable parental separation, 10–20 mg of propofol can be administered slowly to achieve the desired level of sedation.³³³ At this point, the patient is in a state of anxiolysis and sedation such that the parent can leave the operatory.

Preparation for venipuncture

Painful medical procedures such as venipuncture, dental care, immunization, and minor emergency department procedures such as laceration repair are a significant portion of the average child's experience

with painful events.^{334–337} If there is inadequate relief of the pain and anxiety which children experience during painful medical procedures, their experiences may have long-term (or even life-long) negative effects on pain tolerance and pain response.³³⁸ Consequently, it is essential that the oral and maxillofacial surgeon appreciates the potential long-term effects of a painful venipuncture experience, especially in children. These effects have been widely addressed in the medical literature in such fields as emergency medicine, immunology, diabetology, and allergy, all of which require frequent needle injections. Several methodologies have been extensively investigated including nitrous oxide, vapocoolant sprays, and topically applied anesthetics.

Nitrous oxide analgesia has long been used in dentistry and oral and maxillofacial surgery as well as other medical specialties³³⁹ to prepare patients for either venipuncture or intraoral injections. Although it is of some benefit, its weak analgesic properties limit its effectiveness. However, in combination with vapocoolant sprays or topical anesthetics, nitrous oxide has been found to be effective.^{340,341}

In teens and adults, a lengthy wait for a topical anesthetic to take effect in preparation for venipuncture creates scheduling problems.³⁴² Consequently, for these patients most surgeons who use a topical anesthetic prior to venipuncture tend to rely on a vapocoolant. In the past, the most popular agent has been ethyl chloride,^{343–345} but some studies have provided conflicting results as to the efficacy of coolant sprays.^{346,347} However, in recent years there has been renewed interest in the vapocoolant approach, and several new coolant sprays are now available.^{348,349} They have been extensively investigated with randomized double-blind studies. In such a study reported in 2008, a vapocoolant containing 1,1,1,3,3-pentafluoropropane and 1,1,1,2-tetrafluoroethane quickly and effectively reduced pain due to intravenous cannulation and improved the success rate of cannulation. This latter study addressed problems with some prior studies in which application time was more limited. The investigators suggested allowing 5–10 seconds of application of the spray and that the cannulation be attempted within 60 seconds of spray application.³⁵⁰ The author has found a similar approach with ethyl chloride to be effective.

In children, complete elimination of the pain of injection is preferable using topical anesthetics such as EMLA (eutectic mixture of local anesthetic). Adequate anesthesia, sufficient for intravenous cannulation, requires a thick layer³⁵¹ with a contact time of at least 1 hour (preferably 90 minutes)^{352–354} under an occlusive dressing^{355,356} (e.g. Tegaderm®, 3M Health Care, St. Paul, MN 55144). The depth of penetration is approximately 3–5 mm and the duration of action approximately 1–2 hours.^{355,357} Precannulation topical anesthetic application has been investigated extensively both in the US and abroad with positive results in the vast majority of studies.^{356,358–360} EMLA in com-

bination with nitrous oxide can yield a virtually pain-free venipuncture experience for children, with a high approval rating from both parents and children alike.

NPO status

Up until the last 20 years, 8 hours was considered the appropriate NPO period. However, for surgeries scheduled later in the morning or during the afternoon, the old guideline of “nothing after midnight” yields an excessive period of inadequate fluid intake. Consequently, in 1999 the ASA released new guidelines allowing consumption of clear liquids up to 2 hours before administration of anesthesia.³⁶¹ This latter guideline has been followed over the last decade and does not appear to have produced an increase in the incidence of aspiration. However, the patient must be warned that clear liquids does not include pulp-containing juices, such as orange juice, or fluids with fat globules, including milk products.

Delivery system

As discussed above, propofol has now largely replaced methohexital as the preferred major hypnotic agent for total intravenous anesthesia in OMFS offices. Although most anesthesiologists in hospitals and outpatient surgery centers administer propofol by an infusion pump,²⁹⁶ the majority of oral and maxillofacial surgeons tend to use the time-honored incremental bolus or “bump” technique which they have used for several decades with methohexital. However, infusion pumps are slowly increasing in

popularity²⁹⁵ and are receiving coverage at such major meetings as the Anesthesia Update which accompanies the AAOMS Annual Meeting.³⁶²

It is beyond the scope of this chapter to provide an in-depth discussion on the various infusion pumps and their operation. Rather we will briefly describe some of the general principles in setting up and using an infusion pump for the administration of a propofol-based anesthetic. The pump illustrated is the Baxter Infus O.R. Pump used by the author. This partial analog device has been used widely in hospitals and outpatient surgery centers over the last 10–20 years (see Fig. 6b.23).

The author’s technique calls for placement of a three-way stopcock on the 60ml pump syringe, with a conventional (30 inch) intravenous extension tubing attached (Fig. 6b.29a). The three-way stopcock allows additional propofol or remifentanyl to be added to the syringe during surgery without having to remove the syringe from the pump. In addition, at the end of the case, 5ml of saline can be placed in the syringe through the stopcock. Once the pump has restarted, the saline pushes the propofol in the extension tubing into the running intravenous line so that no propofol is wasted. The latter can be helpful if only a small amount of propofol is required at the end of the case during removal of the last tooth. The conventional short anesthesia extension (Fig. 6b.29b) is used for two reasons. The first is its short length and large bore which enable visualization, and thereby detection of crimping. In addition, it is less likely to become crimped because of its larger diameter and wall thickness.



Fig. 6b.29 (a) A three-way stopcock is placed between the pump syringe and the extension tubing. It enables the addition of more propofol, remifentanyl or saline during the surgical procedure. (b) The pump is borne by an anesthesia extension arm attached to the light pole of the operating chair. This eliminates the need for a separate IV stand. The conventional extension is in full view to allow monitoring for kinking.

Converting the patient's weight in pounds to kilograms provides an opportunity to incorporate gender compensation in the rate at which the propofol is administered. It has been well demonstrated that women require higher dosages of propofol, and some have suggested that propofol dosage should be increased by approximately 10–15% in women.^{159–161} The conventional method of converting pounds to kilograms is dividing the patient's weight in pounds by two and subtracting 10%. For women the weight in pounds can be divided by 2 without subtracting 10%. This then yields a body weight approximately 10% higher than the rate determined for a male patient.

The intravenous extension catheter from the pump is placed into the infusion port of the main intravenous catheter as illustrated in Fig. 6b.29b. The syringe used for the incremental boluses of ketamine can then be placed in the same administration port immediately adjacent to the extension catheter used for administration of the propofol. The dosages used for propofol and ketamine are listed in Box 6b.1. Using the combination of propofol and low-dose ketamine, the rates of administration of propofol can be kept at the lower end of the dosage range administered by the pump. Even when a patient is somewhat resistant to medication, it is usually only necessary to increase the rate by 1–2 levels on the pump. This still results in a rate below the "average rate" delivered by the pump. These settings are in keeping with the findings that propofol and ketamine have additive effects in total intravenous anesthesia, i.e. less propofol is required for hypnosis because of the addition of the dissociation which attends the administration of ketamine.



Fig. 6b.30 A piece of sterilization tubing is taped to the face of the pump so that pump adjustments can be made with a gloved hand during the procedure without breaking scrub.

Another adjunct which can be quite helpful in office-administered anesthesia is use of a sterile barrier to enable pump adjustment by the operator–anesthetist. The barrier used by the author is illustrated in Fig. 6b.30. It is made from a piece of sterilization tubing which is cut into 12-inch lengths. These are sterilized and then affixed to the base of the pump at the top and bottom with tape. Then, when adjustment of the pump becomes necessary, the surgeon can quickly adjust the pump without breaking scrub.

Airway management

During intravenous anesthesia airway maintenance is essential. In office-based anesthesia intubation is rarely employed, and other methods of airway maintenance must be used. This had traditionally consisted of either head tilt–chin lift or jaw thrust. In patients with significant obesity, retrognathia, or otherwise compromised airways, head tilt–chin lift and jaw thrust may provide inadequate airway maintenance. A simple intervention requiring no special equipment is the placement of a tongue traction suture (Fig. 6b.31) similar to that which has been used for maxillofacial trauma in the past. A double 3-0 chromic or silk suture is placed through the dorsum of the tongue at the junction of the middle and anterior thirds. The sutures are secured with a Kelly hemostat and the hemostat placed at the contralateral commissure. The traction brings the tongue forward, away from the posterior pharyngeal wall and maintains the airway.

Another intervention is placement of a nasopharyngeal catheter ("trumpet"). However, depend-

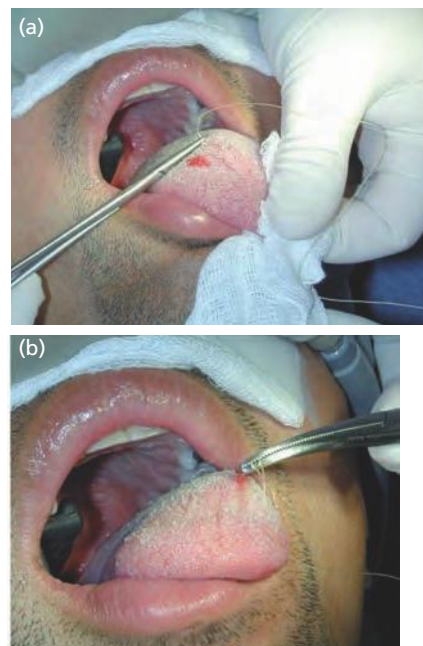


Fig. 6b.31 Tongue traction suture for airway maintenance. (a) Using the routine suture material for closure two sutures are passed through the dorsum of the tongue at the junction of the anterior and middle thirds near the midline. (b) The sutures are secured with a Kelly hemostat which drapes over the corner of the mouth at the commissure.

ing upon the patient's anatomy and the particular brand of catheter, the catheter may be too short and the tip may not pass behind the tongue.³⁶³ Thus airway maintenance may remain inadequate. Consequently, the tip of the tube should be visualized with a Weider retractor to assure that it indeed passes behind the tongue.

The LMA or laryngeal mask airway has gained popularity for both emergency airway intervention as well as administration of tube-delivered anesthesia.³⁶⁴ Since the LMA passes through the oral cavity, the conventional design may be cumbersome for oral and maxillofacial surgery procedures. During recent years a smaller-diameter "armored" tube (the LMA Flexible,[®] LMA North America, Inc., San Diego, CA) has been developed which is smaller, more flexible, and interferes less significantly with surgical access.

A final airway intervention is a mandibular traction wire placed around the mandibular posterior teeth (Fig. 6b.32). The latter allows translation of the mandibular condyle anteriorly during the surgical procedure. As the condyle translates forward, the attachment of the tongue attachments are brought forward with it, and the tongue is displaced anteriorly from contact with the posterior pharyngeal wall. In addition, this mandibular anterior positioning can facilitate access for surgical procedures in the mandibular ramus, such as monocortical ramus grafts used in implant reconstruction.

Monitoring

In the historical overview at the beginning of the chapter, it was noted that oral and maxillofacial surgeons adopted advanced monitoring techniques during the latter part of the 20th century.³⁷ Analysis of closed claims in anesthesia suggest that advanced

monitoring, such as pulse oximetry and capnography may prevent a significant percentage of anesthetic mishaps^{365,366} and anesthetic mortality.³⁶⁷ Standard monitoring would include blood pressure, pulse, EKG, pulse oximetry, and a monitor of respiration. The latter has traditionally included observation of chest excursions, movement of air in the anesthesia bag of the anesthesia machine, and use of a pretracheal stethoscope for monitoring breath sounds.^{368,369} However, within recent years capnography, or measurement of end-tidal carbon dioxide (ET-CO₂) has gradually increased in popularity.^{370,371} The capnograph uses infrared (IR) absorption spectrophotometry, based on the principle that certain gases (here carbon dioxide and water vapor) absorb IR light. As light is passed through the respired gases, the amount of IR light absorbed is proportional to the carbon dioxide present during the various phases of respiration.³⁷² There are two common IR analyzers in use, mainstream and sidestream. With mainstream capnography the detection chamber is placed at the end of the patient's endotracheal tube. These are commonly found in closed-system anesthesia machines used in inpatient surgery.³⁷³ The second type of analyzer is a sidestream analyzer which consists of a tube which aspirates a gas sample from the airway and diverts the gas through a sampling tube to an IR detector for analysis.³⁶⁷ The sampling tubes are susceptible to contamination by water, secretions, and occlusion of the tubing.³⁷² However, they can be easily modified and used in non-intubated patients³⁶⁷ such as those treated in an oral and maxillofacial surgery office (Fig. 6b.33).

The capnograph displays a continuous wave form of carbon dioxide data throughout the inspiratory and expiratory phases of respiration (a capnogram). In addition, it reports a numeric percentage value for the respired CO₂ (Fig. 6b.34). In an open system such as that used in oral and maxillofacial surgery, the absolute CO₂ value has little meaning. However, the trend of the wave form can be interpreted to assess events of essential importance, such as the sudden decrease in ET-CO₂ pressure which is indicative of airway obstruction³⁷⁴ or apnea.³⁷⁵ If the wave form becomes rounded with loss of the plateau, bronchospasm is a possible etiology.³⁷²

Capnography provides a more sensitive monitor of respiratory function than does pulse oximetry. However, although capnography rapidly signals that a significant respiratory event has taken place, it provides marginal information regarding the type of event. From this standpoint the traditional pretracheal stethoscope provides more information,³⁶⁹ and in real time.³⁷⁶ In the past some operators have considered the presence of the monitoring tube affixed to the ear as being cumbersome. Consequently, amplified pretracheal stethoscopes have been developed,^{377,378} and the more recently introduced of these use "Bluetooth technology" which allows a remote monitor either for



Fig. 6b.32 The mandibular traction wire – a 26 gauge stainless steel wire ligature is passed between the first and second molar teeth to the medial aspect of the mandible. It then passes lingual to the posterior teeth and finally to the buccal again between the first premolar and cuspid. The ends are twisted together and traction is placed on the mandible with a wire twister. The latter allows translation of the mandibular condyle anteriorly during the surgical procedure.

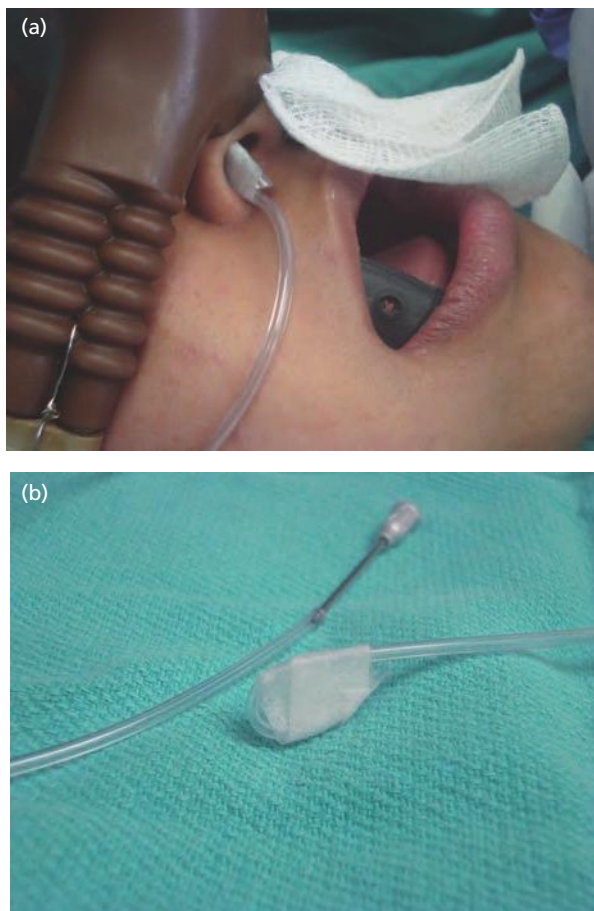


Fig. 6b.33 (a) Capnographic sidestream analyzer tubing. The tip is placed within the right naris. (b) Detail of the tubing illustrating contouring of the end of the tubing and protection with paper tape to prevent plugging with nasal secretions and occlusion by the nasal mucosa. (Photos courtesy of Henry Bennett, DDS, Davis, CA.)

the ear or a separate speaker which can be heard throughout the operatory (Fig. 6b.35).

Regardless of whether the stethoscope is the conventional tube-to-ear variety or amplified, the information which the apparatus provides is invaluable. When secretions, blood or irrigating fluid collect above the larynx, their presence is indicated by a gurgling sound. If the patient is developing a bronchospasm with wheezing and/or ronchi, these adventitious sounds can be readily appreciated.^{379,380}

Capnography has gained popularity in large part due to the inadequacies of pulse oximetry to truly monitor respiratory status in real time.³⁸¹ Pulse oximetry is based on differential absorption of red and infrared light by oxygenated and reduced hemoglobin (Lambert-Beer Law).³⁸² Oxyhemoglobin (HbO₂) absorbs more infrared light (960nm), while deoxyhemoglobin absorbs more red light (660nm). Thus deoxyhemoglobin appears to be somewhat blue, or cyanotic, even to the naked eye. The change in light absorption during arterial pulsations forms the basis for oxygen saturation measurement.³⁷⁴ Unfortunately, attempting to monitor the respiratory status of the patient with isolated pulse oximetry leads to a false



Fig. 6b.34 The capnographic display with a typical trend tracing and the CO₂ value. In this setting the tracing provides the essential monitoring information and the absolute CO₂ value has little meaning. (Photo courtesy of Henry Bennett, DDS, Davis, CA.)



Fig. 6b.35 Bluetooth technology allows a remote monitor for the precordial stethoscope to either the ear or a separate speaker. (Reproduced with permission of Surgical Solutions USA.)

sense of security.³⁸³ Due to the sigmoidal configuration of the oxygen-hemoglobin dissociation curve³⁸⁴ (Fig. 6b.36), a decrease in oxygen saturation from 100% to 90% can easily represent a drop in pO₂ from 100mmHg to almost 60mmHg.³⁸⁴ In addition, there is a time lag in the depression of oxygen saturation between peripheral arterial blood and central arterial blood.³⁸⁵ Due to the limitations of pulse oximetry, investigation into the monitoring of respiratory status resulted in the development of capnography.

Although capnography may be a more sensitive monitor of respiratory status than pulse oximetry, there has been considerable controversy as to whether its use should be considered a standard of care.^{386,387} As yet neither capnography nor auscultation with a pretracheal stethoscope is considered an essential parameter of care in the AAOMS Parameters of Care document.⁵⁰ Each is considered an alternative within a list of five entries including: observation of chest wall excursion, pretracheal stethoscope, observation of the reservoir bag, monitoring of skin mucosa, nail beds and surgical site, and monitoring of expiratory gases such as ET-CO₂.

In the mid-1990s BIS (bispectral index scale) was introduced as a new form of monitoring, based on the electroencephalogram (EEG). This BIS value is

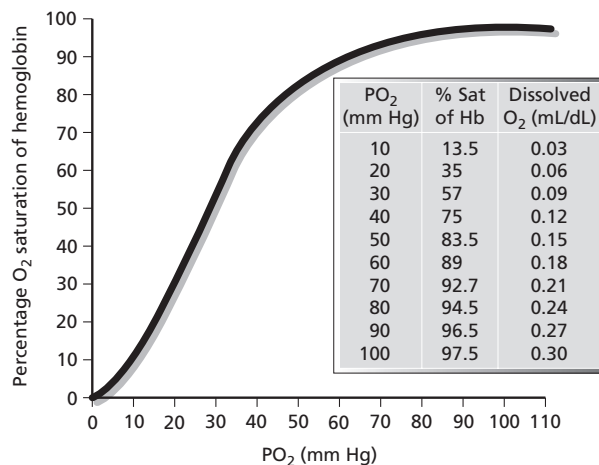


Fig. 6b.36 The oxygen-hemoglobin dissociation curve at pH 7.40 and temperature 38°C. As the oxygen saturation decreases below 90, note the precipitous drop in pO₂. (Reproduced from *Review of Medical Physiology* (Ganong WF, ed.), 22nd edn. New York: McGraw-Hill, 2005. With permission of the McGraw-Hill Companies.)

derived from a combination of the conventional analysis of the EEG using fast Fourier transformation and the interfrequency phase relationships among the underlying sinusoidal components of the EEG.³⁸⁸ An algorithm is used to integrate the two into a proprietary parameter termed the bispectral index.^{389,390} The latter is a dimensionless number which is scaled from zero to 100. A value of 100 represents the EEG of an awake individual, and zero represents complete electrical silence or cortical suppression. The various measured parameters have been integrated into this single variable. It is proposed that BIS values of 65–85 correspond to sedation and values of 40–65 to general anesthesia (Figs 6b.37–6b.39).³⁷⁴ This was derived by measurement of clinically relevant sedative end points and hypnotic drug concentrations in a cohort of anesthetized volunteers.³⁹¹ The algorithm has been revised on several occasions in an attempt to achieve maximal clinical relevancy.

Overall the BIS value is an indication of the potential for awareness and the hypnotic level of the patient to avoid hypnotic overdose.^{392,393} It cannot predict movement or hemodynamic response to stimulation, nor can it truly predict the exact moment at which consciousness returns.³⁸⁸ Since ketamine exhibits excitatory effects on the EEG, BIS cannot be used to accurately monitor hypnosis during ketamine anesthesia.³⁹⁴ However, when ketamine is used in conjunction with propofol, there is an additive interaction which can lead to hypnotic end-points detected by BIS.³⁹⁵ The reliability of BIS as a tool to prevent intraoperative awareness has been debated in the literature.³⁹⁶ Some studies indicate that it is efficacious, while others do not. The concept is an intriguing one, and it is possible that further refinement of the algorithms which form the basis of the BIS indicator will ultimately provide an indicator of unquestioned reliability.



Fig. 6b.37 Typical electrode placement on the forehead for the BIS monitor. (Photograph courtesy Robert Mraule, DDS, MD, Salinas, CA.)

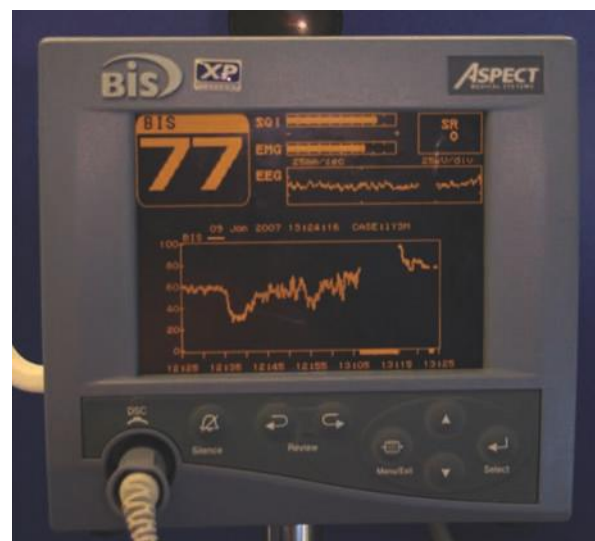


Fig. 6b.38 Typical display pattern on the BIS monitor.

Several studies have appeared in the oral and maxillofacial surgery literature reporting the use of BIS to monitor intravenous anesthesia for oral and maxillofacial surgery procedures. These studies indicated that use of BIS tended to result in a reduction in anesthetic requirement.^{397–400} However, the expense of the disposable electrodes (\$15–20 per patient) have tended to limit use of the technique.

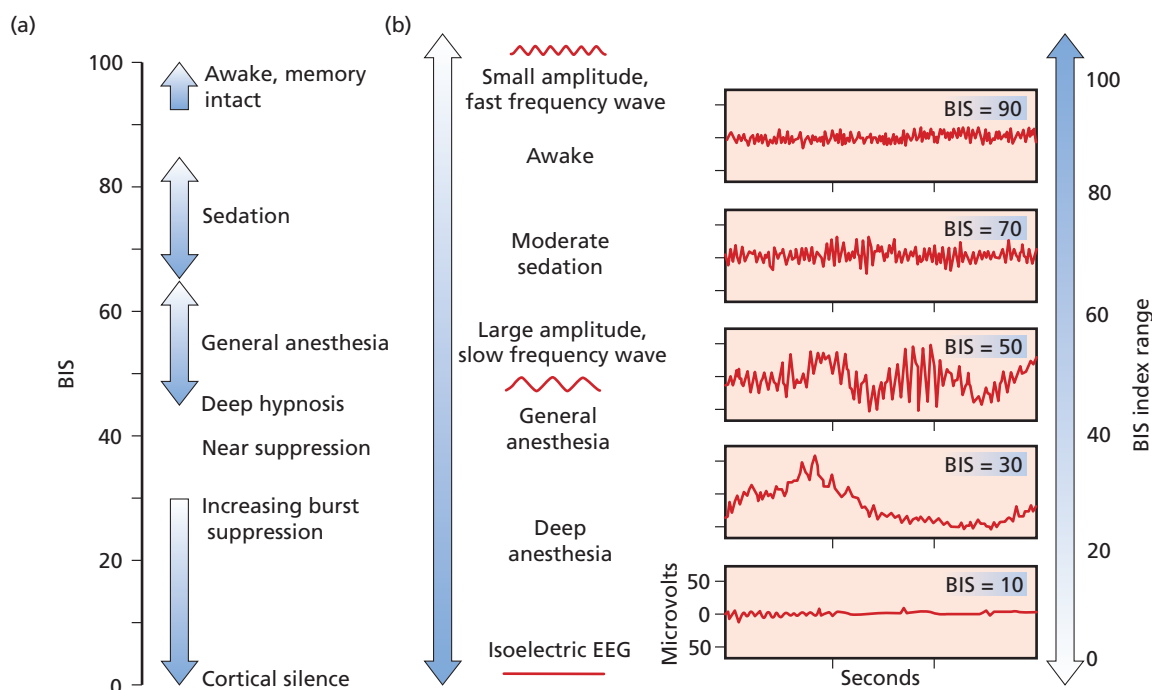


Fig. 6b.39 Correlation of BIS values with level of consciousness. (Reproduced from *Miller's Anesthesia* (Miller RD, ed.), 6th edn. New York: Elsevier/Churchill Livingstone, 2005; 1253, Fig. 31–21 by permission. Copyright © 2005 Elsevier.)

It must be remembered that intraoperative monitoring does not in itself prevent accidents. Rather, Eichhorn has suggested that monitoring standards “have initiated and provoked behavior patterns of thinking and use of equipment in such a way that intraoperative anesthesia accidents have been prevented”.⁴⁰¹

Awareness during anesthesia

As discussed in the previous section, the reliability of BIS to predict intraoperative awareness has been questioned. Unfortunately, there appears to be no tool which provides absolute reliability in determining awareness. The incidence of patients temporarily regaining consciousness or remaining conscious during surgical procedures has been estimated to be approximately 1 case in 2000.⁴⁰² Studies of closed claims reveal that the anesthetic techniques most commonly associated with awareness are those using opioids with muscle relaxants. However, several studies have suggested that awareness is also more likely to occur with nitrous oxide and intravenous agents such as propofol, barbiturates, benzodiazepines, and opioids.⁴⁰³

Prudent prevention strategies for awareness include the use of at least small doses of amnesic drugs such as midazolam and ketamine, as well as avoiding voicing negative or derogatory remarks about the patient or prognosis during surgery.⁴⁰⁴ However, the effects of amnesic agents are predominantly anterograde, and administering the agent after the patient becomes aware is usually of little benefit.⁴⁰⁵ It has also been maintained that awareness under intravenous anesthesia with propofol is far less

likely as long as a constant infusion is maintained with a pump.⁴⁰⁶

The ASA established a task force on intraoperative awareness which reported its findings in 2006.⁴⁰⁷ It concluded that conventional monitoring systems for EKG, blood pressure, heart rate, and ET-CO₂ are valuable to help assess intraoperative consciousness. The task force was equivocal in regard to the use of such monitors as BIS. They also were equivocal regarding the benefit of benzodiazepines in all patients.⁴⁰⁷

Intraoperative fluid management

Historically intravenous access in oral and maxillofacial surgery was provided with straight needles and butterfly-type needles and cannulae without a continuous intravenous infusion. However, the rigid hollow-bore needles were far more likely to cause vein laceration and infiltration than indwelling plastic catheters. Laceration and extravasations of fluid are particularly problematic in areas of extremity mobility such as the antecubital fossa, the wrist, or dorsum of the hand.¹⁴⁰ Over the last 10–20 years it has become common practice for oral and maxillofacial surgeons to use indwelling plastic catheters and provide a continuous intravenous infusion during the perioperative period.

Another change which has taken place within recent years is the nature of the fluid used for intravenous fluid replacement and rehydration. In the past the preferred maintenance fluid was 0.45% normal saline in 5% dextrose (D5/.45).^{408,409} However, it has been shown that the dextrose in such solutions is rap-

idly metabolized, which effectively results in the administration of a hypotonic infusion fluid. Consequently, it is now recommended that maintenance fluids in outpatient surgery consist of an isotonic solution such as normal saline.¹⁴⁰

Since patients must be NPO for their procedures, there is a fluid deficit which continues throughout the perioperative period. In the past, perioperative fluid administration was maintained at a relatively low volume of 1–2 ml/kg. However, studies over the last decade have demonstrated that higher volumes of 15–20 ml/kg are highly beneficial.^{410–412} With the higher rehydration rate, patients have fewer complaints of headache, nausea, vomiting, fatigue, light-headedness, dizziness, and thirst.⁴¹³

Recovery and discharge

With the shorter-acting agents currently employed, such as propofol, ketamine, and remifentanyl, recovery and discharge can take place in either the operating room or a separate recovery area. Regardless, patients should not be discharged until they can be safely transported home and continue to be safely monitored by their non-medical family member or escort. To assure that patients are not prematurely discharged, postanesthetic recovery scores have been developed. The first to gain wide acceptance was developed by Antonio Aldrete at the University of Colorado Medical Center in 1970.⁴¹⁴ The concept of scoring was patterned after the Apgar Scoring System developed by Dr Virginia Apgar for evaluation of neonates.⁴¹⁵ Aldrete's scoring system took into account five categories of recovery issues including activity, respiration, circulation, consciousness, and color (Table 6b.3). The system has been widely used in outpatient surgery and in oral and maxillofacial surgery for several decades.

A more recent alternative discharge scoring system for ambulatory surgery was developed by Frances Chung from the Toronto Hospital in Ontario, Canada in the mid-1990s.^{416–418} Its scoring categories include vital signs, activity level, nausea and vomiting, pain, and surgical bleeding (Table 6b.4). The criteria in this scoring system have greater relevance in oral and maxillofacial surgery than do those of the Aldrete score.

Special patient populations

In the interest of patient safety, a thorough preoperative evaluation should be performed for all patients. However, there are subgroups of patients who present risks which require special diligence in their preoperative evaluation and intraoperative management. These include pediatric and geriatric patients, those with a potentially compromised airway (e.g. the morbidly obese and patients with sleep apnea) and medically compromised patients.

Table 6b.3 The Modified Aldrete Scoring System to determine patient readiness for discharge (BP, blood pressure). (Reproduced from Chung F. Recovery pattern and home-readiness after ambulatory surgery. *Anesth Analg* 1995; 80: 896–902 by permission. Copyright © 1995 Lippincott Williams & Wilkins.)

Activity: able to move voluntarily or on command	
4 extremities	2
2 extremities	1
0 extremities	0
Respiration	
Able to deep breathe and cough freely	2
Dyspnea, shallow or limited breathing	1
Apneic	0
Circulation	
BP $\pm$ 20 mm of preanesthesia level	2
BP $\pm$ 20–50 mm of preanesthesia level	1
BP $\pm$ 50 mm of preanesthesia level	0
Consciousness	
Fully awake	2
Arousable on calling	1
Not responding	0
Color	
Normal	2
Pale, dusky, blotchy	1
Cyanotic	0

Table 6b.4 Postanesthetic discharge scoring system to determine patient readiness for discharge. Total score is 10; patients who score ≥ 9 are considered fit for discharge. (Reproduced from Chung F. Discharge criteria: a new trend. *Can J Anesth* 1995; 42: 1056. With kind permission of Springer Science and Business Media.)

Vital signs	
2 = within 20% of preoperative value	
1 = 20–40% preoperative value	
0 = 40% preoperative value	
Ambulation	
2 = steady gait/no dizziness	
1 = with assistance	
0 = none/dizziness	
Nausea/vomiting	
2 = minimal	
1 = moderate	
0 = severe	
Pain	
2 = minimal	
1 = moderate	
0 = severe	
Surgical bleeding	
2 = minimal	
1 = moderate	
0 = severe	

Pediatric patients

For many pediatric patients and their parents surgery in general, and oral and maxillofacial surgery in particular, is a decidedly anxiety-laden experience.³²⁵ If inadequately managed, this anxiety can make treatment exceedingly difficult for all involved, or indeed result in failure to complete the planned treatment. The two primary methods of dealing with patient and parent anxiety include premedication with an anxiolytic (usually midazolam)⁴¹⁹ and/or parent presence prior to induction of anesthesia (PPIA).^{420–424} A review of the literature suggests that when viewed as isolated approaches, premedication is probably more efficacious than parental presence.⁴¹⁷ However, some studies suggest that a combination of the two is more effective than either used independently.²³² With an adequately prepared parent at the patient's side, the parent can help distract the patient while the venipuncture is accomplished. In addition, the nitrous oxide mask helps to obscure the child's vision while venipuncture is in progress.

A particularly helpful development for pediatric patients has been the introduction of topical anesthetics such as EMLA for venipuncture. As discussed above, the EMLA must be left in place for at least 60–90 minutes to assure a painless injection. In addition, EMLA should be applied to an alternative venipuncture site in the event that the first attempt is unsuccessful. In combination with oral premedication, EMLA enables pediatric patients to progress through their treatment without any memory of painful injections. With the intravenous route established, the surgeon can titrate anesthetic agents, rehydrate the patient and have the intravenous route available in the event of an anesthetic or medical emergency. Various other approaches such as intramuscular injections, rectal suppositories, nasal sprays, etc. have been used. However, none have as high level of patient acceptance or safety as does the combination of oral premedication and establishment of the intravenous route with topical anesthesia.

For premedication midazolam is the most frequently used agent. It is available in a sweet-tasting elixir form and is given at a dose of 0.5 mg/kg. However, in recent years there has been a resurgence of interest in the α_2 agonist clonidine for anesthesia premedication in children. Studies from multiple centers in the US and abroad suggest that in an oral dose of 4–5 μ g/kg clonidine provides not only sedation equivalent to midazolam, but reduction of PONV, reduction of anesthetic requirements, post-operative analgesia, and no untoward effect on respiration.^{425,426} Clonidine will be discussed further in the section "Future trends and advancements – on the horizon", at the end of the chapter.

The primary anesthetic agents, propofol and ketamine, have particular efficacy in children. However, one must be mindful of the more rapid redistribution of these agents in the pediatric population.

Consequently, larger doses on a weight-for-weight basis must be used. Ketamine has been found to be far more efficacious in pediatric populations than in older patients. However, it carries with it the side-effect of hypersalivation, and an appropriate antisialogogue such as glycopyrrolate should be used. The glycopyrrolate tablet can be broken into appropriate dosage segments and crushed into a fine powder which can be administered along with the sweet-tasting benzodiazepine elixir, triazolam or clonidine in pulpless fruit punch.

In teens and adults, the initial low dose bolus of ketamine is often all that is required during the intravenous anesthetic. However, with children small incremental boluses of 2.5–5 mg can be administered every 10–15 minutes to ensure that the patient has sufficient dissociation throughout the procedure. In addition, ketamine has been shown to help preserve functional residual capacity in younger children, which provides an element of safety.⁶⁴

Geriatric patients

Oral and maxillofacial surgeons are seeing an ever-increasing number of geriatric patients in their practices. As discussed above, these patients require certain modifications in the normal routine of intravenous anesthesia. Virtually all of the agents used must be administered in lower doses, often at levels of 50% or less with progressive age. Dose guidelines for propofol are outlined in Table 6b.1.

In the past, midazolam and other benzodiazepines have been used fairly extensively in the treatment of the elderly. They were viewed as being the "safest" agents because of the minimal cardiovascular and respiratory depression with which they are associated. However, there is a growing body of literature which suggests that anesthesia, including outpatient anesthesia similar to that used in oral and maxillofacial surgery offices, can be associated with cognitive dysfunction following surgery. Fortunately, the incidence of cognitive dysfunction appears to be smaller in the "day care" setting.⁴²⁷ It often resolves within 3 months in middle-aged patients,^{428,429} but can be distressingly persistent.

Of the agents in common use, the benzodiazepines are those which have the most profound effects on memory. Amnesia to the surgical event is usually considered a desirable quality of an agent. However, if this impairment of memory persists for long periods following the procedure, the desirable effect becomes an undesirable one. As an appreciation of long-term cognitive dysfunction in older patients has increased among anesthesiologists, many have markedly reduced dosage levels of benzodiazepines or eliminated them altogether (personal communication, Andrew Herlich, DDS, MD). Without a benzodiazepine, there is a somewhat greater likelihood that the patient will experience awareness during the surgical procedure. This should be addressed with the

patient and family prior to the procedure, with assurance that the reason is to prevent any long-term memory effects. Most elderly patients and their families would prefer to have some awareness as opposed to long-term memory effects.

Patients with compromised airway

At the preoperative consultation, evaluation of the airway is essential. Several different guidelines have been developed including the Mallampati score,⁴³⁰ the upper lip bite test (ULBT),⁴³¹ and submandibular measurements from the hyoid bone and thyroid cartilage and sternum to the anterior mandible. The Mallampati criteria and ULBT are illustrated in Figs 6b.40 and 6b.41. Clinical measurements which have been used include the thyro-mental distance, which should be at least 5cm (three wide finger breadths),⁴³² the sterno-mental distance which should be greater than 12.5cm and the hyo-mental distance of approximately 6cm.⁴³³ A neck circumference greater than 43cm (17 inches) has been associated

with a compromised airway and difficult intubation.⁴³⁴ Another critical measurement is the extent of mouth opening as measured between the maxillary and mandibular incisors which should be at least 30–40mm.⁴³⁵ In the literature various authors have commented on the inadequacy of any of these tests and measurements taken independently. However, a composite of several of them is usually considered to yield a predictability of approximately 80%.⁴³⁶

Although airway difficulties may be seen in a variety of settings, special care must be taken in the management of the airway in patients who are morbidly obese or suffer from obstructive sleep apnea (OSA).⁴³⁷ Patients should have the body mass index (BMI) calculated: $BMI = \text{wt. in kg} \div \text{ht. in m}^2$ or $BMI = (\text{wt. in lbs.} \div \text{ht. in inches}^2) \times 703$ and categorized (see Fig. 6b.42).^{438,439} Many feel that patients who have a BMI of greater than 35 may not be acceptable candidates for anesthesia in an office setting.⁴⁴⁰ Sleep apnea often goes undiagnosed or patients may exhibit denial.⁴⁴¹ A valuable screening tool for OSA is the STOP-Bang Scoring Model developed by Chung *et al.* from the Department of Anesthesia at the University of Toronto.⁴⁴² The questionnaire is illustrated in Table 6b.5.

There are several precautions which should be taken in the treatment of morbidly obese patients and patients with sleep apnea. The surgeon should consider performing the procedure with the patient in a semi-upright position. In choosing a blood pressure cuff for these patients, one should choose a cuff with a width greater than one third the circumference of the arm.⁴⁴³ When narrower cuffs are used, a false high systolic blood pressure will be recorded. Close attention to the airway is essential and airway adjuncts such as a tongue traction suture or nasopharyngeal airway may need to be considered. In general, anesthetic and sedative medications should be dosed according to lean body mass as opposed to total body weight. Short-acting medications, such as propofol and low-dose ketamine and remifentanyl, are preferred.⁴⁴⁴

The medically compromised

A comprehensive discussion of medical compromise as it affects anesthesia is beyond the scope of this chapter. However, a valuable tool in the management of these patients is use of faxed medical clearance correspondence. When the clinician encounters aspects of a patient's medical compromise which can influence anesthesia and surgery, a succinct faxed letter should be sent to the patient's physician regarding appropriate management. The surgeon's attempting to contact the physician by phone is often problematic because of scheduling problems in both offices. The fax provides unequivocal written clearance which is dated and timed as opposed to verbal clearance that can be misconstrued or denied.

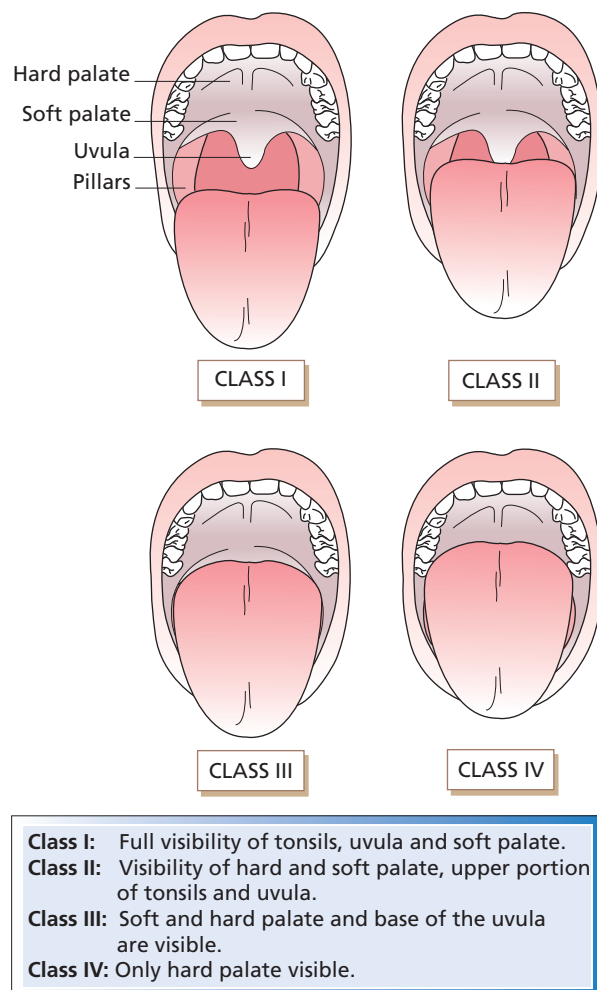


Fig. 6b.40 The Mallampati classification. The patient is instructed to open his mouth maximally and to protrude his tongue as far as he can. (Reproduced from *Clinical Anesthesiology* (Morgan GE, Mikhail M, Murray M, eds.), 4th edn. New York: McGraw-Hill, 2006. With permission of the McGraw-Hill Companies.)

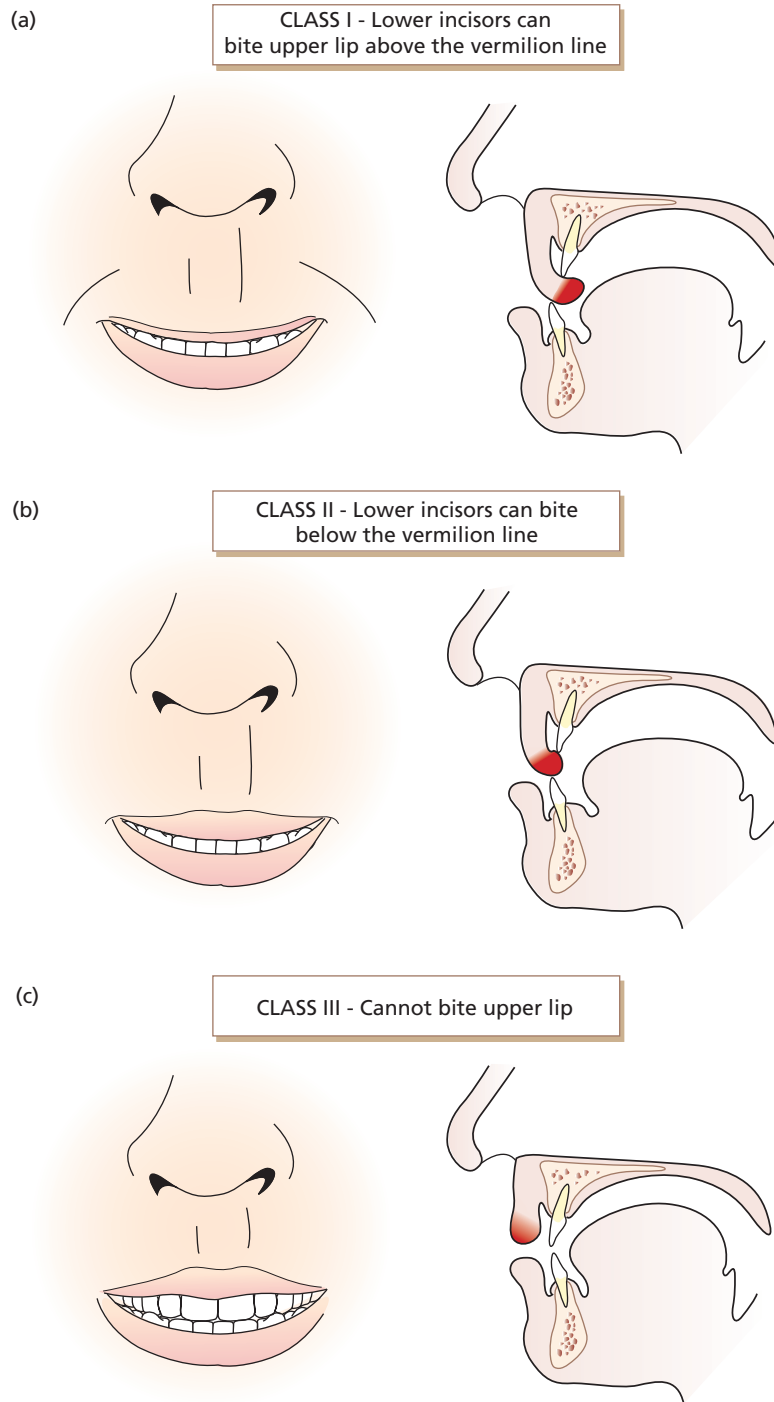


Fig. 6b.41 The upper lip bite test. The patient is instructed to protrude his mandible as far as he can and to bite into the upper lip. (Reproduced from Khan Z, Kashfi A, Ebrahimkhani E. A comparison of the upper lip bite test. *Anesth Analg* 2003; 96: 595–9 by permission. Copyright © 2003 Lippincott Williams & Wilkins.)

In some states, such as California, the status of faxed documents as evidence has been addressed by both legislation and the courts (California Civil Code: 1633.13 and 1633.14, California Evidence Code 1420 & 1421). In addition, the California Court of Appeals held in *Hoffer V. Young*, 38 Cal. App. 4th 52(1995), that a fax was just as good as sending a letter.

When the initial communication is verbal by phone, a confirming fax can be used to document the discussion. Thus the verbal communication is converted into a form of written communication

which serves as far better documentation than the handwritten notes regarding the conversations that are entered by the surgeon in the patient's chart (personal communication, Arthur Curley, JD, Larkspur, CA).

Patients who are at a high level ASA III classification are generally not considered to be acceptable candidates for treatment in an office setting.¹⁴⁰ In such cases it is prudent to consider treatment of the patient in an outpatient surgery center or hospital setting.

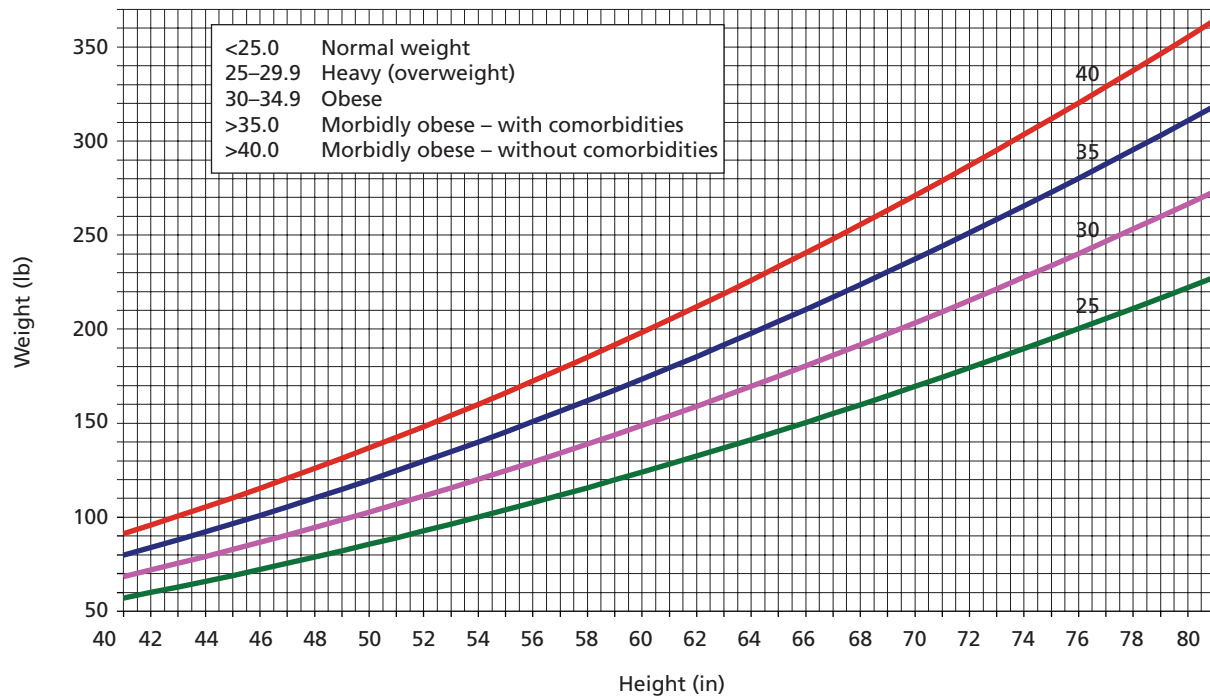


Fig. 6b.42 Body mass index (BMI) graph illustrating the relationship between patient's height in inches and weight in pounds for determination of the BMI.

Table 6b.5 The STOP-Bang screening questionnaire for obstructive sleep apnea. (Reproduced from Chung F, Yegneswaran B, Liao P, *et al.* STOP questionnaire: a tool to screen patients for obstructive sleep apnea. *Anesthesiology* 2008; 108: 812–21 by permission. Copyright © 2008 Lippincott Williams & Wilkins.)

1.	Snoring	
	Do you snore loudly (louder than talking or loud enough to be heard through closed doors)?	
	Yes	No
2.	Tired	
	Do you often feel tired, fatigued, or sleepy during daytime?	
	Yes	No
3.	Observed	
	Has anyone observed you stop breathing during your sleep?	
	Yes	No
4.	Blood pressure	
	Do you have or are you being treated for high blood pressure?	
	Yes	No
5.	BMI	
	BMI more than 35 kg/m ² ?	
	Yes	No
6.	Age	
	Age over 50 years old?	
	Yes	No
7.	Neck circumference	
	Neck circumference greater than 40 cm?	
	Yes	No
8.	Gender	
	Gender male?	
	Yes	No

High risk of OSA: answering yes to three or more items

Low risk of OSA: answering yes to less than three items.

Future trends and advancements – on the horizon

Unquestionably propofol has dramatically changed outpatient anesthesia in many ways. However, one of its major drawbacks is its insolubility in water which has made it necessary for it to be formulated in a liquid emulsion.⁴⁴⁵ Consequently, a water-soluble pro-drug of propofol termed “fospropofol” has now been formulated.⁴⁴⁶ Initial studies indicate that the drug has a somewhat longer time to peak concentration after bolus^{148,445} and a longer half-life. Initial studies have disclosed no major disadvantages to the formulation and even suggest that there may be a tendency for fewer and shorter duration of periods of apnea.⁴⁴⁷

Like propofol, remifentanyl has suffered from stability and preparation problems. Currently the smallest vial marketed is 1 mg of the remifentanyl powder.¹⁰⁴ Since the agent is so potent, this is sufficient agent for the treatment of 15–20 patients. However, the manufacturer states that the solution is only stable for 24 hours.⁴⁴ In most office and outpatient surgery center settings, this frequently leads to considerable waste. Although the problem has been brought to the attention of the manufacturer, smaller vials have not as yet been offered.

Interestingly the α_2 agonist clonidine, originally developed as an antihypertensive,⁴⁴⁸ has been shown to have application for anesthesia premedication, especially in pediatrics. It was introduced for this purpose in 1993 by Mikawa and co-workers.⁴⁴⁹ The agent provides anxiolysis, sedation,^{425,426} enhancement of postoperative analgesia,⁴⁵⁰ as well as reduction of shivering⁴⁵¹ and PONV. However, there were a few negative side-effects such as bradycardia, hypo-

tension or respiratory depression.⁴⁵² Bergendahl *et al.* suggest that the reason for clonidine's lack of routine use in clinical practice is due to the fact that there are multiple generic preparations available. Therefore, there is no financial inducement for the pharmaceutical industry to promote the drug.⁴²⁵

A second α_2 agonist, dexmedetomidine, is eight times more specific for the α_2 receptor than clonidine.^{127,453} The new agent has now been studied as a sedation agent for third molar surgery. Heart rate and blood pressure during surgery were somewhat lower in the dexmedetomidine group and there was less amnesia as opposed to the midazolam group.^{454,455} Since the drug is currently quite expensive, it is likely that its use for third molar surgery will be limited until it becomes available in generic form.

There are two intriguing medications which are awaiting approval in the US by the Federal Drug Administration. The first of these is hardly "new" in Europe, where it has been in use for sometime. This is the S+ enantiomer of ketamine.^{134,205} Of the two isomers it appears to have superior properties, including greater potency, as well as more rapid effect and redistribution. Unfortunately, it has not been found to be without some of the psychotomimetic and cardiovascular side-effects of the racemic mixture.²³⁴ Those using the agent in Europe appear to prefer the S+ isomer variant to the racemic mixture, and it is hoped that FDA approval will be forthcoming.

An especially interesting new drug is sugammadex, a polydextran compound which has been found to encapsulate the non-depolarizing muscle relaxant rocuronium.^{456,457} Anesthesiologists and oral and maxillofacial surgeons have long sought a replacement for the polarizing muscle relaxant succinylcholine. Amongst several undesirable properties are its role as a potential triggering agent for malignant hyperthermia and its tendency to cause bradydysrhythmias.⁴⁵⁸ Alternative non-depolarizing agents such as rocuronium have a far longer duration of action, which make them somewhat impractical for treatment of laryngospasm. However, through encapsulation sugammadex totally inactivates rocuronium quite rapidly. Consequently, rocuronium could then be used just as effectively as succinylcholine for reversal of laryngospasm without the significant liabilities of succinylcholine.⁴⁵⁹ Numerous reports in the literature point to the high safety and lack of adverse side-effects of sugammadex. However, it is still awaiting final approval by the drug administration.

Conclusion

In this chapter we have watched the evolution of anesthesia in oral and maxillofacial surgery from the "one man show" which was Gardner Quincy Colton in the mid-19th century to state-of-the-art outpatient anesthesia practice which exists in oral and maxillo-

facial surgery practices throughout the US today. As we have seen, the success of oral and maxillofacial surgeons in maintaining general anesthesia and sedation as an integral part of their practices can be largely attributed to organizational efforts within the specialty. The AAOMS is strongly supported by its membership and the organization can effectively coordinate its efforts with other organizations such as the ASA, ADA, ADSA, etc. Surgical anesthesia was first demonstrated by dentists, and oral and maxillofacial surgeons, as dental specialists, have been at the forefront of advancements, such as the development of office-based anesthesia. There is every indication that this pre-eminence in the field of anesthesia will continue as the 21st century continues to unfold.

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Chapter 7

Dentofacial Infection

Ashraf Ayoub

This chapter will cover infections of odontogenic origin, including the fascial spaces of the head and neck in which these infections can manifest themselves. The appearance and management of fascial space infections is discussed. Additionally, this chapter will discuss specific infections of the oral cavity and related structures which may be of dental origin. These will include osteomyelitis in various forms, osteoradionecrosis, bisphosphonate-related osteonecrosis of the jaws, cavernous sinus thrombosis, necrotizing fasciitis, actinomycosis, tuberculosis (TB), and syphilis.

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Dentoalveolar abscess

The acute dentoalveolar abscess is a localized suppurative inflammation which involves the teeth and the supporting periodontal structures (Fig. 7.1). The pathogenic microorganisms which are predominantly anaerobes normally gain entry to the periapical tissue through the necrotic pulp of carious teeth¹ or via the gingival crevice of the periodontal ligament. The most typical clinical features are severe pain and exquisite tenderness on percussion or touching the tooth during mastication. The pain is due to the local release of inflammatory mediators such as kinins and histamines, and fluid exudates causing increased pressure in the confines of underlying alveolar bone. Pyrexia and lymphadenopathy can be common symptoms associated with this infection. Prompt evacuation of pus by either extracting the tooth or removing the infective pulp reduces the internal pressure and the patient's pain.

The fate of the acute dentoalveolar abscess depends on the number and the virulence of the invading microorganisms, as well as the resistance of the patient. The spread of the infection will be governed by the position of the tooth in the alveolus and rela-

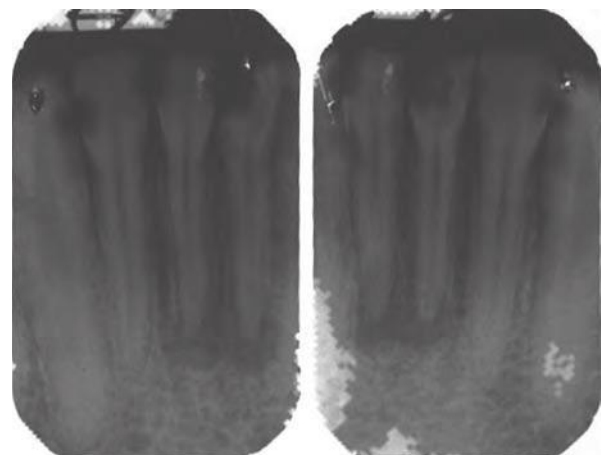


Fig. 7.1 Radiograph of dental abscess.

Introduction

Inflammation, infection, and pus formation were first described in Egyptian papyri. Inflammation is defined as reaction of the tissues to an invasion by pathogenic microorganisms. These reactions tend to destroy and limit the spread of infectious agents and repair the damaged tissues. The inflammatory process involves fluid and cellular exudates in an attempt to destroy and engulf the invading microorganism.

tionship of the apices of the infected teeth to muscle attachments and their relative proximity to the lingual and buccal surfaces of the jaw bones.

Infection originating from a dental abscess may spread to involve the deeper tissues of the head and neck region, once the abscess has broken through the surface (usually buccally) of the alveolar bone. On the other hand, the invading microorganisms may be destroyed by the natural local resistance of the body, prescription of antibiotics, and removal of the source of infection by extraction or endodontic means. The lesion would then resolve. However, in some instances, lack of appropriate treatment may lead to the development of a chronic dentoalveolar abscess where the lesion never heals by itself but persists as a lesion of low-grade virulence, characteristic of the typical granuloma. Chronic dentoalveolar abscesses and periapical granulomas are generally asymptomatic unless, or until, reignited in an acute phase. Only the development of an acute exacerbation would lead to the cardinal signs of inflammation such as tenderness, pain, redness, and swelling. Sinus formation, either intraorally or extraorally, is not an infrequent feature of this chronic infection. The chronic lesion has the potential to stimulate epithelial cells and initiate the development of a dental cyst.

Radiographic appearance

In the acute phase, the earliest changes are thinning of the periodontal space followed by loss of apical lamina dura, then radiolucency with ill defined margins. Chronic abscesses or apical granuloma are usually associated with a well circumscribed radiolucency.

Site and spread of infection

Bone, muscle, aponeurosis or fascia, neurovascular bundles, and skin, can act as barriers against spread of infection. However none of these tissue barriers or boundaries is so restrictive or containing that they necessarily prevent the spread of infection into contiguous anatomic spaces (Fig. 7.2).² The following are the most common routes for the spread of infection from dentoalveolar infections:

- upper lip;
- canine fossa;
- infraorbital region;
- buccal space (5 in Fig. 7.2);
- palate;
- submasseteric (3 in Fig. 7.2);
- retropharyngeal;
- lateral pharyngeal (1 in Fig. 7.2);
- pterygomandibular space (2 in Fig. 7.2);
- infratemporal fossa;
- parotid space;
- buccinator space (4 in Fig. 7.2);
- suprahyoid region;

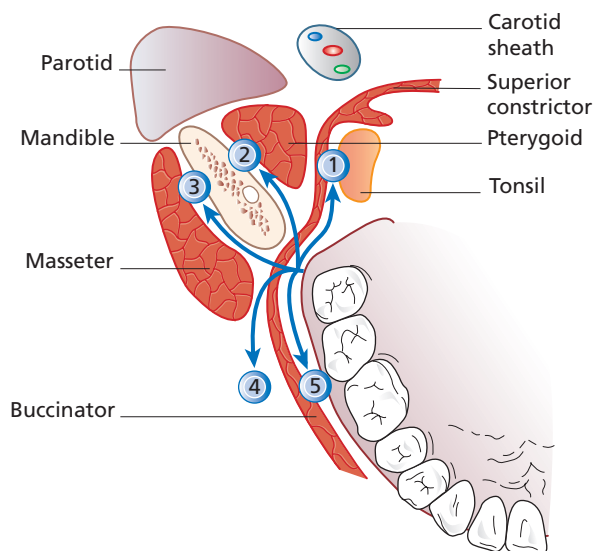


Fig. 7.2 Direction of spread of infection, and the fascial spaces involved, from the lower third molar site.

- submandibular space;
- submental space;
- sublingual space.

Upper lip

Infection at the base of the upper lip originates from the upper anterior teeth. It spreads on to the orbicularis muscle from the labial sulcus between the levator labii superioris muscle and the levator angularis oris muscle.

Canine fossa

Spread of infection to the canine fossa usually originates from maxillary canine or upper premolar teeth, often presenting above the buccinator muscle attachment. These swellings obliterate the nasolabial fold (Fig. 7.3).

This space is in close proximity to the lower eyelids, and therefore early management is mandatory to avoid circumorbital infection. There is a risk of spread cranially via the external angular vein which may become thrombosed.

Buccal space

The attachment of the buccinator muscle to the base of the alveolar process can control the spread of infection in the region of the mandibular and maxillary molars. The infection spreads intraorally superficial to the buccinator muscle in front of the anterior border of the masseter muscle. Therefore the clinical manifestation of the infection of this space is characterized by swelling confined to the cheek (Fig. 7.4). However, infection may spread superiorly towards the temporal space, inferiorly to the submandibular space or posteriorly into the masseteric space. In some



Fig. 7.3 Clinical infection of the upper teeth spreading to the canine fossa.



Fig. 7.4 Clinical infection of the buccal space.

cases infection may spread to the surface of the skin, leading to fistula formation (Fig. 7.5).

Palate

The palate is usually involved by infection originating from the maxillary lateral incisor or the palatal roots of the posterior teeth. The infection spreads from the apices of these teeth, perforating the palatal alveolar bone, and pus accumulates below the palatal mucoperiosteum (Fig. 7.6). It is important to be aware



Fig. 7.5 Cheek infection secondary to spread of infection from an upper molar.



Fig. 7.6 A palatal abscess from an upper lateral incisor.

that, although the lateral incisor is the commonest source of palatal abscess, most still present labially.

Pterygomandibular space

Infection in this space is manifested by trismus due to the involvement of the pterygoid muscles. This space is bounded medially by the medial pterygoid muscle and laterally by the medial surface of the mandible, anteriorly by the pterygomandibular raphe, and posteriorly by the deep lobe of the parotid gland. The lateral pterygoid muscle forms the roof of this space (Fig. 7.7).

Submasseteric space

The commonest source of infection in this space is from lower third molar pericoronitis. This space is bound medially by the masseter muscle and laterally by the outer surface of the ramus of the mandible (Fig. 7.8). It is in direct communication with the lateral pharyngeal space posteriorly. The temporalis muscle divides the superior part of this space into two portions the superficial temporal space, which is bounded by temporalis muscle medially, and the deep temporal space with the temporalis muscle laterally and the periosteum of the temporal bone medially.

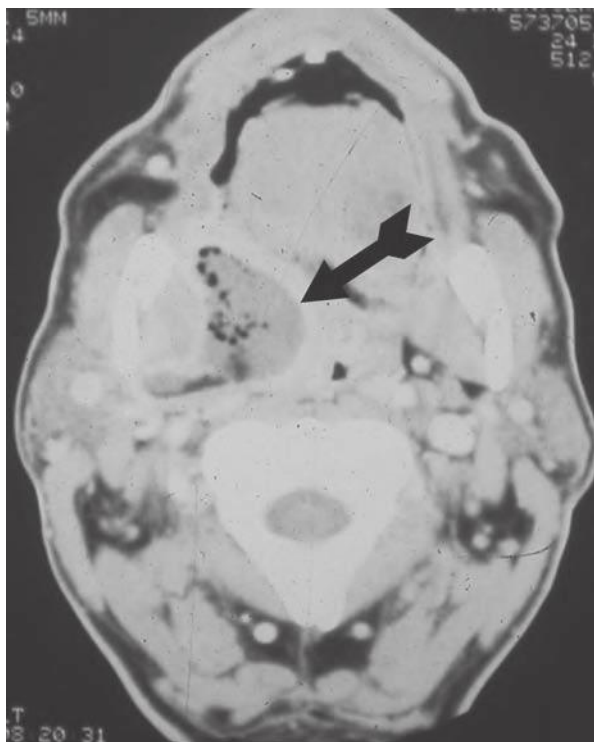


Fig. 7.7 Axial computed tomography (CT) scan of pterygomandibular space infection.

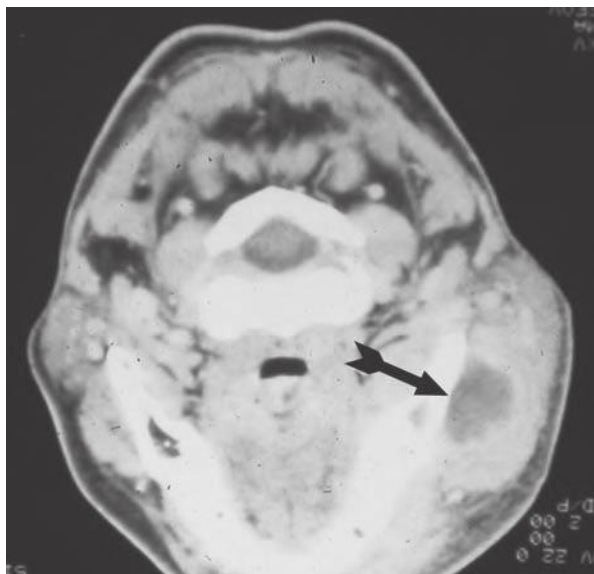


Fig. 7.8 Axial CT scan of a submasseteric abscess.

Severe trismus due to spasm of the masseter muscle is a characteristic feature of involvement of this fascial space.

Infratemporal space

Extension of infection from maxillary molars can pass into this space. Infection may also spread from the pterygomandibular, parotid or lateral pharyngeal region to the infratemporal space. The patient then complains of pain, particularly with mouth opening, some dysphagia, and difficulty with lateral mandibular movements. This space is located behind the zygomatic bone posterior to the maxilla and medial to the insertion of the medial pterygoid muscle. The infratemporal space is bounded superiorly by the greater wing of the sphenoid and is in close proximity to the inferior orbital fissure with a possible risk of spread of infection to the orbit.

Parotid space

Involvement of this space may be an extension of infection from the middle ear or the mastoid region. Infection from the masseteric or the lateral pharyngeal space may also spread to the parotid region. Therefore, the most characteristic feature of involvement of this space is swelling of the parotid gland region below the ear lobe. This space contains several important structures which may be affected by infections. These include the 7th cranial nerve, auriculotemporal nerve, facial vein, the parotid lymph node, and, more deeply, the external carotid with its branches.

Submandibular space

This space is located below the mylohyoid muscle, medial to the ramus and body of the mandible. It is bounded anteriorly by the attachments of the anterior belly of the digastric muscle and posteriorly by the posterior belly of digastric muscle and stylomandibular ligament. Infection from the posterior mandibular teeth may pass lingually below the attachment of the mylohyoid muscle into this space. Clinically, swelling of the submandibular regions tends to obliterate the angle of the mandible, causing pain and redness of the skin overlying this region. Dysphagia is usually a marked symptom (Fig. 7.9).

Submental space

This space lies between the two anterior bellies of the digastric muscle. Anteriorly and laterally this space is bounded by the body of the mandible (Fig. 7.10). It is contained superficially by the platysma muscle and deeply and superiorly by the mylohyoid muscle. The infection of this space usually arises from mandibular anterior teeth where the infection perforates the lingual cortex; swelling of the submental region is a characteristic clinical feature. The skin over the swell-



Fig. 7.9 Clinical picture of submandibular space infection.



Fig. 7.10 Clinical picture of submental abscess.

ing is stretched and hardened, and the patient experiences considerable pain and difficulty with swallowing. The infection may progress buccally causing swelling in the labial sulcus and over the chin.

Sublingual space

Infection spreads into this space as the result of perforation of the lingual cortex above the attachment of the mylohyoid muscle. This space is bounded superiorly by mucous membrane and inferiorly by the mylohyoid muscle. The genioglossus and geniohyoid muscles form the medial boundary. Laterally, this space is bounded by the lingual surface of the mandible. Infection in this space will raise the floor of the mouth and displace the tongue medially and posteriorly. The tongue displacement may compromise the airway and immediate intervention may be required. Dysphagia and difficulty in speech are also common.

Pharyngeal space

This space is located on the lateral side of the neck, bounded medially by the superior constrictor muscle

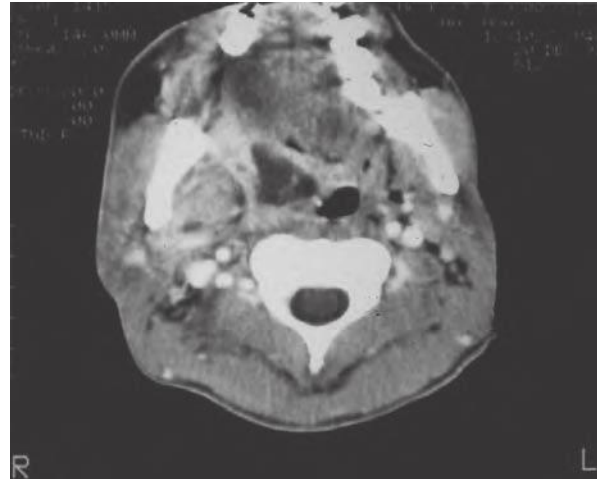


Fig. 7.11 Axial CT scan of pharyngeal space infection.

of the pharynx and posterolaterally by the parotid space. Infection in this space may originate from mandibular molars or third molar pericoronal supuration. This could also be a site of spread of infection from the parotid space or fascial space around the body of the mandible. The lateral pharyngeal space contains the carotid sheath, glossopharyngeal nerve, accessory nerve, and the hypoglossal nerve, as well as the sympathetic trunk. Therefore, spread of infection into this space carries the significant danger of spread into a descending neck infection and involvement of the mediastinum. Clinically, stiffness of the neck, swelling of the lateral wall of the pharynx, medial displacement of the tonsils, dysphagia, and trismus are among the characteristic clinical features of involvement of this space (Fig. 7.11).

Retropharyngeal space

This space is located between the posterior wall of the pharynx and the prevertebral fascia. This space is in direct communication with the base of the skull superiorly and the mediastinum inferiorly. It has the same characteristic clinical features as infection of the lateral pharyngeal space and carries the significant complication of a descending neck infection.³

Microbiology of dental infections

Recent reports conformed that there is usually a polymicrobial cause for oral/dental infection comprising facultative anaerobes, such as viridans group streptococci and the *Streptococcus anginosus* group, with predominantly strict anaerobes, such as anaerobic cocci, *Prevotella* and *Fusobacterium* species. The use of sophisticated non-culture methods has identified a wider range of organisms such as *Treponema* species and anaerobic Gram-positive rods such as *Bulleidia extructa*, *Cryptobacterium curtum*, and *Mogibacterium timidum*.⁴

Management of patients with orofacial infection

The management of orofacial infection involves surgical intervention to drain localized pus and medical support for the patient.⁵

1. Incision and drainage. This could be carried out either intraorally or extraorally depending on the location of the infection (Fig. 7.12). Aspiration of pus prior to incision allows a more accurate sampling method as it reduces contamination and helps preserve anaerobic bacteria. Fluctuation of the swelling indicates the presence of pus and is defined as fluid transmission using bi-digital palpation.
2. Antibiotics. Antibiotics can be provided empirically or a specific antibiotic given based on culture and sensitivity tests. Penicillin has the potential to be the first-line agent in the treatment of odontogenic infections. Most other beta-lactam antibiotics, including fourth-generation cephalosporins, were not found to have greater effectiveness than penicillin.⁶ Amoxicillin is a useful broad-spectrum drug in this context although many clinicians prefer the specific anti-anaerobic effect of metronidazole.⁷
3. Analgesics. Analgesics provide temporary relief of pain until the causative factors of infection have been brought under control. The choice of analgesic should be based on patient's suitability. Non-steroidal anti-inflammatory drugs are used in mild to moderate pain. Opioid analgesics, such as dihydrocodeine and pethidine, are used for severe pain. Paracetamol, ibuprofen and aspirin are adequate for most mild pain secondary to dental infection. Analgesics need to be given with care, especially when narcotics are used, as they carry the risk of depression of respiration.
4. Identification and elimination of the source of infection. Identification is achieved by both clinical and radiographic assessment. Elimination of the source is by root canal therapy, extraction or periradicular surgery.⁸



Fig. 7.12 Clinical picture of extraoral incision and drainage of a submandibular abscess.

Ludwig's angina

Bilateral involvement of the submandibular, submental, and sublingual spaces with a suppurating infection may lead to diffuse cellulitis classically called Ludwig's angina. This is demonstrated by induration of the swollen skin of the neck, tenderness, elevation of the floor of the mouth, trismus, and dysphagia. The tongue usually shows a varying degree of edema.

The infection of Ludwig's angina may be secondary to the spread of infection from mandibular teeth or penetrating injury to the floor of the mouth. Ludwig's angina is a life-threatening condition which demands emergency treatment.⁹ The treatment procedure consists of:

1. Protection of the airway.
2. Hospitalization and administration of intravenous antibiotics and fluids.
3. Incision and drainage extraorally of the submandibular and submental space with through-and-through drainage when necessary.
4. Relief of pain. This is achieved by incision and drainage in addition to the administration of analgesics.

Identification and treatment of the causative factor, often extraction of an infected lower tooth, is necessary after dealing with the acute condition. Emergency cricothyroidotomy or tracheostomy may be necessary to deal with respiratory obstruction. A tracheostomy may have to be made low down to avoid the infected area and may have to be carried out while the patient is awake due to a tendency to lose the airway in the supine position.

Osteomyelitis

Osteomyelitis means the spread of infection through the soft tissue component or marrow of the bone which may include periosteum, neurovascular bundles, narrow spaces of the spongy bone, and the Haversian systems of the cortical bone. It is more common in the mandible than in the maxilla due to the less profuse nature of the blood supply and the density of the cortical plates of the mandible. The most common cause of suppurative osteomyelitis is an odontogenic infection.¹⁰

The patient complains of a deep-seated pain and swelling due to the ongoing inflammatory edema and spread of infection in the bone. The associated teeth are tender to percussion and may be loose. There may be regional lymphadenitis and alteration of sensation of the lower lip due to the involvement of the inferior alveolar nerve and pressure from the inflammatory process. The acute inflammatory reaction involving fluid exudate into the medulla of the mandible causes this pressure effect on the nerve and

may also cause thrombosis of vessels leading to necrosis and sequestrum (dead bone) formation.

Depending on the signs and symptoms, osteomyelitis can be classified into acute, subacute, and chronic forms. Radiographic changes do not appear immediately in the acute suppurative form of osteomyelitis as it may take about 2 weeks for the trabecular pattern of bone to change and areas of radiolucency to start to appear, usually accompanied by a periostitis. If the acute osteomyelitis is not treated effectively this will lead to chronic suppurative osteomyelitis. The infection may be a manifestation of lowered patient resistance; this sometimes occurs in immunosuppressed patients on medication or those suffering from an impaired immune defense, as in acute leukemia, human immunodeficiency virus (HIV) infection, poorly controlled diabetes mellitus or malnutrition.

Damage to the jawbones secondary to radiation, osteosclerosis or trauma may be a predisposing cause of chronic osteomyelitis. Clinically, the disease is dominated by pain and the development of intraoral and/or extraoral sinuses. Induration of soft tissues overlying the infected segments of the jawbones is marked and the distension of the periosteum with pus or inflammatory exudate which may cause trismus and difficulty swallowing. Regional lymph nodes are usually tender and enlarged. Pathological fracture may develop if the inferior border of the mandible is damaged by the infection process. The radiographic picture of chronic osteomyelitis is loss of detail of the trabecular pattern of the osseous architecture giving the bone a mottled or moth-eaten appearance (Fig 7.13). The ischemic or necrotic islands of bone tend to sequestrate, appearing more radiopaque than the surrounding bone and these form a sequestrum of necrotic bone. In younger persons subperiosteal new bone formation appears adjacent to the diseased area. This new bone, known as involucrum, tends to be structureless or granular in appearance radiographically and may surround the necrotic sequestrum and pus lying within the bone.

Management of osteomyelitis involves:

1. Culture and sensitivity testing to provide specific antibiotic therapy which generally tends to be given intravenously and for a prolonged period (several weeks).
2. Drainage and debridement, including sequestrectomy.



Fig. 7.13 Radiograph of osteomyelitis.

3. Removal of source of infection and, if necessary, decortication of the mandible.
4. Possible resection and reconstruction of the affected bone after infection is controlled.

Chronic sclerosing non-suppurative osteomyelitis (Garré's osteomyelitis)

This is a chronic form of osteomyelitis with characteristic deposition of bone, subperiosteally and endoperiosteally, in response to an inflammatory process often initiated by a dental infection of low virulence. It affects the mandible in young individuals. Clinically, the condition has the appearance of a nodular, firm swelling related to the inferior border of the mandible. The florid proliferative periostitis is characteristic of this condition in addition to the multilayers of bone formation giving the "onion skin"-like appearance radiographically (Fig. 7.14). Computed tomography (CT) scanning may help to identify the cause of infection but normally there is no sequestration of the expanded bone.

This condition may persist over a long period of time. A corticotomy and exploration of the damaged bone may be helpful. Appropriate antimicrobial therapy is required to deal with the infection. However, relapse is common. The bone gradually remodels over 6–12 months. A focus of infection is rarely identified. The condition tends to slowly resolve as patients reach adulthood but may require lengthy steroid therapy which downregulates the inflammatory process.

Diffuse sclerosing osteomyelitis of the mandible

This is an inflammatory disease of the mandible whose cause and pathogenesis is still largely unknown. The disease can occur at any age but rarely in young children and often lasts for years with recurrent pain and swelling. The radiographic appearance is variable with areas of diffuse rarefaction and some sclerosis (Fig. 7.15). Organisms cannot usually be cultured from the lesion, although it has been claimed that with meticulous culturing techniques, organisms, including *Actinomyces* species and *Eikenella corrodens*, can be recovered.¹¹ However, many believe



Fig. 7.14 Panoramic radiograph showing subperiosteal bone formation in Garré's osteomyelitis.



Fig. 7.15 Clinical picture of bisphosphonate-related necrosis of the mandible.

that the disease may be due to an autoimmune process. Various therapies have been suggested including antibiotics, anti-inflammatories, corticosteroids, hyperbaric oxygen, and intravenous bisphosphonate therapy. Only partial success has been claimed, and surgical treatment may be necessary, consisting of either debridement of the lesion or decortication, or even resection of the mandible.¹² Recurrence often occurs, though ultimately the condition often burns itself out leaving a dense sclerotic mandible.

Osteoradionecrosis

This is a type of bone necrosis which occurs following radiotherapy to the jaw region and often becomes secondarily infected.¹³ Radiotherapy induces endarteritis obliterans which reduces vascularity and renders the bone vulnerable to infection. Once secondary infection develops it spreads through the bone but sequestration is delayed in these cases. Patients who have had radiotherapy are potentially at risk of developing this type of osteomyelitis and the mandible is particularly at risk if it has received more than 55 Gy of radiation. Extraction and other surgical procedures should be carried out as atraumatically as possible. Primary closure of the socket and pre- and postoperative antibiotic treatment, antiseptic mouthwash and good oral hygiene are essential. The use of hyperbaric oxygen to increase the blood supply of the affected bone has proven successful in the management of these cases as have other new and experimental treatments.¹⁴ Better collimation of the beam of radiation and protection of tissues adjacent to tumors have reduced, although not eliminated, this unpleasant sequel.

Osteonecrosis secondary to bisphosphonate therapy

Bisphosphonates reduce pain and bone destruction due to metastatic disease, particularly in patients with multiple myeloma, breast and prostate carcinoma. The medication inhibits bone resorption by reducing osteoclastic activity.¹⁵ Long-term administration of high-dose intravenous bisphosphonates may lead to osteonecrosis of the jawbones. This is



Fig. 7.16 Bisphosphonate-related osteonecrosis of the jaws (BRONJ), maxillary tuberosity region.

mainly due to a reduction of the vascularity, which together with inhibition of osteoclastic activity, reduces bone turnover. Both are required to protect the bone from the risk of necrosis and superadded infection. There is a lesser risk of this condition occurring in patients taking oral bisphosphonates to prevent osteoporosis.

The mandible is most often affected and the disease usually arises after dental treatment (Fig. 7.16). The patient may present with either a non-healing extraction socket or exposed bone which does not respond to conservative management and antibiotic therapy. Extraction of infected or periodontally involved teeth should be carried out before the administration of bisphosphonates, if at all possible. Surgery should be avoided whenever possible. It has been suggested that the assessment of the bone's reparative ability can be assessed by measuring the terminal polypeptide chain (CTX).¹⁶ Perioperative and postoperative antibiotics are essential for extractions. Chlorhexidine mouthrinse pre- and postextraction are also considered valuable. In non-urgent cases the risk may be reduced if the bisphosphonate is withheld for 3 months prior to surgery. This must, however, be done only in consultation with the physician prescribing the drug.

Cavernous sinus thrombosis

Thrombosis of the cavernous sinus is an extremely serious condition which may occur secondary to an acute infectious process of the dental, nasal, and orbital regions. The cavernous sinus is a fold between the inner and the outer layer of dura matter. It is lined by endothelial cells and drains venous blood from the brain. Each sinus extends from the superior orbital fissure anteriorly, through the apex of the petrous part of the temporal bone posteriorly. There are two general mechanisms by which infection may reach the cavernous sinus through the venous network of the dentofacial region. The first is the facial, or afferent system, which comprises the facial vein which communicates with the superior ophthalmic veins,

since these veins are in direct communication with the cavernous sinus through the superior orbital fissure. The second mechanism is the pterygoid venous plexus, or the efferent system. Infection may reach the pterygoid venous plexus in a retrograde fashion through the posterior facial vein or the internal maxillary vein. Infection from the pterygoid venous plexus would reach the cavernous sinus via the inferior ophthalmic vein or the veins of the foramen ovale and lacerum (Fig. 7.17).

The signs and symptoms of cavernous sinus thrombosis are those related to venous obstruction, and involvement of the nerves within the vicinity of the cavernous sinus (the oculomotor nerve, trochlear nerve, and the ophthalmic and maxillary divisions of the trigeminal nerve). Edema of the retina, eyelids and bridge of the nose, as well as ocular swelling are the main characteristics of cavernous sinus thrombosis. This is due to distension of the tissues behind the eyeball. Involvement of the cranial nerves in the septic condition causes ptosis of the upper eyelid, dilatation of the pupil of the eye, and restriction of eye movement. In addition, the patient may show symptoms of septic shock which includes pyrexia, profuse sweating, vomiting, and coma. Treatment of this condition should be prompt. The patient should be admitted to hospital, the airway should be maintained, and fluids should be administered. Antibiotics should be administered immediately and adequately. Anticoagulant therapies are used to prevent extension of the thrombus and interference with the venous drainage of the brain. Drainage of pus should be carried out as soon as possible. Permanent cranial nerve damage, brain damage, and death have been reported following a septic cavernous sinus thrombosis of dental origin.

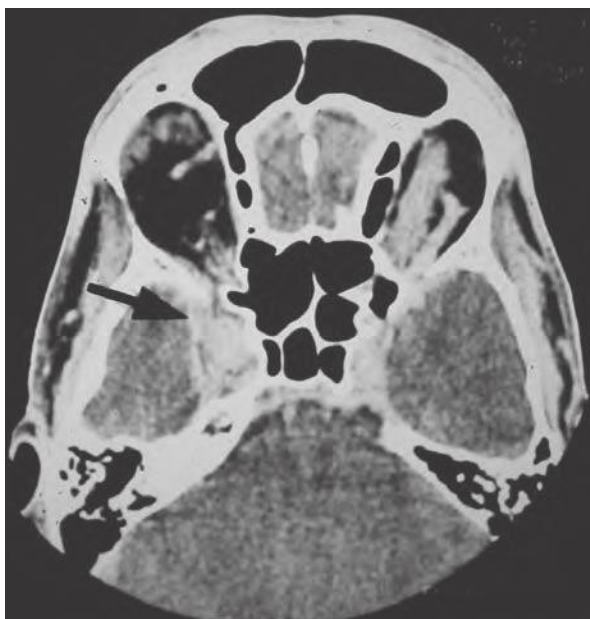


Fig. 7.17 Axial CT scan showing cavernous sinus thrombosis (arrowed).

Necrotizing fasciitis

This is a rapid spreading infection of the skin causing necrosis of the subcutaneous tissue.¹⁷ A combination of aerobic and anaerobic microorganisms is responsible for this condition which occurs most often in immunocompromised patients. A dental source of infection is rare but possible (Fig. 7.18).

Clinically, there is loss of skin due to the necrosis of the underlying subcutaneous tissue. Other areas of the skin may appear erythematous, edematous, rapidly changing to purplish and black in color. The patient requires aggressive and immediate surgical intervention.¹⁸ The necrotic tissue requires wide excision to healthy margins. Intravenous fluid support is given together with a combination of clindamycin, penicillin, and metronidazole until alternative therapy is chosen following culture and sensitivity. Death has been reported from this condition.

Actinomycosis

This is a chronic suppurative granulomatous infective process which is characterized by the development of swelling in the region of the face and neck. It is normally a soft tissue infection but can occasionally involve bone. The causative microorganism is *Actinomyces israelii* which is present in the normal oral flora.

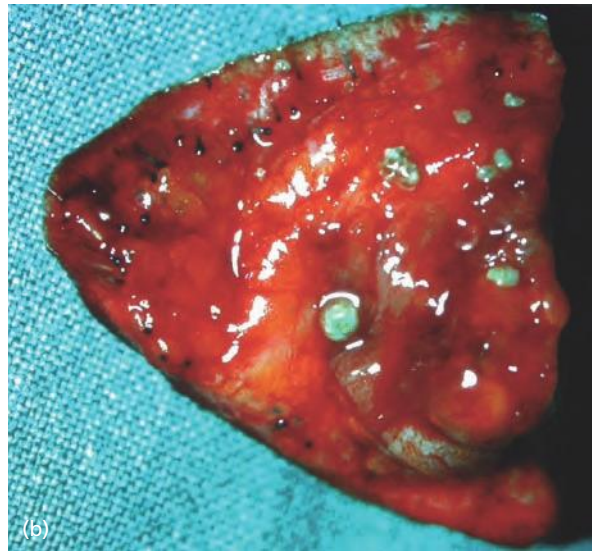
Damage to the tissue, resulting from either lower tooth extractions or jaw fractures, creates a condition of low oxygen tension in which the organism becomes invasive. The condition starts as a swelling, which may occur up to several weeks after the trauma, usually within the submandibular region. The swelling appears first as a firm and indurated lesion and the overlying skin is usually inflamed, firm but may have a bluish color. Within the swelling multiple abscesses may form with sinuses draining fluid containing yellow granules (sulfur granules) which appear microscopically as a mass of Gram-positive mycelia and polymorphs (Fig. 7.19). Radiographic examination may reveal little destruction of affected bone as the



Fig. 7.18 Necrotizing fasciitis of the submandibular region.



Fig. 7.19 Mandibular actinomycosis showing (a) a raised firm and inflamed swelling and (b) sulfur granules in the resected specimen.



infection is essentially one of the soft tissue. Penicillin is the drug of choice in addition to adequate incision and drainage. The organism is penicillin-sensitive but it takes time for the antibiotic to penetrate the granulomatous reaction of the body. Antibiotic treatment must be continued for at least 6 weeks. Surgical removal of any infection will facilitate the recovery.

***Mycobacterium* infection of the oral mucosa and jawbones**

This is a specific granulomatous condition which is caused by *Mycobacterium tuberculosis*. This condition is rare but tends to be more common in developing countries. The tongue is the organ most commonly affected by this condition (Fig. 7.20). The lesion may be primary (often from infected milk) usually presenting as enlarged nodes in the neck, or can be secondary to pulmonary tuberculosis (when the causative organism is coughed up in infected sputum). *Mycobacterium tuberculosis* is present in the sputum and gains access to the deeper parts of the oral mucosa as the result of minor trauma following dental extractions or due to abrasion of the mucous mem-

brane. The invading microorganism may also gain access to the jawbones and cause a tuberculosis osteomyelitis. Clinically, the affected lesion will be a swelling with multiple draining sinuses. Looseness of the teeth and sequestration of the affected bone may be clinically apparent. With new subperiosteal bone formation, an involucrum is rarely observed. Enlargement of the submandibular and cervical lymph nodes (a condition known as scrofula), which are rubbery in consistency, with erythematous overlying skin, is a characteristic of infection with *Mycobacterium bovis*; in time an extraoral sinus develops.

Histological examination shows caseation (a “cheesy” form of necrosis) in the centre of the granuloma. The presence of tuberculous bacilli in the specimen, appearance of the primary focus in the chest radiograph, and a biopsy of the lesion will confirm the diagnosis. Management of this case will include medical treatment and surgical intervention. Isonicotinic acid hydrazide (INH), para-aminosalicylic acid (PAS), and rifampicin are still the drugs of choice. Incision and drainage of the localized abscess and sequestrectomy of dead bone are indicated. In addition, extraction of the affected teeth and



Fig. 7.20 Tuberculosis of the tongue. The lesion in this case is secondary to pulmonary TB and coughing up infected sputum.

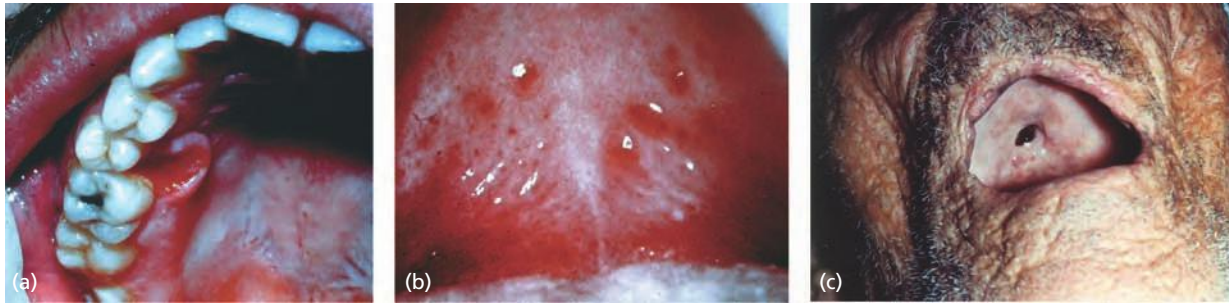


Fig. 7.21 Photos of (a) primary chancre, (b) secondary snail track ulcers of the soft palate, and (c) tertiary gumma of the palate. The chancre and snail track ulcers are highly infectious.

the surgical excision of the involved cervical lymph nodes may be recommended.

Syphilis

This is a chronic infectious disease which is caused by the spirochete *Treponema pallidum*. Though now rare, primary (the chancre), secondary (skin rashes, lymphadenopathy, mucous patches, and snail track ulcers), and tertiary (gumma or syphilitic leukoplakia) may be found in the oral cavity (Fig. 7.21). The first and second stages are highly infectious. Bony changes may occur in the tertiary stage of syphilis. The periosteum is a common site for the development of gumma, with the midline of the palate being classically involved leading in time to oronasal fistula. This appears radiographically as peeling of the periosteum away from the underlying bone and the formation of sclerotic bony margins at the periphery. Gumma may extend to the underlying bone and cause syphilitic osteomyelitis. The condition is diagnosed by the identification of the *Treponema pallidum* using dark field microscopy, serological tests, and biopsy of the granulomatous tissue. Long-term penicillin is the drug of choice in addition to local measures to deal with damaged soft tissue, sequestered bone, and involved teeth. The fourth stage of syphilis is rare; it affects the cardiovascular system causing aortic aneurysms or aortic valve incompetence. The central nervous system may be involved which may lead to dementia or spinal cord disease.

Congenital syphilis is rare because treatment during pregnancy eradicates the infection. The characteristic features of an infected baby include saddle-nose deformity, tapering widely separated incisors (Hutchinson's teeth), domed-shaped molars (Moon's or mulberry molars), and papules at the angle of the mouth which heal forming radiating scars (rhagades).

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Chapter 8

Armamentarium for Basic Procedures

Ala Al-Musawi

This chapter will deal with the basic instrumentation required for both soft tissue and hard tissue oral and maxillofacial surgery procedures. As well as basic instrumentation, it will include dental forceps and dental elevators, the rationale and choice of suture materials, and current instrument sterilization techniques. It will also discuss new innovations in surgical instruments.

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Basic instrumentation for soft tissue procedures

Basic soft tissue instrumentation consists of a scalpel for making incisions (Fig. 8.1), toothed and non-toothed forceps (Figs 8.2–8.4) for grasping tissues and arresting bleeding vessels, Allis-type clamps (Fig. 8.5) for appropriately holding biopsy specimens and tissue margins, retractors (Figs 8.6, 8.7), as well as a range of forceps ranging from smaller mosquito-type forceps to larger Kelly and Schmidt-type forceps (Fig. 8.8), needle-holding forceps (Fig. 8.9), and appropriate scissors (Figs 8.10–8.12). All of these instruments can be combined to make an appropriate soft tissue instrument tray.



Fig. 8.1 Scalpel.



Fig. 8.2 Toothed forceps.



Fig. 8.3 Non-toothed forceps.



Fig. 8.4 Toothed forceps.



Fig. 8.5 Allis clamp.



Fig. 8.6 Minnesota retractor.



Fig. 8.7 Weider retractor.



Fig. 8.8 Mosquito and Kelly-type forceps.



Fig. 8.9 Needle holders.



Fig. 8.10 Dean scissors.



Fig. 8.11 Iris scissors.



Fig. 8.12 Metzenbaum scissors.

Basic hard tissue instrumentation

For surgery involving the mandible or maxilla, the basic soft tissue instrumentation is required, and additionally, one requires periosteal elevators (Figs 8.13, 8.14) to raise periosteum from bone, periosteal retractors (Figs 8.6, 8.15), curettes (Fig. 8.16) and rongeurs (Fig. 8.17) for removing and scraping bone, and bone files (Fig. 8.18) for smoothing the bone.



Fig. 8.13 Woodson periosteal elevator.



Fig. 8.14 Molt #9 periosteal elevator.



Fig. 8.15 Langenbeck retractor.



Fig. 8.16 Curettes.



Fig. 8.17 Rongeurs.



Fig. 8.18 Bone files.

Drills

An appropriate drill for removing bone (Fig. 8.19) will be either gas-powered or electric-powered and will not exhaust gas or air through the tip of the handpiece, since this gas can force debris and bacteria into the bone and can even cause an air embolus. Any gas used to power the drill should exhaust back along the tubing into the operatory or into a scavenging device. For preference, drills should have an automatic irrigation system built into them which is linked to the drill control and can be used in conjunction with a sterile irrigant solution. Burs should be appropriate bone-cutting burs rather than burs intended to cut enamel and dentin.



Fig. 8.19 Surgical drill.

Dental elevators

Elevators are often used in conjunction with dental forceps to remove teeth and roots.¹⁻⁴ The general principle in the use of the elevators is that the surrounding alveolar bone is used as a fulcrum for the action of the elevator rather than the adjacent tooth, unless it is intended to also remove the adjacent tooth. If the alveolar bone is used as a fulcrum and it is crushed by the elevator, this bone should be removed with a curette or bone file. Elevators that have a right-angle point of elevation can exert significantly higher forces on the mandible than those with an in-line direction of elevation. Care should be taken with such instruments (e.g. Cryer's elevators) since fracture of the mandible is possible if these instruments are used inappropriately. Similarly, instruments with a so-called T-bar handle can exert even more force on the mandible, and particular care should be used with these instruments (e.g. Winter's elevators). Examples of dental elevators are illustrated in Figs 8.20–8.27.



Fig. 8.20 Straight elevator.



Fig. 8.21 Straight elevator.



Fig. 8.22 Coupland's chisel.



Fig. 8.23 Cryer's elevators.



Fig. 8.24 Potts elevators.



Fig. 8.25 Crane's elevators.



Fig. 8.26 Root pick elevators.



Fig. 8.27 Warwick-James elevator.

Dental forceps

Dental forceps have remained largely unchanged since they were introduced by Charles Tomes in the 1800s. Prior to this, instruments such as the dental key and pelican were employed. Dental forceps essentially come in two types. The first is the in-line forceps where the handle of the forceps is in line with the dental arch. To a certain extent, these forceps are safer but rely on the inherent strength in the surgeon's wrist. A particular example of an in-line forceps is the cowhorn forceps which is meant to be placed between the bifurcation of the roots of a lower molar and will either remove the molar intact or will split the molar through the bifurcation enabling both sections to be removed separately.

The second type of forceps is the so-called Ash forceps (because they were marketed through Claudius Ash and Sons, dental supply house), and these are forceps that have a handle that comes out at right angles to the arch. Potentially, these forceps can create much greater force on the tooth, through the principle of levers. It is possible to fracture the mandible with these instruments, so care must be taken. There are different Ash forceps for lower molars, premolars, and incisors. Many dentists, however, prefer to use premolar forceps for the molars, grasping the mesial root only so that either the whole tooth will be removed, or if it splits, the mesial root will be removed making the distal root easier to remove on its own. Although Ash-type forceps are sometimes used for maxillary teeth, they are usually reserved for mandibular teeth, and maxillary teeth are removed with in-line forceps. These can be specifically for the left and right maxillary molars and then separate ones for the premolars and straight forceps for the upper canines and incisors. Illustrations of appropriate instrumentation are shown in Figs 8.28–8.34.



Fig. 8.28 Upper right/left molar forceps: 53 R/L.



Fig. 8.29 Upper premolar forceps: 150.



Fig. 8.30 Upper incisor and canine forceps.



Fig. 8.31 Lower premolar in-line forceps: 151.



Fig. 8.32 Cowhorn forceps.

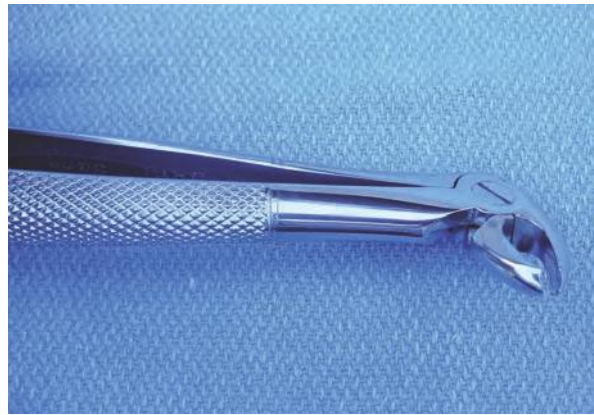


Fig. 8.33 Ash lower molar forceps.



Fig. 8.34 Ash lower premolar incisor and canine forceps.

Ancillary instrumentation

In addition to the instrumentation already shown, additional instrumentation required for most surgical procedures includes:

1. Suction, which is normally electric or vacuum driven, and suitable suction tips that work well intraorally are illustrated in Figs 8.35 and 8.36.
2. Cautery, diathermy, or electrosurgical instrumentation is often required for surgical procedures for both cutting and coagulation. There are technical differences between these systems. In a monopolar diathermy system (Fig. 8.37) there is a small active electrode and a large indifferent electrode, which takes the form of plate in contact with the body. The small active electrode is the diathermy tip but its effect is not localized as it spreads towards the indifferent electrode. By altering the waveform the tip can predominately cut or coagulate. In a bipo-



Fig. 8.35 Yankauer suction tip.



Fig. 8.36 Surgical suction tip.

lar system (Fig. 8.38) both tips of a pair of forceps are insulated from each other and form the two electrodes. The current and tissue damage is localized to the tissue between the forceps tips and does not spread and there is no large indifferent electrode in another part of the body. Because of this there is no current passing through the rest of the body so it is safe for patients with pacemakers etc. Desiccation is a cauterizing process whereby a single wire has an area of high resistance so it can become hot at that point and cauterize. Desiccation instruments tend to be of lower power and energy levels and are often disposable (Fig. 8.39).

3. Bone wax (Fig. 8.40) is also often used when bleeding is encountered during intraosseous surgery. It can be squeezed into the cancellous spaces of the bone and will cause hemostasis. Bone wax is ilable which are resorbable. In a dental socket where bleeding is a problem, one normally uses a material such as Surgicel® (Fig. 8.41) or Gelfoam® (Fig. 8.42).



Fig. 8.37 The active electrode (tip) and indifferent electrode (plate) in a monopolar diathermy system.

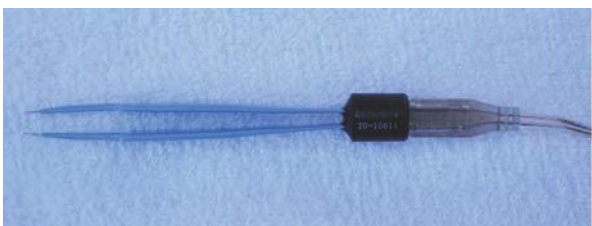


Fig. 8.38 The insulated tips of a bipolar diathermy system. Each tip is a separate electrode.



Fig. 8.39 A disposable low-power cautery unit.

Sterilization of instruments

Many instruments used today are disposable single-use instruments, and no attempt should ever be made to reuse these instruments. In theory, the more single-use instruments are used, the better the chain of sterility.



Fig. 8.40 Bone wax.



Fig. 8.41 Surgicel®.



Fig. 8.42 Gelfoam®.



Fig. 8.43 Steam autoclave.

For instruments that are not single use and must be reused, they must be sterilized between surgical procedures. This involves removal of all organic and inorganic debris from the instrument, and this can be done manually but is better performed by ultrasonic cleaning. Following ultrasonic cleaning, the instruments should be packaged in appropriate bags and sterilized. For most instruments, autoclaving to a temperature of 134°C for 3 minutes, or 121°C for 15 minutes will sterilize instruments of all known bacteria and viruses (Fig. 8.43). To be effective, however, the steam must penetrate through to the instruments, and the package must not be blocked by other packages that prevent the full effects of the steam to be effective.

For more delicate instruments, ethylene oxide can be used for sterilization, but this is not universally available and usually takes up to 24 hours for instruments to be available following ethylene oxide sterilization, since time must be allowed for the ethylene oxide to disperse following appropriate sterilization.

Radiation is used to sterilize some instruments, but more often, it is used to sterilize alloplastic grafts including bone grafts. Bone grafts sterilized by radiation may maintain more activity than those sterilized by ethylene oxide.

Lower-temperature autoclaves that are combined with formaldehyde are available and do ensure sterilization at a lower temperature for some instruments.

There is no known reliable method of removing or sterilizing prions from surgical instruments, and in the present state of knowledge, if one is operating on a patient with known or suspected prion disease, all instruments must be appropriately disposed of following the procedure, and no effort must be made to reuse the instruments. Most authorities have regulations in place for how and where to perform surgery in a patient with suspected prion disease.

Suture materials and needles

Suture materials can be classified in a number of different ways (Fig. 8.44).

Resorbable and non-resorbable

Resorbable sutures include catgut in its various forms, polyglycolic acid (Dexon®), polyglycolide-lactide



Fig. 8.44 Sutures: fast resorbing gut; regular gut; chromic gut; polyglycolic acid (Dexon®) in different forms; polyglycolide-lactide (Vicryl®) in different forms; polyglactin irradiated (Vicryl Rapide®); polyglactin solid (Monocryl®); polydioxanone (PDS®) in different forms; polyglyconate (Maxon®) in different forms; silk; nylon; polypropylene (Prolene®).

(Vicryl®), polydioxanone (PDS®), and polyglyconate (Maxon®). Resorbable sutures are essentially classified as fast resorbing, medium resorbing, and long resorbing. The fast-resorbing sutures include catgut where fast-resorbing gut resorbs in 2–3 days, regular catgut resorbs in 5–7 days, and chromic catgut resorbs in 10–14 days. Intermediate-resorbing materials include polyglycolic acid and polyglycolide-lactide, whose resorption time is 4–6 weeks when used intraorally. The solid form of polyglycolic acid, which is marketed as Monocryl, also resorbs in 4–6 weeks. By irradiating polyglycolic acid, a faster-resorbing version is available which resorbs in 14–21 days and is marketed as Vicryl Rapide®. Long-resorbing sutures include polydioxanone and polyglyconate. Intraorally, these resorb in around 120 days, although they start to lose their strength after about 40 days. Non-resorbable sutures include silk, nylon, polypropylene (Prolene®), cotton, and Gore-Tex®.

Braided and solid

Braided sutures consist of multiple filaments woven together. The advantage of a braided suture is that it is easy to handle and easy to knot, but disadvantages include the fact that they tend to leave more prominent stitch marks on the skin and can lead to “wick-ing” where fluids (possibly containing bacteria) can be attracted to the suture by capillary action. A solid suture consists of a solid tube of the suture material which means it will have memory and be harder to handle and harder to knot, but will leave less prominent stitch marks and will not have a tendency to “wick”. Braided sutures include silk, cotton, and Gore-Tex® as well as the synthetic materials polyglycolic acid, polyglycolide-lactide, polydioxanone, and polyglyconate. Solid sutures include catgut, nylon, polypropylene, and the synthetic materials polyglycolic acid, polyglycolide-lactide, and polydioxanone and polyglyconate, which can be manufactured in either variety.

Naturally occurring materials or synthetic

Naturally occurring materials include silk and cotton as well as catgut. Since catgut is a biological product derived from the intestines of sheep, concern has been raised about the possibility of transmission of prion disease via catgut sutures, and at the present time, this remains theoretical but debatable. Catgut sutures normally resorb in 5–7 days, but by treating the catgut with chromic acid (chromic gut), its handling properties can be improved, its tissue irritability can be decreased, and its resorption time increased to 10–14 days. If, however, catgut is irradiated, it will resorb more rapidly and is commercially available as “fast-resorbing gut”. This will resorb in 2–3 days and is useful for sutures around the eyelids. Catgut is resorbed by enzymatic action, and this will cause some tissue irritation, which is often seen around catgut sutures.

Synthetic sutures are usually resorbed by oxidation or hydrolysis, causing less tissue reaction. Examples of synthetic resorbable materials include polyglycolic acid, polyglycolide–lactide, polydioxanone, and polyglyconate. These synthetic materials are generally produced by extrusion and, therefore, can be made in many different configurations. They can usually be manufactured in almost any color, and even in a candy stripe design, and many manufacturers do offer a choice of colors. They can be manufactured as either braided or solid configurations, and also as a combination whereby a braided suture is then coated with a thin layer of the liquid material; this produces essentially a braided suture with a smooth outer layer, which has the handling advantages of a braided material with the lack of irritability of a solid material and also the lack of ability of the tendency to “wick”. These are properties that cannot be matched by natural materials. However, the synthetic materials do tend to be more expensive than natural materials.

Uses for the various suture materials

In general, one likes to use a resorbable material in the mouth so that sutures do not need to be removed. Since only single-layer suturing is normally appro-

priate intraorally, this is normally with a 3-0 or 4-0 thickness material, and plain catgut, chromic catgut, polyglycolic acid or polyglycolide–lactide are normally appropriate. Extraorally, tissues are often closed in layers, and as such, the appropriate material determines the layer. Fascia can normally be repaired with an intermediate resorption time material such as polyglycolic acid or polyglycolide–lactide, but chromic catgut may also be appropriate. Muscle layer requires a material that will retain its strength for 2–3 weeks, and again polyglycolic acid or polyglycolide–lactide are appropriate. For the subcutaneous tissues, a suture is required which will maintain its strength for around 14 days, and again chromic catgut, polyglycolic acid or polyglycolide–lactide are appropriate. For the skin, a 5-0 or 6-0 nylon is appropriate, or a running subcuticular suture which is normally carried out with a longer-term resorbable solid suture is appropriate. Monocryl, polydioxanone, or polyglyconate would be appropriate for this purpose.

Suture needles

Most suture materials come ready swaged to the needle. Although needles with an eye are still manufactured, and clinicians can thread their own needles, less tissue damage is caused by a ready swaged suture. Needles come in different varieties and combinations. For oral surgical use, a needle which is part of the circumference of a circle is normally used, and straight or J-shaped needles have little application. For personal preference, anything between three eighths and five eighths of a circle are appropriate, and for the many procedures in the posterior part of the mouth, a one half round or five eighths round needle is felt to be more appropriate. For extraoral suturing or suturing towards the anterior part of the mouth, a three eighths needle may be more appropriate.

Needles can be round bodied to suture delicate tissues since there is less chance of the suture cutting through the tissues, but for many oral tissues, a cutting needle is appropriate. Cutting needles are triangular in cross-section and can be forward cutting or reverse cutting depending on the way they are made (Fig. 8.45). For intraoral use, a reverse-cutting needle is often appropriate, since a forward-cutting needle

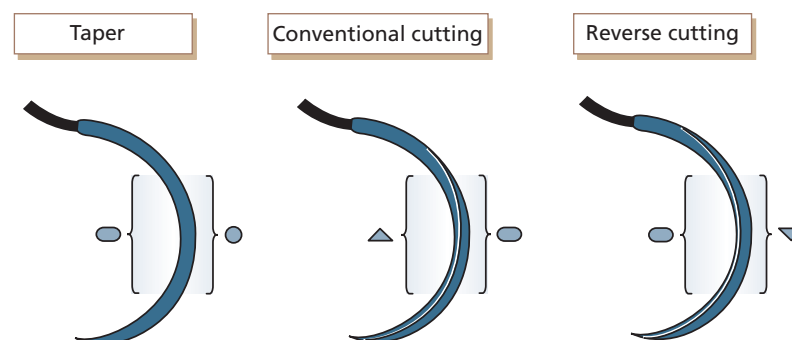


Fig. 8.45 Needle patterns. Round-bodied needle; cutting needle; reverse-cutting needle.

has a tendency to cut through the tissues as you pull it through, sometimes pulling the suture out. Combination needles are available, such as a taper cut needle which is a cutting needle for the first one third of the needle and then becomes round bodied so that as you pull it through the tissues, it does not cause further damage.



Fig. 8.46 Piezoelectric bone-removal system.

New innovations in surgical instrumentation

Although over the years many different systems have been suggested for removing bone, none has superseded the dental drill. More recently, a piezo instrument using high-frequency vibration to remove bone has been advocated (Figs 8.46, 8.47).⁵ Initially, these instruments lacked power and could only remove small areas of relatively thin bone. However, new generations of these instruments are expected to have greater power and more flexibility to change the frequency and wavelength of the vibrations to achieve a more efficient cutting action. It is claimed that the heat generated and peripheral damage produced are much less than with a dental drill, and, therefore, with further improvements, they may have a place in bone removal in oral and maxillofacial surgery.



Fig. 8.47 Piezoelectric bone-removal system.

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Chapter 9

Basic Surgical Principles

M. Anthony Pogrel and Fabio Kricheldorf

This chapter covers the basic principles of aseptic technique, cutaneous and intraoral incisions, the principles of soft tissue biopsy techniques, and the principles of suturing of wounds and lacerations.

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Aseptic technique

Since the late 1800s, when bacteria were first recognized, the concept in surgery has turned from accepting pathogenic agents, and hoping that the body can overcome them, to attempting to eliminate them from the surgical field. In hospitals, most operations are carried out using a truly aseptic technique. All instruments used are sterile, having been autoclaved or all organisms eliminated by some other physiochemical means. In addition, the operating room itself is surgically clean, and all trays, etc., on which instruments rest are sterile. The surgeon wears sterile gloves and a sterile gown, and a mask, head covering, and shoe covers or separate footwear that is only worn in the operating room. An aseptic technique is employed whereby sterile and non-sterile objects do not contact each other so that there is a continuous chain of sterility; the only organisms that come in contact with the patient are their own endogenous organisms. In this way, infections should not occur, and, in theory at least, antibiotics and other chemotherapeutic agents should be unnecessary if no exogenous infecting agents are introduced. In practice this type of sterility, although ideal, is imperfect. Infections do still occur in the operating room for a variety of reasons and

antibiotics do still need to be employed in many cases.

Questions have been raised over the years as to the necessity of wearing caps, masks, and shoe coverings.^{1,2} Studies have even shown that in longer procedures masks often become reservoirs of the surgeon's saliva and nasal secretions, and organisms may be demonstrated to fall from the mask to the patient. It has been suggested that for longer procedures, not wearing a mask, but also not talking, may disseminate fewer organisms.

Nevertheless, the rules for carrying out invasive surgery in the operating room environment of a hospital are fairly standard and clear cut. The rationale in a dentist's, or oral surgeon's office, is, however, less clear cut. For many years, dentistry and relatively minor oral surgical procedures were carried out in an outpatient office without the use of gloves, cap, or mask and with only a clean gown or coat. This changed in the 1980s when new generations of pathogenic organisms were identified including hepatitis B and hepatitis C viruses, and human immunodeficiency virus (HIV). Following this, to protect both the dentist and the patient, dentists started to wear gloves, caps, and masks.

At the present time, there is no standard for the level of asepsis to be practiced in a dental office or for oral surgery performed in an outpatient setting. In all cases, dentists will now wear caps, masks, and gloves, but in most cases they will continue to wear their outside shoes. Gowns are often worn but are often not sterile but rather cleaned and laundered and changed between patients. Gloves are normally sterile for invasive procedures but are generally non-sterile for non-invasive procedures. Aseptic technique is not strictly followed in most cases, and, although all instruments are sterile and are normally placed on a sterile surface, they may on occasion come in contact with non-sterile instruments and techniques. For this reason, the chain of sterility is less secure in an office environment, and thus antibiotics tend to be used more frequently, and often prophylactically.

Principles of surgical incisions

Surgical incisions are used to gain access to the area, either intraorally or extraorally, that is the object of the surgery. Other instruments such as retractors pull the tissues aside in order to visualize the tissues exposed. In general terms, incisions should be no longer than is minimally necessary, and the skin or mucosal incision should be the shortest incision with incisions in the deeper layers being longer. In this way, the skin or mucosal surface can be slid from side to side to gain maximal access to the deeper layers without the necessity of increasing the length of the surface incision.

Incisions are most often made with a surgical scalpel, and for oral and maxillofacial surgery, the #15 blade with its rounded tip is still the most popular, although the #11 blade with its pointed tip and the #12C blade with its smaller rounded tip are popular for some procedures with some surgeons. Incisions should be made with one single firm movement using the palm of the hand as a support for the scalpel handle to avoid undesirable instability.

Incisions can also be made with electrosurgery or a laser, or even combined instruments such as a scalpel blade with electrosurgery capacity built into it, although these latter instruments are often expensive, somewhat unreliable, and not widely used. Electrosurgical cutting, although it produces a relatively bloodless field, does so at the expense of surface cauterization and does produce more wound breakdown, scarring and wound contracture, and so is generally not employed in esthetic areas.

Skin incisions around the face are best sited either in established skin creases, the site of future skin creases (in young patients), or in the relaxed skin tension lines (RSTL) (Fig. 9.1).³ These lines generally run at right angles to the direction of the underlying musculature and are roughly equivalent to the original lines described by Langer in 1861,^{4,5} which he produced by puncturing the skin of fresh cadavers with

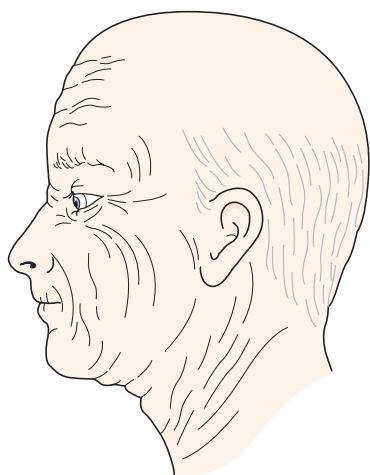


Fig. 9.1 The relaxed skin tension lines of the face which are the sites of choice for surgical incisions. In general, they run at right angles to the underlying muscles.

a dagger. He then observed how much the incision gaped compared with the site of the wound. From this he drew lines over the face where wounds do not tend to gape. These lines really represent a function of skin extensibility and reflect the way the skin was stretched by the dagger as it entered the tissues. Although there are differences between these and the relaxed skin tension lines, or the so-called lines of minimal tension of Converse,⁶ they all follow the same general principles. Differences do occur on the face since the original Langer's lines sometimes cross the natural crease lines at right angles. If incisions are placed in this way, they will produce minimal scarring and the best esthetic results. One also needs to be cognizant of any underlying nerves, particularly the branches of the facial nerve. Incisions made on the oral mucosa are generally full thickness over the mandible and maxilla and go down to bone.

Types of intraoral incisions

1. The straight vertical incision is shown in Fig. 9.2, and produces the most esthetic result with minimal scarring. It is indicated for obtaining access to deeper lesions, for tunneling procedures, and for many minimally invasive intraoral procedures. Access, however, is limited.
2. The straight horizontal incision in the buccal sulcus as shown in Fig. 9.3 is often indicated for management of periapical pathology, impacted teeth, tumors, and sinus procedures. It does, however, tend to produce more scarring than a vertical incision.
3. The intrapapillary incision, sulcular incision, or gingival margin incision is carried out using a scalpel at a reverse bevel and sectioning the interdental papillae and some of the supracrestal and transeptal fibers of the periodontal ligament. It is shown in Fig. 9.4 and gives excellent access with minimal scarring and can be used buccally, palatally, and lingually. However, there can be problems around crowns and bridges and it can cause gingival recession, root exposure, and occasionally subsequent gingival problems.

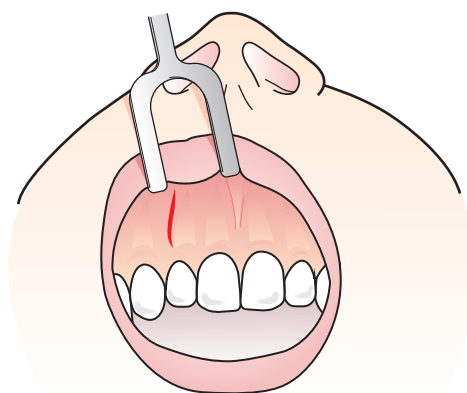


Fig. 9.2 A straight vertical incision intraorally for access to a variety of soft tissue and bone lesions.

4. The gingival margin incision can be combined with a releasing incision at an angle of about 70° to allow improved access to a bony lesion but maintain adequate vascularization of the flap (Fig. 9.5). This is a popular incision, but one disadvantage is that if the releasing incision is made anteriorly, the appearance of the papilla that is included in the releasing incision may become atypical, and the vertical scar produced may be visible on people with smile lines in the esthetic zone which expose the gingiva.
5. A releasing incision can be made at each end of a gingival margin incision. The releasing incision should be divergent to protect the vascularity of the flap, particularly in its distal regions, and the further back the releasing incisions are made in the

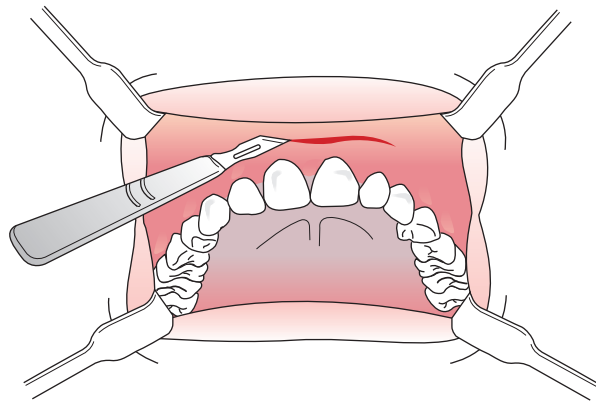


Fig. 9.3 A horizontal sulcular incision suitable for access to periapical pathology and sinus procedures.

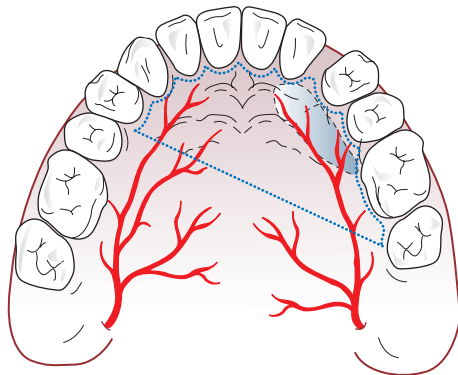


Fig. 9.4 An intrapapillary or sulcular incision used to give access to the palatal surface of the alveolus.

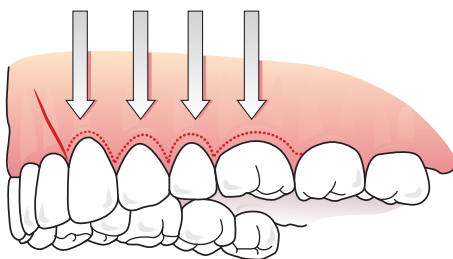


Fig. 9.5 A gingival margin incision incorporating a releasing incision which is wider at its base than its apex to protect the vascularity of the flap (arrows). If the releasing incision is in the esthetic zone of the mouth, the esthetics may be compromised.

oral cavity, the less they will be visible when the patient smiles (Fig. 9.6). It is a general rule of all soft tissue flaps that the base should be wider than the apex to protect the vascularity.

6. Incisions for third molars are generally a Winter-type incision, which comes down the external oblique ridge to the disto-buccal line angle of the second molar and then continues around the gingival margin of the first and second molar, occasionally with a releasing incision anteriorly if necessary (Fig. 9.7). This gives good access but it can be difficult to suture the papillae and can cause gingival recession on occasions. An alternative incision for third molars runs down the external oblique ridge to the disto-buccal line angle of the second molar and then proceeds as a releasing incision down into the sulcus terminating around the posterior edge of the first molar so as not to encounter a small arteriole which is often present opposite the first molar (Fig. 9.8).

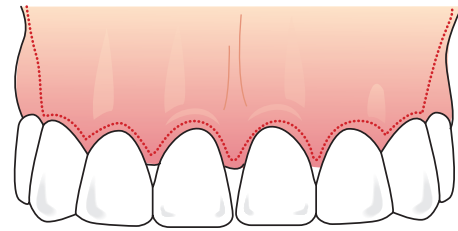


Fig. 9.6 A releasing incision at each end of a gingival margin incision gives excellent access with the base wider than the apex to protect the vascularity.

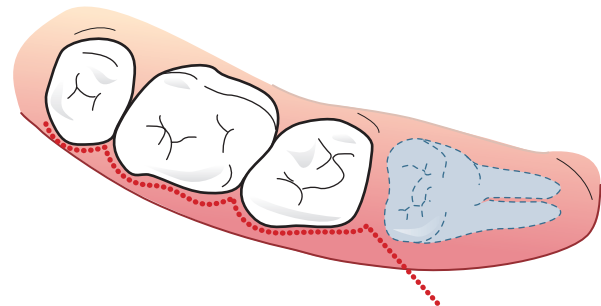


Fig. 9.7 The Winter-type incision for access to a third molar. An anterior releasing incision can be made if necessary.

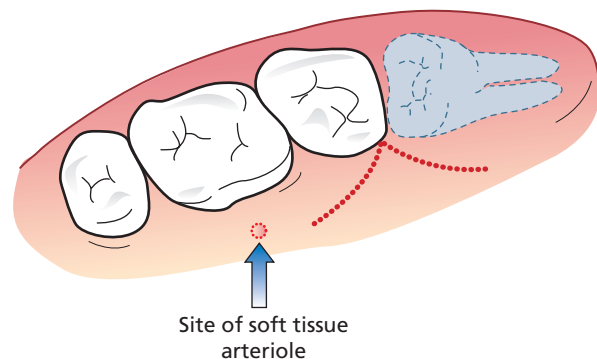


Fig. 9.8 An alternative third molar incision which does not involve access to the gingival sulcus of adjacent teeth.

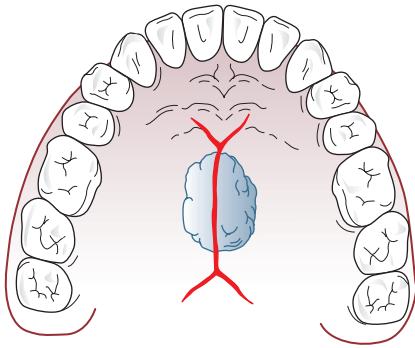


Fig. 9.9 The double Y-type incision which gives excellent access for a palatal torus or similar lesion.

7. For lesions such as a palatal torus, a double Y-type incision down to bone is often indicated. This incision leaves a good blood supply for the palatal mucosa and allows excellent access to the torus which can then be removed with a combination of drills and chisels (Fig. 9.9).

Following the initial incision, if the deeper soft tissues are to be incised, this can be done with either a sharp or a blunt technique. A sharp technique will employ a scalpel or dissecting scissors and involves a thorough knowledge of the anatomy of the area to avoid damaging important structures. In some cases, a #15 scalpel blade laid on its side and used in a scraping motion can be a compromise method of quickly going through tissue layers but leaving adequate opportunity to visualize important structures. With a blunt technique, a pair of mosquito, or similar, forceps is placed in the tissue layer closed, is then opened, and then withdrawn and closed outside the body. In this way, an incision is gradually widened (with sharp dissection or excision of structures where necessary) without damaging or crushing structures. The mosquito forceps should not be closed within a cavity since important structures may be trapped between the blades.

When access is being gained to the bone of the mandible or maxilla, periosteal elevators will need to be employed to gain access to the bone itself. Periosteum should be retracted with care with a periosteal elevator, bearing in mind that as the subject ages a greater proportion of the blood supply to the bone comes from the periosteum, particularly in the mandible, and, therefore, it should be preserved wherever possible.

Principles of soft tissue biopsy

A biopsy entails obtaining material suitable for establishing a tissue diagnosis. In principle, a biopsy can be either incisional, where a representative portion of a lesion is removed for examination, or it can be excisional, where the whole lesion is removed.

Biopsy can be performed with a scalpel, electro-surgery, laser (Fig. 9.10), tissue punch (Fig. 9.11), or a combination. A scalpel is most frequently employed, and once a portion of the lesion has been excised, care must be taken when handling the tissue so as not to crush or distort the tissue. This means that forceps must be used judiciously in securing the specimen. The use of a scalpel, however, does enable the soft tissue to be sutured back with minimal distortion. If a tissue punch is used, there is usually less distortion of the specimen, but since the punch is circular, a round defect is produced which is more difficult to close and often has to be left open to granulate. Nevertheless, a tissue punch, either 3 mm or 5 mm in diameter, is an excellent way to obtain a palatal biopsy since the punch can be taken down to palatal bone with ease to obtain a full-thickness specimen.⁷ In most cases, the punch is used to sever the lesion laterally from the surrounding tissue and a pair of forceps is then used



Fig. 9.10 A surgical carbon dioxide laser which can be used for soft tissue surgery, but in practice has limited use and has not replaced the surgical scalpel. Courtesy of Luxar.



Fig. 9.11 A dermatological tissue punch which can be very useful for obtaining deep biopsy specimens, particularly from the palate where the punch can be inserted down to palatal bone.

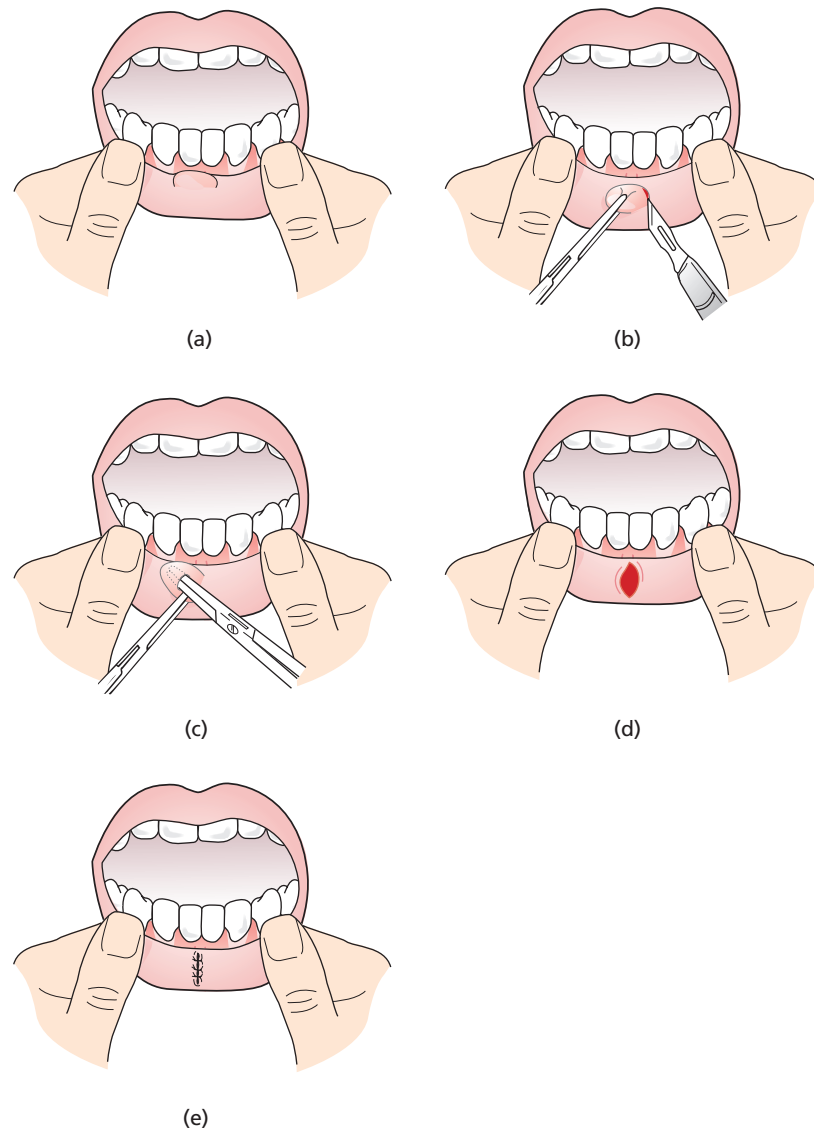


Fig. 9.12 The longitudinal incision for removal of a lip lesion.

to retract on the specimen which can be dissected from the underlying bed with scissors or periosteal elevator as appropriate. Although electrosurgery or laser can be used to obtain a biopsy specimen for pathological examination, this is generally not preferred since the electrosurgery or laser will both cause coagulation or vaporization damage to the bed of the tissue which makes examination of the margins more difficult and problematic.

Biopsy of the mucosal surface of the lip

Excisional biopsy of lesions such as irritation fibromas, mucus retention cysts, and other minor pathology of the lips is common. Although these are often viewed as very straightforward lesions to remove, there is morbidity associated with them since the lips are such sensitive and minutely controlled organs. Most of the lesions occur on the lower lip since it is

much more often traumatized than the upper lip. If the lesion is removed via a longitudinal incision, this produces the least scarring but can interfere with the small branches of either the facial nerve or the mental nerve in the lips. It is sometimes surprising how often excision of a small mucocele can leave the patient with either a slight weakness of part of that area of the lower lip or a small area of paresthesia due to interference with minor nerve branches. Interference with the 7th nerve is usually not a problem in normal movements but only becomes apparent when a tighter lip seal is required such as blowing a musical instrument or blowing against closed lips when some escape of air will be noted. These problems become more frequent the closer one is to the commissure.

In order to minimize the risk of this occurring, incisions should be made through the mucosa only and any further tissue removal should be by blunt dissection to cause minimal trauma to any nerve branches (Fig. 9.12).

them are some proportion of the circumference of the circle), but some come in other shapes such as a J shape. A round-bodied needle is non-cutting and is generally used on friable internal organs. A cutting needle is triangular in cross-section with one edge sharpened to cut through the tissues. A cutting needle is necessary for mucosa and skin and also some of the fascial layers of the head and neck.

If the needle is bent so that the cutting edge is on the inside of the circle, it is called a forward-cutting needle since it cuts on its inside edge; whereas, if it is bent so that the cutting edge is on the outside of the circle, it is called a reverse-cutting needle since it cuts away from the direction the needle is passed. In most oral surgical procedures, the reverse-cutting needle is preferred, since a cutting needle will cut through the tissues too often (Fig. 9.14).

Today, many needles combine the best of all properties with a reverse-cutting tip and then a round-bodied needle so that the reverse-cutting portion makes the initial incision into the tissues, and then the round-bodied portion of the needle passes through the tissues without causing any further damage. These combined needles go by a number of different names such as taper cut.

Suturing techniques

Once the suture has been passed through the tissues, it must be tied in a knot. Knots can be tied with an instrument or with the hands, but in each case, the principle is the same. In general, a monofilament suture will require more knots than a braided suture since it is more likely to become untied. In most cases, the knot needs to start with a double overhand or double thumb knot, which is essentially a simple thumb knot with an extra twist on it, so that it is less likely to “give” or “slip” (Fig. 9.15). One more thumb

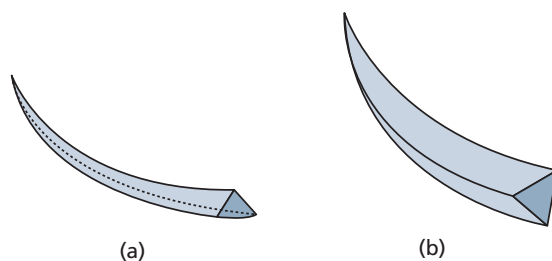


Fig. 9.14 The contours of (a) a forward-cutting and (b) a reverse-cutting needle. The reverse-cutting needle is usually more applicable in the oral cavity since it will not cut through the tissues.



Fig. 9.15 A double overhand, or double thumb knot.

knot is then thrown, and if this is in the same direction as the first knot, it will form a surgeon's knot which will not “give”, but if it is thrown in the opposite direction, it will form a granny knot which can loosen or be tightened (Fig. 9.16). This can be an advantage on occasions when one wishes to adjust a knot to exactly the right position and tension. However, the final knot that is thrown must convert the previous knot to a reef or square knot so that when it is tightened the knot will not loosen. Thus, there are variations in the technique, but in all cases, the first knot thrown must be able to hold the tissues in position whilst the second knot may be a square knot if the position of the first knot is ideal or can be a granny knot if further tension is required, but the final knot must convert the next-to-final knot to a reef or square knot.

As far as suturing techniques are concerned, the most frequent sutures used are simple interrupted sutures, and although there are no absolute rules, placing around three sutures per cm of length is a good compromise between having too many sutures and, therefore, too many stitch marks and too many possibilities of infection, versus not having enough sutures and having a widened scar. By making the base of the suture wider than the skin or mucosal puncture marks, a slightly everted wound edge is produced, which will flatten as it matures to blend with the adjacent skin or mucosa (Fig. 9.17).

Variations include both the horizontal and vertical mattress sutures which are generally employed intra-orally to obtain a watertight closure (Fig. 9.18). The vertical mattress suture, in particular, not only obtains a watertight closure but produces an everted suture line with a lot of tissue in contact to have the maximal chance of healing well. In particular, vertical mattress

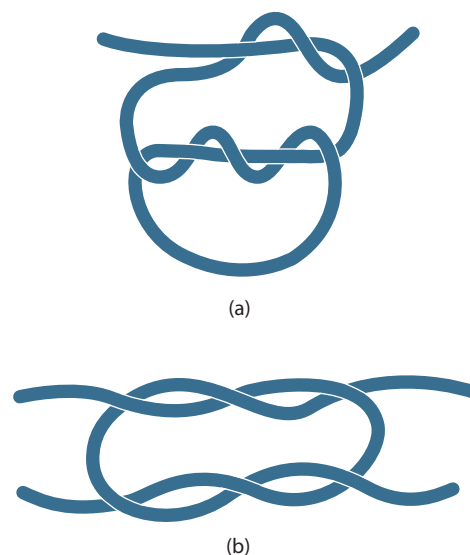


Fig. 9.16 (a) Conversion of a double thumb knot to a square knot which will not slip. (b) Conversion of a double thumb knot to a granny knot which can be adjusted. When it has been adjusted to the correct position it will require a third thumb knot to convert the last two into a reef or square knot.

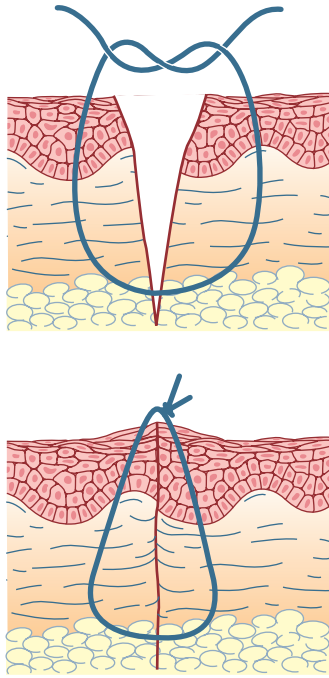
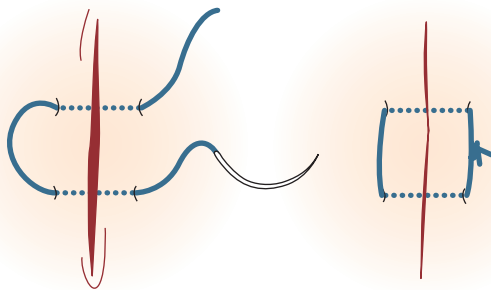
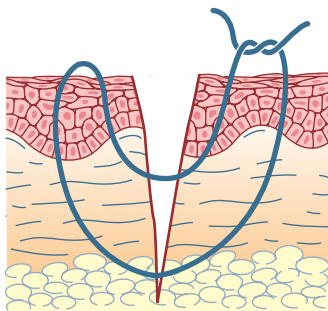


Fig. 9.17 Interrupted sutures placed six to the inch, or three to the centimeter. Note the deep pass of the needle is wider than superficially to cause eversion of the wound edges.

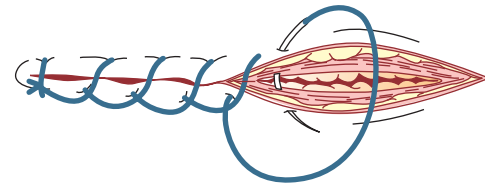


(a)

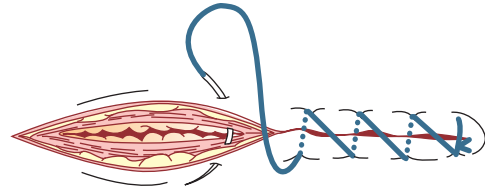


(b)

Fig. 9.18 (a) Horizontal mattress suture. (b) Vertical mattress suture. The horizontal mattress suture will provide a watertight seal, while the vertical mattress suture will provide excellent wound apposition with the best possibility of healing, particularly in a site where healing may be compromised, e.g. over a dead space.



(a)



(b)

Fig. 9.19 (a) A locking (suitable for intraoral use) and (b) a non-locking (suitable for cutaneous use) continuous suture.

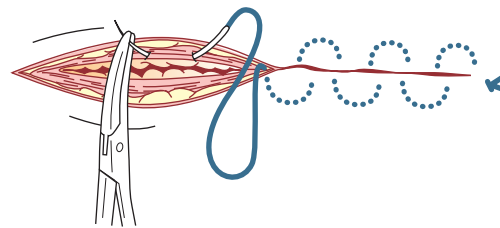


Fig. 9.20 The concept of a running subcuticular suture which is inserted in the white line of the dermis and advances by 3 mm with each pass of the needle but starts 1 mm behind the last pass needle on the opposite side of the wound. Various systems are available for starting and completing a running subcuticular suture,¹¹ but, in general, the knot is tied on the skin. Ideally, these sutures are left in place for at least 3 weeks to ensure the thinnest scar.

sutures can be used when suturing over a dead space such as a cyst cavity or an oroantral fistula. A horizontal mattress suture produces a watertight suture line.

Continuous sutures can be either locking or non-locking (Fig. 9.19). They are generally non-locking on the skin for added cosmesis, but in the oral cavity they can be locked for better closure and better waterproofing of the suture line. The risk with a continuous suture is that if it does break, the whole suture is rendered worthless; whereas, with separate interrupted sutures, the loss of one suture may not compromise the whole suture line.

A running subcuticular suture (Fig. 9.20) is an excellent skin closure technique providing the correct principles are adhered to. The running subcuticular suture needs to be inserted at the white line of the dermal junction, and each needle pass needs to be 2–3 mm but backing up 1 mm on the previous needle pass on the opposite side of the wound. If a non-resorbable material such as nylon is used, the suture

needs to come to the surface every 2cm to facilitate removal, but if a resorbable suture has been employed, this is not necessary. Running subcuticular sutures have the advantage that they can be left in for the optimal time for collagen strength across the suture line, which is about 3 weeks, without causing unacceptable stitch marks or infection which would occur if any type of suture was left in the skin for three weeks.^{9,10} Running subcuticular sutures can be in nylon or polypropylene, which are removed after 3 weeks, or a slow resorbing monofilament or coated multifilament suture, such as polydioxanone or polygluconate. Therefore, running subcuticular sutures¹¹ are particularly applicable in the areas of maximal cosmesis but can generally only be used with linear incisions. They are normally augmented with adhesive strips on the surface.

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Chapter 10

Complications Associated with Dentoalveolar Surgery

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This chapter's purpose is to review common complications associated with dentoalveolar procedures, primarily tooth extraction. While low risk, these procedures are routinely performed by general dentists and dental specialists. As such, the annual volume of these procedures results in a high number of actual complications. Unexpected complications from routine oral surgical procedures can lead to poor outcomes, deterioration of the patient–doctor relationship, and litigation. A clear understanding of the risks and benefits of dentoalveolar procedures on behalf of both the patient and practitioner is critical to modern dentoalveolar surgery.

Complications encountered with dentoalveolar surgery can be broadly classified into two groups: those side-effects and complications associated with any surgical procedure (pain, bleeding, swelling and infection) and complications specifically related to dentoalveolar procedures (oroantral communications, root displacement, alveolar osteitis). This chapter will review these complications, their frequencies, risk factors, diagnostic criteria, and management.

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Side-effects of dentoalveolar procedures

All operative procedures result in some degree of postoperative bleeding and inflammation, typically presenting as pain and swelling. As the body's natural mechanisms for wound repair and tissue regeneration are activated, a host of mediators will be concentrated in the wound area, resulting in activation of

nociceptive pathways and an increase in vascular permeability. While a detailed discussion of these processes is beyond the scope of this chapter, the well informed practitioner should have a general sense of the time course associated with these processes so as to gauge whether a patient's complaint of postoperative bleeding, pain, or swelling represents a normal response to surgical trauma or an aberrant reaction.

Bleeding

Etiology and prevalence

Postoperative bleeding is a side-effect of dentoalveolar procedures. In healthy patients, postoperative bleeding is typically minimal and self-limited by the clotting processes. The time course for clot formation is typically 6–12 hours postoperatively. Continuous active bleeding after this time period is considered to be excessive. In general, it is important to distinguish active bleeding from surgical site oozing. Patients will often complain of excessive bleeding because they have noticed blood in their saliva. Oozing should resolve within 36–72 hours postoperatively, should respond to pressure, and is generally a nuisance for the patient. In contrast, patients with an active bleed will often complain of their mouth filling with blood immediately after removing a gauze dressing or other local pressure measure.

Risk factors and prevention

Among the most important steps in the management of excessive postoperative bleeding is recognition of the at-risk patient. Numerous risk factors for bleeding have been identified in the literature. During the preoperative assessment, a detailed history should be obtained, including a history of disorders associated with coagulation, e.g. hemophilia, von Willebrand disease, use of medications such as antiplatelet agents like aspirin and clopidogrel (Plavix), vitamin K antagonists like warfarin (Coumadin), or heparin or its derivatives, e.g. enoxaparin (Lovenox) or

fondaparinux (Arixtra), an individual or family history of bleeding with surgical procedures, excessive bleeding upon loss of deciduous teeth, and, in females, a history of menorrhagia.

As the average age of oral surgical patients increases due to prolonged life, practitioners will encounter a greater number of anticoagulated patients. Appropriate adjuvant therapy, such as discontinuation of anticoagulant medications, factor infusions, or use of clot-stabilizing medications, should be considered in patients with risk factors for bleeding or known bleeding diatheses. Of note, patients taking clopidogrel, aspirin and other non-steroidal anti-inflammatory medication do not need to stop their medications prior to routine dentoalveolar procedures.¹

Patients on warfarin pose a common and special problem for the oral surgeon. The underlying medical problem, e.g. long-standing atrial fibrillation, deep vein thrombosis, prosthetic heart valve, or myocardial infarction, often prohibits discontinuing the anticoagulant. An acceptable, but uncommon, management strategy is to hospitalize the patient, discontinue the warfarin, and maintain the patient on a heparin bridge until the INR (international normalized ratio = $[PT_{(patient)}/PT_{(normal)}]^{ISI}$, where PT is prothrombin time and ISI refers to the international sensitivity index) is normal. An alternative option is to discontinue the warfarin preoperatively.

Care should be taken in considering the type of dentoalveolar surgery being performed. Many minor oral surgical procedures (such as single tooth extraction) can be done while the patient is anticoagulated, based on the coagulation profile.² In general, for patients on warfarin, a PT-INR <2.5 is generally acceptable if extraction of multiple (more than four) teeth is required. For extraction of one to three teeth, with no posterior teeth or surgical extractions, an INR of <3.0 is acceptable. Keeping this in mind for the patient who may need multiple extractions, staged visits may be most appropriate so discontinuation of the anticoagulants can be avoided.

Treatment

Excessive postoperative bleeding can be prevented intraoperatively by meticulous tissue management and local measures. In general, careful removal of granulated/infected tissue, minimal manipulation of surgical flaps to avoid tearing, use of local anesthetic with vasoconstrictor, primary wound closure, and the application of topical agents, such as absorbable gelatin sponge, oxidized regenerated cellulose fabric, or chitosan bandage, can limit most postoperative bleeding. Direct pressure with gauze in the immediate postoperative setting is an important method of limiting bleeding as the initial clot forms. Patients should be instructed to continue to apply pressure to the wound until bleeding has stopped.

Bleeding that persists after the initial phase of clot formation should be treated first with local measures, starting with direct pressure to the surgical site. Careful examination of the operative site with illumination and magnification and good suction can be invaluable to identify the source of bleeding. It is not uncommon to identify an incompletely formed clot, a "liver clot", that is mobile and continues to aggravate the site. Careful removal of the clot is critical to control the bleeding successfully. Use of local vasoconstricting agents, such as local anesthetics with a vasoconstrictor such as epinephrine, appropriate once the source of bleeding has been identified. If the vasoconstrictor is applied to the area prior to identification of the bleeding vessel, identification will be complicated. The wound may need to be repacked with a local hemostatic agent and sutured. Arterial bleeds that cannot be controlled with local measures should be treated with ligation or electrocautery. If bleeding persists, embolization, proximal vessel ligation, or other endovascular procedures may be required.^{3,4}

Pain

Etiology and prevalence

As with bleeding, postoperative pain is a side-effect of operative intervention. Pain associated with routine dentoalveolar procedures usually begins with the resolution of local anesthesia (6–12 hours) and typically peaks between 24 and 48 hours postoperatively.

Risk factors and prevention

Prevention of pain via intraoperative measures and adequate postoperative pain control measures is essential. Intraoperatively, minimizing tissue trauma and careful tissue manipulation will decrease inflammation and thus decrease pain. There is evidence that the administration of preoperative non-steroidal anti-inflammatory medications (salicylates, cyclooxygenase (COX)-2 inhibitors) can reduce the severity of postoperative pain.^{5–7} Postoperatively, the use of non-steroidal medications, as well as narcotic preparations with acetaminophen (APAP) (hydrocodone, oxycodone, tramadol) have been shown to be useful for treatment of moderate to severe postoperative pain.^{8–10} In addition, the use of long-acting anesthetics, e.g. 0.5% bupivacaine with 1:200 000 epinephrine, can be beneficial in delaying the onset of pain, which may allow the patient to start postoperative analgesics prior to the onset of pain. Some standard preparations for management of postoperative pain are listed in Table 10.1.¹¹

Swelling

Etiology and prevalence

Edema following the surgical removal of teeth and other routine dentoalveolar procedures is an expected

Table 10.1 Common medications used for postoperative pain control.¹²

Medication	Mode of action	Indication	Adult dose	Pediatric dose	Common side-effects
Acetaminophen (APAP)	Central prostaglandin synthesis inhibition	Mild to moderate pain	325–650 mg q4–6h not to exceed 4 g daily, 2 g daily in liver-compromised patients	<12 years of age, 10–15 mg/kg q4–6h, not to exceed 2500 mg daily	Hepatotoxic in high doses or when given in presence of ethanol
Ibuprofen/aspirin	Peripheral prostaglandin synthesis inhibition	Mild to moderate pain	Aspirin: 325–650 mg q4–6h not to exceed 3500 mg daily Ibuprofen: 200, 400, 600 q4–6h or 800 mg q8h not to exceed 3200 mg daily	Ibuprofen: >6 months of age 5–10 mg/kg PO q6–8h (suspension 50 mg/1.25 ml, 100 mg/5 ml)	GI upset, platelet suppression, renal hypoperfusion Consider coadministration of antacid or proton pump inhibitor
Opioids: Codeine Hydrocodone Oxycodone	Opioid receptor antagonist	Moderate to severe pain	In combination with APAP: 30 mg codeine, 300 mg APAP 5 mg hydrocodone, 500 mg APAP (Vicodin) 5 mg oxycodone 325 mg APAP (Percocet)	Codeine, 0.5–1 mg/kg, combine with APAP (see above dosing) q4–6h	Additive Constipation – consider coadministration of stool softener or laxative May cause drowsiness or sedation

Table 10.2 Common steroid preparations.^{11,15}

Medication	Preoperative evening	Day of surgery	Postoperative day 1	Postoperative day 2
Methylprednisolone sodium succinate (Solu-Medrol)	16 mg PO at bedtime for a morning case - Nothing for an afternoon case	- Morning case: 125 mg IV at start of case - Afternoon case: give 16 mg PO 4 hours prior to start of case; 125 mg IV at start of case - Night of surgery: 16 mg PO at bedtime	8 mg PO q6h	8 mg PO q6h
Methylprednisolone acetate (Depo-Medrol) long-acting	16 mg PO at bedtime for a morning case - Nothing for an afternoon case	- Morning case: 40 mg IM after local anesthesia administration - Afternoon case: 16 mg PO 4 hours prior to start of case; 40 mg IM after local anesthesia administration	Nothing	Nothing
Dexamethasone (Decadron)	8 mg PO at bedtime for a morning case - Nothing for an afternoon case	- Morning case: 16 mg PO 4 hours prior to start of case or 8–12 mg IV at start of case - Afternoon case: give 16 mg PO 4 hours prior to start of case; 16 mg PO 4 hours prior or 8–12 mg IV at start of case 8 mg PO night of surgery	16 mg PO in the morning	Nothing

finding during the postoperative course. The onset of swelling is typically between 12 and 24 hours following the procedure, with a peak swelling noted 48–72 hours postoperatively. Swelling typically begins to subside at 4 days postoperatively, with most patients experiencing resolution of surgical edema within 1 week postoperatively.^{3,13}

Prevention and treatment

It is important to inform patients of this time course and that swelling is an anticipated postoperative

finding. In the early postoperative period, the use of ice may help with management of swelling. In addition, patients should be told to sleep with the head of their bed elevated and not to sleep on their side, so as to avoid dependent swelling. Finally, perioperative steroids may be used to prevent swelling in patients undergoing significantly invasive procedures (e.g. third molar extraction). While the use of perioperative steroids may produce moderate decreases in swelling, these medications are typically short in action. Standard preparations for use are listed in Table 10.2.¹⁴

Postoperative complications

Surgical site infection

Etiology and prevalence

Because the oral cavity is home to a wide variety of bacterial flora, any intraoral wound will be exposed to a broad spectrum of aerobic, anaerobic, and facultative organisms with pathogenic potential. As such, postoperative infections should be primary among concerns for the practitioner when performing oral surgical procedures. Though the routine use of antibiotics for prevention of postoperative infections is still debated,¹⁶ there are several measures that can be implemented to reduce the likelihood of postoperative wound infection. A recent large-scale prospective study suggested that the infection rate following third molar removal was approximately 1%.¹⁴ For other, less invasive dentoalveolar procedures, the infection rate is typically lower.

Prevention

Prevention of postoperative infection begins with identification of the patient at risk. A number of studies have demonstrated that the incidence of postoperative inflammatory complications increases with age, smoking, pre-existing infection/pathology in the surgical area, oral contraceptive use, and lack of surgical experience. When dealing with impacted teeth, mandibular third molars have been shown to have a higher rate of postoperative infections than maxillary teeth.

As with other common complications, careful tissue management, debridement/curettage of necrotic/infected tissue, and thorough irrigation of the wound site will help to reduce the bacterial inocula within the wound site and reduce the possibility of infection.

Treatment

Patients presenting with infections will typically complain of persistent pain and swelling that is not improving with time, a foul taste, drainage from the wound, and limitation of jaw motion (trismus). Fever is variable and depends on the magnitude of the infection. Early recognition of an infectious process, typically a cellulitis, requires prompt treatment with an empiric course of antibiotics with broad-spectrum coverage for Gram-positive and anaerobic organisms (Table 10.3). If the symptoms have persisted for more than 48–72 hours after the procedure, an abscess may have formed, in which case incision and drainage of the abscess may be indicated, with collection of exudate for culture and sensitivity testing to guide antibiotic therapy (Fig. 10.1). Prompt recognition and treatment are necessary to prevent the spread of infection into the submandibular, sublingual, submental, retropharyngeal spaces and spaces of the deep neck, which can result in airway compromise and the necessity for emergency airway management.

Alveolar osteitis

Prevalence and etiology

Among the most common complications associated with oral surgical procedures, specifically the removal of impacted teeth, is alveolar osteitis (AO), or “dry socket”. The reported incidence is as high as 30% in some studies, though there exists a wide variation in reported incidence due to inconsistent diagnostic criteria.^{3,18} This condition, which was classically thought to be infectious in nature, is now understood to be associated with malformation, disruption, or other loss of a newly formed blood clot from an extraction socket. Patients presenting with AO will usually complain of a severe throbbing, radiating pain, often

Table 10.3 Common antibiotic formulations for treatment of surgical site infections.^{12,17}

Medication	Indication	Coverage	Adult dose/route of administration	Pediatric dose/route of administration
Penicillin VK	First line for odontogenic infections	Streptococci, oral anaerobes	250–500 mg PO q6h for 7 days	25–50 mg/kg/day q6–12h for 7 days (suspension 125, 250 mg/5 ml)
Cephalexin, Cephadroxil	Need for bacteriocidal broader coverage	Gram-positive cocci, some Gram-negative rods, oral anaerobes	250–500 mg PO q6h for 7 days	25–50 mg/kg/day divided q12h for 7 days, severe infections 100 mg/kg/day (suspension 100, 125, 250 mg/5 ml)
Amoxicillin	Need for broad spectrum, infections >3 days	Gram-positive cocci, <i>Escherichia coli</i> , <i>Haemophilus influenzae</i> , oral anaerobes	250–500 mg PO q8h or 500–875 mg PO q12h for 7 days	40 mg/kg/day PO divided q8h or 45 mg/kg/day q12h for 7 days (suspension 125, 250 mg/5 ml)
Clindamycin	Need for broad spectrum, oral anaerobes, penicillin-allergic patient	Gram-positive cocci, anaerobes	150–450 mg PO q6h for 7 days	8–25 mg/kg/day suspension PO divided q8h or q6h for 7 days (suspension 75 mg/5 ml)



Fig. 10.1 Postoperative surgical site infection. Purulent exudate can be seen emanating from the surgical site following extraction of tooth 32.

associated with a malodor from the surgical site. Trismus is an associated sign, related more to pain than swelling. Recognition of AO is based on the presence of new-onset severe pain, typically 3–5 days postoperatively, at which point pain and swelling associated with the operation should be beginning to subside. The lack of constitutional symptoms (fever), significant swelling, or intraoral discharge may help to distinguish AO from a surgical site infection. This distinction is important, since antibiotic treatment will not resolve AO. Physical exam findings may include a crypt-like socket with exposed bone and erythematous soft-tissue margins, food debris, or other detritus in the socket and extreme tenderness to palpation (Fig. 10.2). Radiographic examination should also be obtained to rule out the presence of a retained tooth structure or other surgical site complication, such as alveolar fracture.

Prevention

Preoperatively, there are a number of risk factors that have been identified that should alert the vigilant practitioner of the patient at risk. As with postoperative infections, age, oral contraceptive use, surgical experience, smoking, and mandibular surgery have been associated with AO.¹⁸ In addition, poor oral hygiene and pre-existing infections have been shown to increase the risk for AO.

Intraoperatively, thorough lavage of the surgical site has been shown to be associated with a decreased



Fig. 10.2 Alveolar osteitis. This patient, a 30-year-old smoker, who was also on oral contraceptives, presented with severe pain of sudden onset 4 days after extraction of tooth 20. The clot is absent and exposed bone can be seen (arrow).

incidence of AO.¹⁹ The use of topical medicaments and antibiotics, clot stabilizers (Gelfoam), platelet-rich plasma, and medicated mouthrinses have also been suggested for prevention of AO, with varying results.^{20–23}

Treatment

Once a diagnosis of AO has been made, treatment should be instituted immediately. As the condition is self-limiting, the treatment is supportive, with pain control being the primary goal. Treatment typically consists of gentle irrigation of the wound area with warm saline and application of medicated packing to the area, e.g. eugenol dressings, and aggressive use of oral analgesics. The packing should be changed every 24 hours until symptoms subside.

Fractures

Prevalence and etiology

While rare, fractures of the dentoalveolar process or mandible should be considered in the differential diagnosis of persistent pain or swelling after dentoalveolar procedures. Fractures are the result of using excessive force during tooth extraction (Fig. 10.3). If unrecognized and untreated, such fractures can lead to malocclusion, malunion, infection, and paresthesia.

Prevention

Preoperatively, there are few risk factors for fracture that have been identified. There is some evidence to suggest that age is a risk factor for fractures, presumably because of the loss of bone density, elasticity, and strength which are common findings in elderly patients. Atrophic mandibles or mandibles with large intrabony lesions are at risk for fracture.

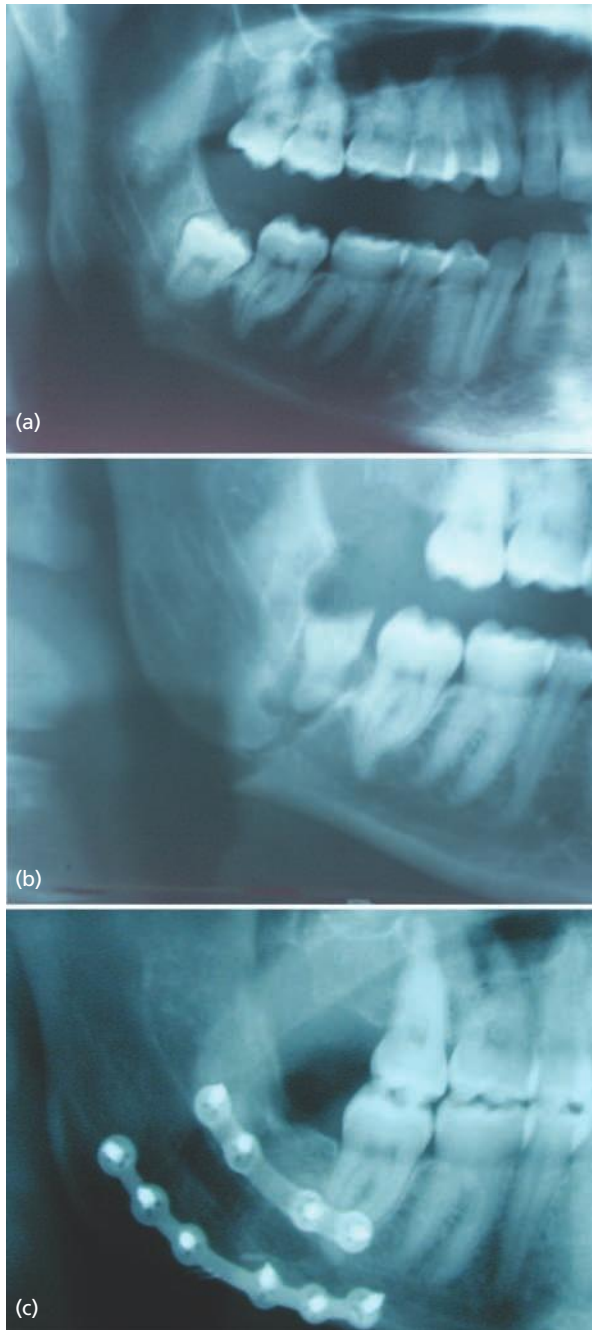


Fig. 10.3 Mandibular angle fracture upon attempted extraction of mandibular third molar. (a) Preoperative radiograph showing vertically impacted tooth 32. (b) The tooth has been partially extracted, with the roots remaining; there is a fracture of the mandibular angle. (c) After reduction of right mandibular angle fracture. (Photos provided courtesy of Leonard B. Kaban, DMD, MD.)

Treatment

Recognition of the fracture is the most important management. Intraoperatively, recognition of an alveolar process or mandibular fracture can result from observing a mobile alveolar or mandibular segment or malocclusion. Postoperatively, any patient complaining of malocclusion, pain and swelling disproportionate to the procedure, tooth displacement or mobility, or persistent numbness should be evaluated for a possible fracture.

Once a fracture is identified, typically with the aid of imaging studies, e.g. periapical or panoramic radiographs or computed tomography (CT), treatment is guided by the nature of the fracture and functional limitation. Treatment ranges from dietary modifications (blenderized diet) and early immobilization to reduction and fixation of the fracture.

Root fracture

Prevalence and etiology

Root tip fractures are common after tooth extraction, especially in posterior, multirooted teeth. Fracture of the roots, or root tips, is typically due to excessive forces applied during extraction with inadequate separation of the roots from the extraction socket and is commonly unavoidable due to the root anatomy and bone quality. When such forces are applied, the torque generated will typically cause a fracture at the junction between that portion of the root still attached to the socket and that portion already freed from the alveolar wall.

Prevention

The prevention of root fractures is primarily based on use of proper surgical technique, minimizing excessive forces, and carefully ensuring that teeth are adequately elevated and mobilized prior to luxation. Recognition of teeth at risk for root fracture is also an important preventative measure. Multirooted posterior teeth, roots that are curved, canines or other anterior teeth that have root dilacerations, or teeth with widely spaced, thin roots all have an increased risk for fracture. Inadvertent root fractures can be avoided by operative planning and prudent sectioning of a tooth prior to elevation and removal.

Treatment

The most important step in treating a fractured root or retained root tip is recognition that such an incident has occurred. Once the tooth has been removed, it should be carefully inspected to confirm that the roots were removed completely. Reconstitute the fragments of a sectioned tooth to confirm complete removal.

If a root fracture is noted, the socket should be irrigated thoroughly and an attempt should be made to directly visualize the retained root/ root tip. For teeth without preoperative evidence of periapical pathology or infection, small root tips (<3mm) can be left in place without adverse effects. In fact, for posterior teeth, the risk of causing damage to the maxillary sinus or inferior alveolar nerve may often outweigh the risk of leaving the fragment in place.

If there is associated pathology with the tooth preoperatively, the root fragment should be removed. Once the fragment is directly visualized, root tip

picks/elevators should be used to carefully separate the fragment from the alveolar socket, with special care taken not to apply apical pressure to the fragment. This gentle manipulation should be done until the root is mobilized, at which point it can be removed.

Root or tooth displacement

Prevalence and etiology

Displacement of root tips or tooth fragments is an uncommon but distressing complication nonetheless. Given the proximity of various fascial spaces to maxillary and mandibular third molars, displacement of tooth fragments can occur into the infratemporal fossa and maxillary sinus or submandibular space and inferior alveolar canal, respectively.

Prevention

Displacement of tooth fragments into these fascial spaces can often be prevented by using careful surgical technique. For example, when elevating an impacted maxillary third molar, the use of a periosteal elevator posterior to the distal aspect of the crown can serve as a barrier to displacement into the infratemporal fossa. In the event of displacement into the infratemporal fossa, a minimal attempt should be made to visualize the fragment and remove it. If this attempt is unsuccessful, the wound should be closed and the patient should be given a course of antibiotics. Future exploration of the region should be anticipated and CT, plain film imaging or both should be obtained to aid in localization and surgical treatment planning.

Careful examination of preoperative radiographs can be useful in evaluating the association between the roots of impacted maxillary third molars and the maxillary sinus. In older patients, or those with significant periodontal disease, pneumatization of the

maxillary alveolus can increase the likelihood of an association between the roots of the teeth and the maxillary sinus. In these cases, careful attention must be directed to avoiding excessive apical pressure to the teeth during the process of extracting teeth.

Treatment

In the event that a root fragment or tooth is displaced into the maxillary sinus (Fig. 10.4), the first step in treatment is localization of the fragment and prevention of further displacement. Typically this can be achieved by seating the patient in the upright position and obtaining periapical radiographs of the region of interest. In the event that a root or fragment is visualized, various methods can be used for retrieval. The simplest method is forcing positive pressure through the sinus to displace the root – this can be accomplished by closing the nostrils and having the patient attempt to exhale through the nose. Alternative methods include attempting to remove the fragment using a thin suction tip or packing the socket with iodoform gauze and removing the gauze in a smooth stroke, hoping that the fragment will be caught in the gauze. In the event that such measures prove unsuccessful, the maxillary sinus can be explored to directly visualize and remove the fragment. This can be done as a simultaneous procedure or at a future visit. If done at a future visit, the patient should be placed on sinus precautions (Table 10.4), antibiotics and nasal decongestants.

Table 10.4 Sinus precautions.

Sinus precautions	Ancillary medications
No nose blowing	Pseudoephedrine (30 mg, PO, q4h prn congestion for 7 days)
Open-mouthed sneezes	Amoxicillin 875 mg + clavulanic acid 125 mg PO, q12h for 14 days or similar coverage
No smoking	Saline nasal spray (two puffs, each nostril, q4h for 7 days)
No straws	

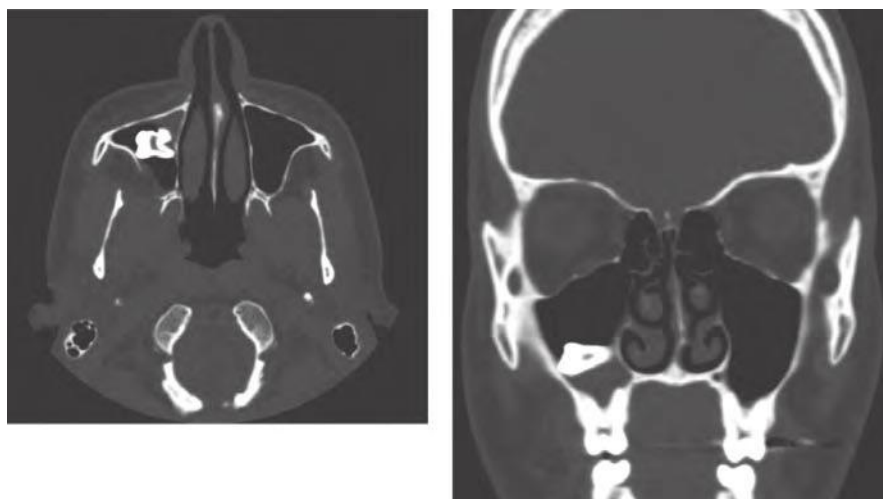


Fig. 10.4 Tooth displaced into maxillary sinus.

Displacement of mandibular teeth into either the submandibular space or inferior alveolar canal is a rare occurrence. However, given the vital structures in these regions, any suspicion of displacement must be evaluated fully. Visualizing any fragments is best accomplished using periapical films, with occlusal films in addition for submandibular space invasions. Once the fragment is identified, a gentle attempt should be made to remove the fragment from the inferior alveolar nerve (IAN) canal or the submandibular space, but care should be taken not to compromise the spaces and further displace the tooth. In the event that the fragment cannot be easily removed, delayed removal may be indicated and the patient should be placed on antibiotics. For fragments in the IAN canal, if the patient does not demonstrate any signs of infection or paresthesia in the postoperative course, the fragment may be left without concern. For fragments in the submandibular space, exploration of the region with lingual dissection and separation of the mylohyoid muscle from the mandible is indicated approximately 6 weeks after the initial operation. This delay allows for tissue fibrosis to occur around the displaced fragment, thereby stabilizing its position.

Oroantral communication

Prevalence and etiology

Following extraction of maxillary posterior teeth, oroantral (sinus) communications are common, commonly unrecognized, and do not need treatment. Persistent, symptomatic oroantral communications

are uncommon with a frequency of $<1\%$.³ Oroantral communications may result from excessive manipulation of the operative site or poor technique. It should be readily acknowledged that communications typically result from intimate anatomic associations between the roots of the teeth and the floor of the maxillary sinus and may be unavoidable (Fig. 10.5).

Prevention

As with displacement of teeth into the maxillary sinus, prevention of oroantral communications starts with identification of the patient at risk. Evaluation of preoperative radiographs for any evidence of encroachment of the roots upon the floor of the sinus should alert the surgeon to the likelihood of this complication. Upon removal of the tooth, the socket should be curetted gently, or not at all. If the tooth is not removed completely, judicious exploration should be undertaken, so as not to: (1) displace the remnant into the sinus; or (2) perforate the sinus floor while attempting to remove the fragment. A self-limiting oroantral communication may be an unavoidable side-effect of tooth removal due to the anatomic relationship between the roots and the sinus. Informing the patient of a high likelihood of an oroantral communication prior to tooth removal is preferable to explaining it after tooth removal.

Treatment

Diagnosis of a sinus communication is often made by having the patient force air through the nasal cavity while the nares are closed. If a large communication

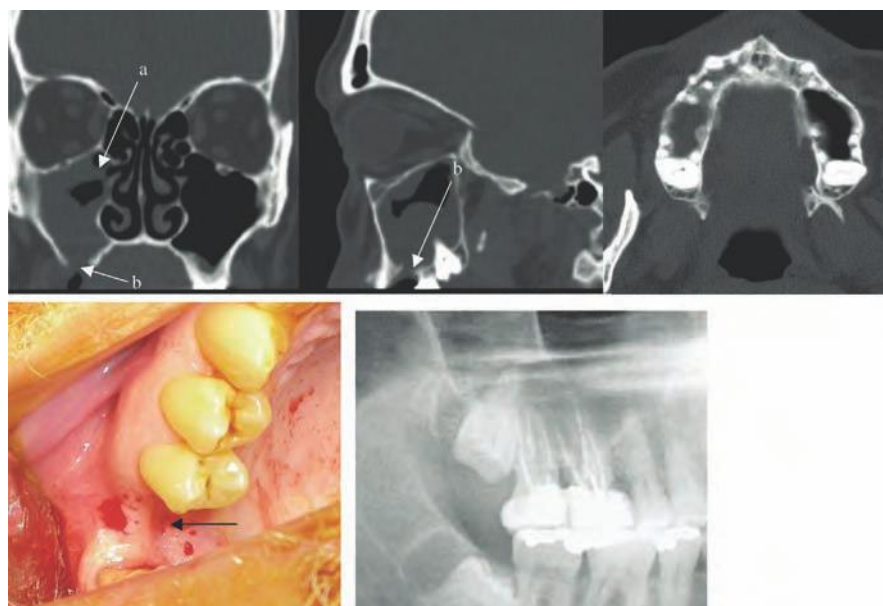


Fig. 10.5 Persistent oroantral communication following extraction of multiple periodontally involved teeth. The sinus on the right side is partially obliterated with inflammatory tissue (arrow a) and the bony defect can be readily visualized (arrow b) and is much larger than the apparent size of the lesion based on clinical examination (arrow in clinical photograph). Preoperative panoramic radiograph demonstrates close proximity of the roots to the maxillary sinus and a poorly defined margin of sinus floor around the apices of the dental roots.

exists, air bubbles should be visible within the socket. This method may prove ineffective for small communications.

In the event that a communication is discovered, either by tactile sensation or forced air maneuver, the size of the defect and patient complaint guide treatment.²⁴ As a general principle, any patient with a communication should be placed on sinus precautions, antibiotics, and nasal decongestants (Table 10.4).

Most oroantral communications heal spontaneously with little intervention. At the time of the procedure, primary closure of the extraction socket is not indicated. The patient should be placed on broad-spectrum antibiotics, e.g. penicillin or clindamycin, and sinus precautions. Monitor the patient closely over the postoperative period to confirm closure of the oroantral communication. If an oroantral fistula develops, standard procedures to produce a layered closure of the wound and management of the sinus are indicated. Clinicians should remember that the size of the visible fistula is much smaller than the actual bony defect (Fig. 10.5).

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Chapter 11a

Normal Wound Healing

Anh Le and Vivek Shetty

This chapter covers the description of the inflammatory phase, proliferation phase, and maturation and remodeling phase of normal wound healing. Specialized healing, to include skin and oral mucosal healing, bone and extraction socket healing, and implant site healing, are also discussed. The use of adjuvants to wound healing, including growth factors and hyperbaric oxygen, is covered, as are skin substitutes.

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severe tissue loss, or poor apposition of the wound margins, the healing process is prolonged with excess granulation and connective tissue formation, and is described as healing by *second intention*. Occasionally, the surgeon may attempt healing by *third intention* to address the challenges posed by infected or contaminated traumatic wounds with severe tissue loss. Here, the surgical repair is staged by allowing the wound to granulate initially and heal by second intention, followed subsequently by delayed primary closure.

Fig. 11a.1 illustrates the normal biology of healing, which consists of several distinct but overlapping phases including the inflammatory, proliferative, and remodeling phases.¹ Although rates and patterns of healing depend on a host of local, systemic, and surgical factors, the phases of oral mucosal healing closely approximate dermal healing. In general, wounds in the oral cavity appear to heal faster than dermal wounds. Oral wounds, despite being exposed to a bacteria-laden, moist, and seemingly hostile environment, heal perfectly well and re-epithelialize rapidly, with minimal or no scar formation.

Physiology

Any traumatic insult that creates a wound also initiates a complex cascade of closely synchronized cellular and molecular events that drive the process of tissue restitution. Normal healing is a primal life-preserving property that protects the integrity of the organism as a whole by repairing the damaged tissue architecture. Although normal healing may slightly differ from tissue to tissue, the components of the wound healing process are quite similar throughout the body. From a surgical viewpoint, the nature of the healing process is dictated by several factors, including the anatomical site injured, the type of tissue involved, the timing of the repair, and how well the wound margins are approximated. Early primary closure with adequate apposition of the wound margins usually results in healing by *first intention* wherein the wound heals uneventfully with minimal scar formation. In the presence of wound infection,

Inflammatory phase

Following injury the wounded site attempts to regain its normal physiologic state (homeostasis). Blood coagulation and platelet aggregation generate a fibrin clot within the vessel lumen and provide a provisional matrix for cell migration. Pro-coagulant factors, including fibrinogen, fibronectin and thrombospondin, are released by the injured cells and initiate the coagulation cascade. Concomitantly, the injured tissue and platelets begin to release several wound healing modulators including platelet-derived growth factor (PDGF), vascular endothelial growth factor (VEGF), and transforming growth factor β (TGF- β). The local cytokines and chemo-attractants recruit inflammatory cells which begin to remove damaged tissues and bacteria from the injured area. Clinical signs include localized edema, pain, redness, and increased warmth at the wound site. Neutrophils are the predominant inflammatory cells during the initial

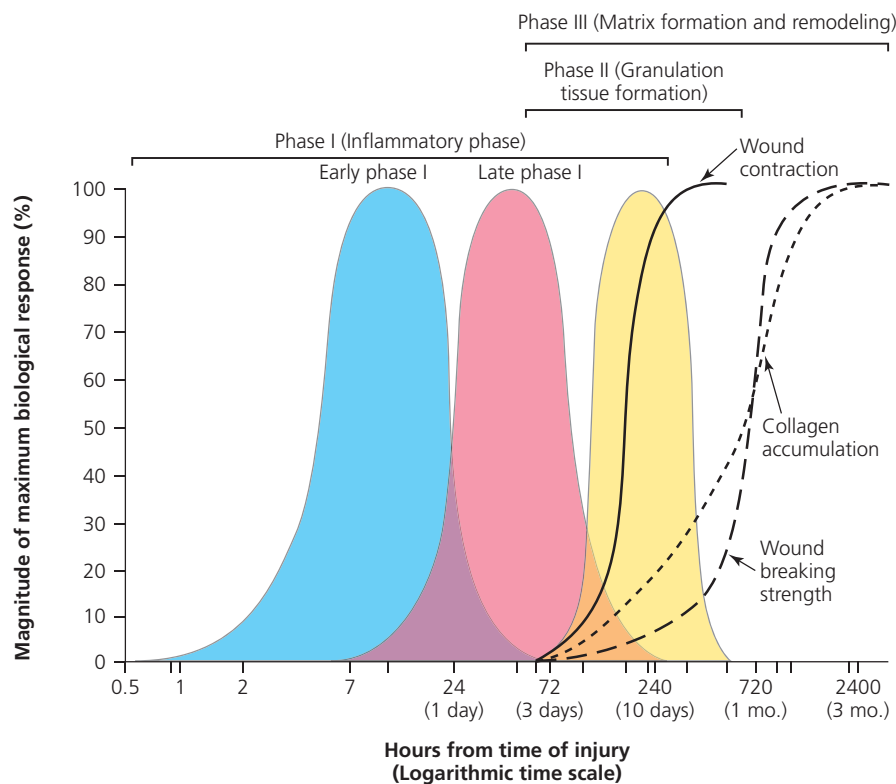


Fig. 11a.1 The three overlapping phases of wound healing.

2–3 days following injury, but are rapidly outnumbered by macrophages derived from mobilized monocytes (Fig. 11a.2a).

Proliferation phase

The proliferation phase is a period of intense replication of cells and is characterized by the migration and proliferation of fibroblasts and smooth muscle cells into the wound milieu. Fibroblasts are the major cells responsible for the production of collagen and proteoglycans. They interact with the surrounding matrix via receptors known as integrins that regulate the level of collagen gene expression and collagenase induction. Collagen restores strength and integrity to the reparative tissue, whereas proteoglycans function as moisture storage. Concurrent with these events is the process of neovascularization, whereby new blood vessels are formed and lymphatics are recanalized in the healing tissues. This essential process re-establishes transport of the nutrients and oxygen to the local injured site. In a synergistic way, the new capillaries supply nourishment to the developing collagen, while the collagen fibers structurally support the new capillary beds. Epithelial cells originating from hair follicles, sebaceous glands, and margins of the wound edges proliferate and resurface the wound above the basement membrane. In contrast to skin, the process of re-epithelialization progresses more rapidly in the oral mucosal wound. The oral epithelial cells migrate directly on to the moist, exposed surface of the fibrin

clot instead of under the dry exudate (scab) of a dermal wound.² The rapid re-epithelialization protects the wound from further insults from the oral environment, including food debris, foreign particles and microorganisms (Fig. 11a.2b).

Maturation/remodeling phase

The remodeling phase is the final stage of tissue repair and is distinguished by a continual turnover of collagen molecules, as precursor collagen is broken down and new collagen synthesized. The tensile strength of the wound is gradually restored as the collagen fibers are realigned and increasingly cross-linked to each other. The maximal tensile strength of a healed wound is reached in 6–12 months post injury but never reaches the strength of unwounded tissue. Eventually, active collagen synthesis achieves equilibrium with collagenolysis. The homeostasis can be impacted by disruptive processes such as poor oxygen perfusion,³ lack of nutrients, and wound infection, which favor collagen breakdown and wound dehiscence.

Specialized healing

Skin and oral mucosa (including grafts)

Healing of both oral mucosal and dermal wounds proceeds through the same stages – hemostasis,

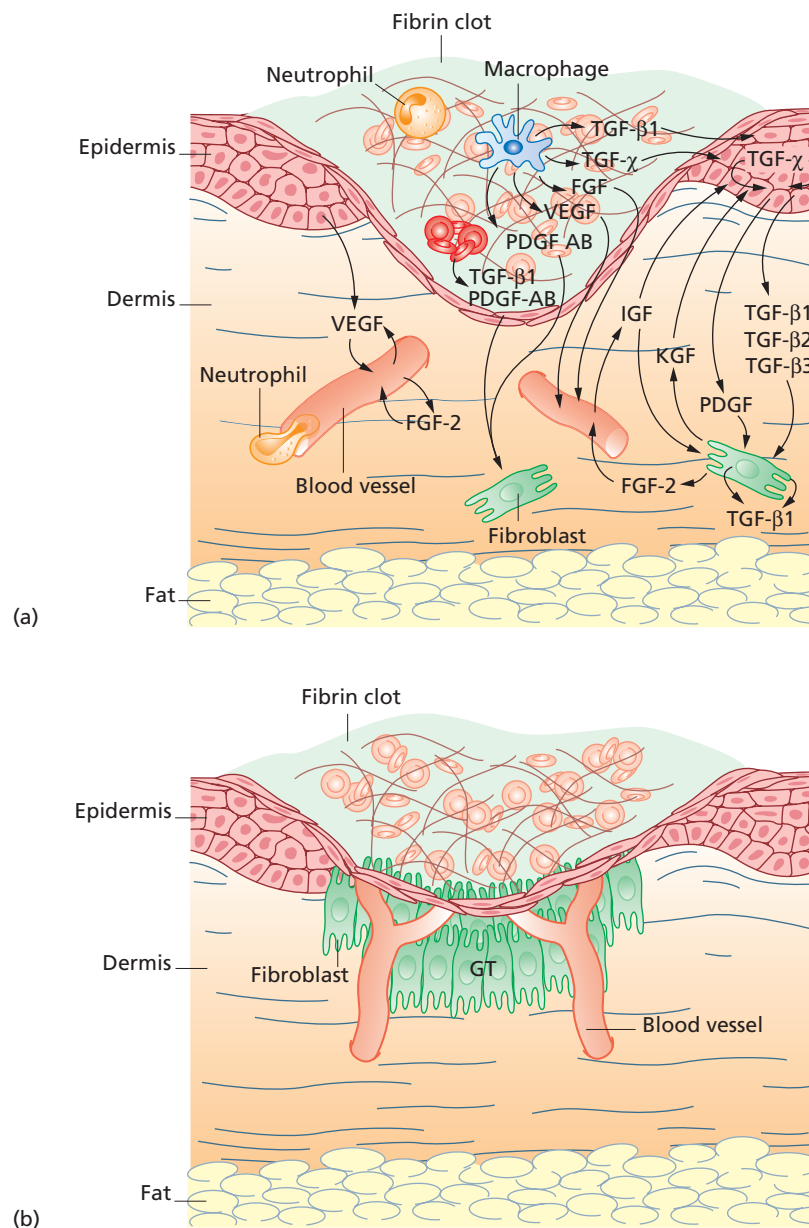


Fig. 11a.2 (a) Initiation of wound healing. Tissue injury precipitates blood clotting, platelet aggregation, and migration of leukocytes, including neutrophils and macrophages, to the site of injury. Initially, the blood clot is composed of fibrin and fibronectin, serving as a scaffold for cell migration and aggregated platelets, which release growth factors into the surrounding tissue. By day 3, the clot has contracted and accumulated numerous neutrophils, which phagocytose and kill microorganisms, and macrophages, which produce and secrete growth factors into the wound environment. Activated fibroblasts and endothelial cells in the periwound stroma begin to express integrins and secrete growth factors. (b) Repair of acute wound. Under normal physiologic conditions the wound continues to heal with an ingrowth of granulation tissue composed of fibroblasts, macrophages, and establishment of neovasculature. Marginal epidermis migrates over the newly forming tissue. Proteases restricted to the leading edge of migrating tissue cells facilitate their invasion of the clot and ingrowth into the wound. Many growth factors released by platelets and secreted by macrophages during the initial phase of healing stimulate tissue cells as they move into the wound. (Reprinted with permission from Clark R. *et al.*, *J Invest Dermatol.* 2007 May; 127(5): 1018–29. Copyright © 2007 Macmillan Publishers Ltd.)

inflammation, proliferation, and remodeling of the extracellular matrix. Differences between the two tissues relate mostly to the immediate environment (moist, saliva-bathed oral wounds versus the dry, air-exposed dermal wounds), the rapidity of the re-epithelialization, and the lack of scar formation. Oral mucosal wounds heal by first or second intention. Healing by first intention occurs with the apposition of the incised wound margins without exposure to the oral environment and minimal loss of tissue.

Partial-thickness wounds

Partial-thickness wounds, generated by abrasions or by graft harvesting, usually heal by epithelialization from wound margins and from remnant epidermal appendages, such as hair follicles and ducts in the wound bed. Following hemostasis, the partial-thickness wound forms a scab in skin or a fibrinous pseudomembrane in oral mucosa. Topical dressings in the form of fibrin matrix or synthetic materials to

keep the wounds moist can help re-epithelialization and decrease wound sensitivity. Gauze dressings impregnated with various antibacterial substances and occlusive plastic films are often used for this purpose. Systemic antibiotics do not work well for this, and should be used only if there is cellulitis in the surrounding tissues.⁴

Full-thickness wounds

Full-thickness injuries imply a complete loss of the epithelium and its appendages. Full-thickness wounds can be generated by tumor resection, trauma, burns, infection, radiation, or vascular compromise. Left alone, these injuries will heal gradually by granulation and epithelialization. However, infection may intervene before the protective epithelium is completely restored. If the wound bed is relatively septic and well vascularized, a full- or partial-thickness skin graft can be placed to enhance healing. An alternative is the use of a vascular flap to cover the soft tissue defect.

Bone/extraction wounds

Healing of an extraction socket is a specialized example of healing by second intention.⁵ Immediately after the removal of the tooth, the clot is formed in the alveolar socket within the first 24–48 hours, followed by engorgement and dilation of blood vessels, leukocytic migration, and formation of a fibrin layer. In the first week, the fibrin clot reorganizes into a temporary scaffold for cell migration and angiogenesis. Epithelium at the wound periphery grows over the surface of the organizing clot. Osteoclasts accumulate along the alveolar bone crest setting the stage for active crestal resorption. In the second week, the clot continues to get organized through fibroplasia and neoangiogenesis. Trabeculae of osteoid slowly extend into the clot from the alveolus and osteoclastic resorption of the cortical margin of the alveolar socket is more distinct. By the third week, the extraction socket is filled with granulation tissue and early woven bone islands form around the periphery of the wound. The surface of the wound is completely re-epithelialized with minimal or no scar formation. Active bone remodeling by deposition and resorption continues for several more weeks. Radiographic evidence of bone formation does not become apparent until 6–8 weeks following tooth extraction. As bone remodeling proceeds, the extraction site becomes less distinct and is inconspicuous after 6–8 months.

Occasionally the blood clot fails to form or may disintegrate causing a localized alveolar osteitis. In such instances, healing is delayed considerably and the socket fills gradually. Because of the absence of a healthy granulation tissue matrix, the apposition of regenerated bone to remaining alveolar bone takes place at a much slower rate. Compared to a normal socket, the infected socket remains open or partially

covered with hyperplastic epithelium for an extended period.

Implant healing

Wound healing around dental implants involves two processes, the healing of bone at the implant interface and the healing of soft tissue to the implant. Bone healing at the implant interface varies depending on the bone density at the implant site. Cortical bone requires a longer healing time than trabecular bone. At the implant surface, glycosaminoglycans secreted by osteocytes were initially deposited to coat the oxide layer, followed by a layer of osteoid or woven bone synthesized by osteoblasts. The greater the implant surface, the better the implant osseointegration. Thus, longer or wider-diameter implants will have more surface area available for osseointegration. The initial deposition of bone must occur before epithelium migration over fibrous connective tissue. The use of woven membranes to prevent fibroblast migration and soft tissue in-growth was initially proposed to selectively aid bone formation. Conditions that contribute to no movement during healing are the primary factor for a direct bone-to-implant interface. Other factors associated with implant failure include diabetes mellitus, head and neck radiation, and smoking.⁶ Once the bone-to-implant interface is established, the interface approaches the properties of the strongest load-bearing lamellar bone.

Wound healing adjuncts

An increased understanding of the wound healing processes has generated a heightened interest in manipulating the wound microenvironment to facilitate healing. The traditional passive ways of treating wounds are rapidly giving way to approaches that actively modulate the environment of the wound to accelerate the healing process. These approaches include treatments that selectively jump-start the wound into the healing cascade, stimulate cell recruitment and proliferation, increase oxygenation and perfusion of the local tissues, or mechanically protect the wound from further insults.

Growth factors

Various topical exogenous recombinant growth factors have been investigated as agents to accelerate the wound healing process. These include platelet-derived growth factor (PDGF), vascular endothelial growth factor (VEGF), epidermal growth factor (EGF), transforming growth factor (TGF), basic fibroblast growth factor (bFGF), tumor necrosis factor (TNF), and interleukin (IL). However, the potential of these extrinsic agents has not yet been realized clinically and may relate to figuring out which growth factors to put into the wound – and when and at what

dose. To date, only a single recombinant growth factor – recombinant human platelet-derived growth factor-BB form (PDGF-BB: Becaplermin, Ortho-McNeil Pharmaceutical, Raritan, NJ) – has been approved by the US Food and Drug Administration for the treatment of cutaneous ulcers, specifically diabetic foot, ankle, or leg ulcer. Results from several controlled clinical trials show that PDGF-BB topical gel was effective in healing lower extremity diabetic ulcers and significantly decreased their healing time when compared to the placebo group.^{7,8}

More recently, recombinant human keratinocyte growth factor 2 (KGF-2: Repifermin, Human Genome Sciences, Inc., Rockville, MD) has been shown to accelerate wound healing in experimental animal models. It enhances both the formation of granulation tissue in both young and old rabbits and wound closure of the human meshed skin graft explanted on athymic nude rats.^{9,10} The safety assessment of the drug showed that KGF-2 was well tolerated in humans with no differences in adverse events.¹¹

Hyperbaric oxygen therapy

Hyperbaric oxygen therapy (HBOT) is occasionally used in wound healing to raise tissue oxygen tension to a level that facilitates healing.¹² In HBOT, 100% oxygen is delivered to the patient at pressures between 1.5 and 2.4 atmospheres. This stimulates the growth of fibroblasts and endothelial cells, increases the killing ability of leukocytes and is lethal for anaerobic bacteria. Multiple studies of HBOT in the treatment of human diabetic patients suggest that HBOT can be an effective adjunct in the management of diabetic wounds.¹³ Animal studies suggest that HBOT could be beneficial in the treatment of osteomyelitis and soft tissue infection.^{14,15} Adverse effects of HBOT are barotrauma of the ear, seizure and pulmonary oxygen toxicity.

Skin substitutes

Immediate wound coverage is critical for the acceleration of wound healing. When the surface area is large, wounds can be covered by synthetic and natural dressings. The human skin substitutes available are grouped into three major types and serve as excellent alternatives to autografts.^{16,17} The first type consists of grafts of cultured epidermal cells with no dermal components (Epicel, Genzyme Tissue Repair Corp., Cambridge, MA). The second type has only dermal components (Alloderm, Life Cell Corp., Woodlands, TX; Dermagraft, Advanced Tissue Sciences Inc., La Jolla, CA). The third type consists of a bilayer of both dermal and epidermal elements (Apligraf, Organogenesis Inc., Canton, MA; Integra, Johnson & Johnson Medical, Integra Life Sciences Corp.,

Plainsboro, NJ). The chief effect of most skin substitutes is to promote wound healing by stimulating the recipient host to produce a variety of wound healing cytokines. The use of cultured skin to cover wounds is particularly attractive inasmuch as the living cells already know how to produce growth factors at the right time and in the right amounts.

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Chapter 11b

Compromised Wound Healing

Gösta Granström

In this chapter, the definitions of compromised wounds are described, as are possible background factors for a wound to be non-healing. Known factors at the tissue, cellular, and subcellular levels that inhibit healing are discussed. An evidence-based approach to handling of compromised wounds is given. Specific compromised wounds such as those appearing in irradiated tissues are presented, with special reference to osteoradionecrosis. The chapter also discusses other factors that can predispose to a compromised wound, including old age, osteoporosis, and diabetes mellitus. Finally, bisphosphonate-induced osteonecrosis is presented.

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Introduction

It is of the utmost importance for the oral and maxillofacial surgeon to realize that the patient, in whom surgery is to be performed, might have compromised tissues. The most common of these is the irradiated patient. A standard surgical procedure in such a patient could be associated with complications, and

result in a non-healing wound, tissue breakdown, bone sequestra, fistulas, and continuous draining. If, on the other hand, the surgeon is aware of the possible risks, precautions might be taken to prevent these problems ahead of surgery.

Compromised wounds

If wound healing fails to follow a normal sequence of events, then healing may be defined as compromised. A common feature of compromised wounds is that they appear arrested at a stage of inflammation. Continuous inflammation and the presence of bacteria may result in the generation of exudates containing disordered cytokine / growth factor network, high levels of proteolytic enzymes that destroy tissue proteins, growth factors, and extracellular matrix.^{1,2} Cells from the wound margin proliferate, but fail to migrate over the wound bed to re-epithelialize the wound because the extracellular matrix degrades and the wound remains open.

Microbiology of compromised wounds

Detailed microbiological investigations have shown that the wound bacteria are polymicrobial, involving both aerobic and anaerobic bacteria.³ Wound contamination occurs primarily from sources in close proximity to the wound site such as the surrounding skin, nares or mouth. In particular, the oral cavity is abundantly populated with a wide variety of microorganisms, where anaerobic bacteria predominate. In compromised wounds, the presence of devitalized tissue, hypoxia, and ischemia can stimulate microbial growth and hence the microbiology of these wounds is often complex. A diversity of both aerobic and anaerobic bacteria is common, and the prevalence of anaerobes has been observed particularly in infected compromised wounds.³ This may, in part, be attributed to the fact that many of the anaerobes commonly

found can possess a variety of virulence factors that impair the host response, especially different proteolytic enzymes. Interactions between aerobic and anaerobic bacteria are known to occur in compromised wounds, causing an increase in overall pathogenic effect.⁴ Aerobic bacteria can induce hypoxia by consuming local oxygen, thus favoring anaerobes. Compromised wounds are often densely populated with Gram-negative bacteria, and endotoxin content in the wound can be high.³

Another important factor in the compromised wound is the blood supply. Blood supports the wound with oxygen, nutrients, and inflammatory cells, all of which are necessary for healing. Oxygen is critical to wound healing, stimulating collagen production and migration of epithelial cells. Oxygen can further inhibit bacterial growth (anaerobes), and stimulate the number and migratory activity of polymorphonuclear cells (PMNs).⁴ Low local tissue oxygen (pO_2) is known to reduce the bacterial inhibitory capacity of PMNs. This has been reported at pO_2 below 30 mmHg^{5,6} and similar or lower values have been recorded in compromised wounds.⁷ In well perfused tissue with pO_2 above 40 mmHg, the probability of wound healing is higher.⁴ At suboptimal tissue pO_2 , the accumulation of hypoxic or anoxic tissue provides an ideal growth environment for a wide variety of microorganisms. Oxygen is thus critical to many processes that can influence the probability of healing in a compromised wound.

Proteolytic enzymes

The marginal cells of the wound are able to secrete different proteolytic enzymes. In the normal healing wound this activity is controlled. Proteases are required to destroy necrotic tissue and extracellular matrix to allow migration of cells and capillaries over the wound. Further, extracellular matrix is remodeled so that it achieves maximum strength. However, in compromised wounds there can be a lack of inhibitory factors to regulate protease activity. There can also be an overproduction of proteases. This loss of control of proteolytic enzyme activity has great potential for disruption of the healing process.⁸

Growth factors and extracellular matrix

The increase in protease activity in compromised wounds can result in degradation of different growth factors needed for regulation of the healing process.^{9,10} Extracellular matrix is primarily produced by fibroblasts. It determines the strength and elasticity by regulating collagen and elastin production. Fibronectin and laminin, also known as proteoglycans, are found in various combinations depending on wound age.⁸ While the mechanical properties of

the matrix are important for tissue function it is clear that it also acts as a reservoir for growth factors when required during healing.¹¹ Additionally matrix components such as vitronectin bind to surface cells and induce them to migrate over the wound surface as part of the re-epithelialization process.¹² Destruction of matrix by uncontrolled protease activity is believed to be an important factor preventing healing of compromised wounds.⁸

Treatment possibilities

In order to improve the possibility for healing of the compromised wound, it is necessary to improve the local environment so that it is unfavorable to microorganisms and favorable to the host repair mechanisms. Consequently, the removal of necrotic tissue, stimulation of endogenous antimicrobial and proteolytic activities, and the use of supplemental agents to address aspects of wound pathogenesis are necessary for the progression of wound healing.

Prevention of infections

Compared to compromised wounds, infection in postsurgical wounds is relatively predictable and can be significantly influenced by preoperative and intraoperative procedures. In situations where excessive bacterial contamination is expected, the probability of wound infection is greatly increased, and consequently prophylactic antibiotic therapy may be helpful.^{13,14}

Oxygen tension of tissues

Tissue oxygen tension is a key factor to prevent wound infection.⁵ A study to test different oxygen tension levels showed that the infection rate in patients receiving 80% inspired oxygen during and immediately after surgery was 54% less than in patients receiving 30% inspired oxygen (infection rates of 5.2% and 11.2% respectively).¹⁵ Hyperbaric oxygen therapy (HBOT), involving the intermittent inhalation of 100% oxygen at pressures greater than 100 kPa, has also been used as an adjunctive therapy in aggressive soft tissue infections and compromised wounds.¹⁶ Tissue oxygen levels of 400–500 mmHg achieved by HBOT facilitate the oxidative bacterial killing capacity of PMNs, directly inhibit growth of anaerobic bacteria, and promote collagen synthesis by fibroblasts.¹⁶ It has recently been shown that HBOT and basic fibroblast growth factor (bFGF) act synergistically, which may further promote healing in the compromised wound.¹⁷

Debridement (removal of necrotic tissue)

The presence of devitalized, necrotic tissue, and wound exudates causes compromised wounds.^{18,19}

Removal of such tissue is therefore important. Debridement can be achieved in several ways, and the choice of method is normally determined by the surgeon. With life-threatening medical conditions, such as necrotizing soft tissue infections and infected burn wounds, aggressive surgical debridement and intensive antibiotic therapy are essential to limit tissue loss and preserve life.^{20,21} Surgical removal of necrotic tissue, together with jaw reconstruction, is widely used in maxillofacial surgery for compromised wounds, especially of the lower jaw. This facilitates the restoration of the local blood supply, which is essential for healing. However, repeated surgical debridement of a non-healing wound can be traumatic to the patient and consequently slower and more passive topical therapies are often preferred for routine maintenance.

Another form of debridement that is occasionally used for removal of necrotic tissue and can be termed biosurgery, is maggot therapy. Maggots can lyse and ingest necrotic tissue and reduce bacterial growth. Secretions in the gut of the maggot are believed to have antimicrobial properties, with efficacy against *Staphylococcus aureus* and different streptococci.²²

Regulation of the amount of tissue exudate is also important for wound healing.¹⁸ Both the volume and composition of wound exudate can influence healing, and in compromised wounds, proteinase-rich exudates are likely to delay healing and cause damage to surrounding intact skin. Excess exudate is traditionally managed by a combination of absorbent dressings and topical antimicrobial agents. Vacuum therapy is sometimes used in infected and compromised wounds. This involves application of subatmospheric pressure (less than 100 kPa) to an enclosed wound environment. It has been reported to restore blood flow, remove excess exudate, reduce wound bacterial growth, reduce tissue edema, and improve granulation tissue formation.^{23,24}

Regulation of proteolytic activity

As discussed earlier, the presence of endogenous or exogenous proteinase activity can contribute to poor healing in compromised wounds. Consequently, therapeutic aims to regulate proteases, different enzyme inhibitors, and oxygen metabolites, as well as strategies to affect bacterial proteinase activity have been developed.²⁵ In the future, strategies will probably focus on good wound care practices involving compression therapy to reduce chronic inflammation, and advanced dressings that can facilitate regulation of proteinase activity in compromised wounds.⁸

Topical antimicrobial agents

Topical antibiotics and antiseptic agents have a long history in prevention and treatment of skin and wound infections. Antiseptic agents are gaining renewed interest and usage because they have a lower

risk to induce bacterial resistance than antibiotics. Molecular iodine is one of the most potent and broad-spectrum antimicrobial agents.²⁶

Some naturally occurring antimicrobial peptides that provide an endogenous defence mechanism in organisms ranging from bacteria to mammals have proved to be effective in compromised wound therapy.²⁷ Defensins and magainins are examples of such cationic peptides that operate by bonding to, and increasing the permeability of, bacterial membranes, with subsequent cell lysis and death.²⁸ A magainin derivative has been shown to exhibit broad-spectrum antimicrobial activity against more than 2500 Gram-positive and Gram-negative aerobic and anaerobic bacteria from infected ulcers.²⁸ These data suggest that antimicrobial peptides may provide a new strategy in the topical management of compromised wounds in the future.

Dressings

Since the 1960s, maintaining a moist wound environment has been recognized as being optimal for wound healing, and, as a consequence, moisture-retentive dressings, such as hydrocolloids, polyurethane films and foams, are now widely used. However, since a moist wound may contain a nutritious environment, this may also stimulate microbial growth. Infection might, therefore, occur if such conditions are maintained in a wound. In reality, this rarely occurs, and lower infection rates have been reported in wounds under moisture-retentive dressings compared with the infection rate under traditional gauze dressings.²⁹ The increasing availability of moisture-retentive dressings that contain antimicrobial agents may help to reduce infection and provide a new generation of products designed to optimize conditions for wound healing.

Specific compromised wounds

Non-healing wounds in irradiated tissue

Maxillofacial surgeons should be aware of the negative effects of oncologic therapy, especially in the oral cavity, to avoid serious problems. Radiotherapy is a well established treatment for advanced head and neck cancer. Early and late oral complications may occur in patients submitted to radiotherapy. Among the problems seen, the most frequent are: mucositis of the oral mucosa, xerostomia, trismus of the temporomandibular joint, increased caries, periodontitis, subcutaneous fibrosis, and osteoradionecrosis (ORN).³⁰⁻³² It is important for the surgeon to know the radiation dose delivered and the extent of the radiation fields, to avoid, or at least minimize, the risk for severe complications such as ORN when performing surgery in irradiated tissues.

Irradiated tissues have a decreased repair and remodeling ability after radiotherapy. The changes induced after radiotherapy are believed to be due to reduced cell turnover leading to reduced blood supply and reduced ability for bone repair.³³ Increased loss of attachment in the periodontium of teeth exposed to high-dose radiotherapy has been detected.^{34,35} These alterations may contribute to increased risk for infections, tooth loss, difficult-to-heal wounds, and ORN.

Osteoradionecrosis

ORN is perhaps the most feared late complication of irradiation, affecting the mandible more often than the other bones of the head and neck. It can occur as a complication after external beam radiotherapy, after brachytherapy, and as a result of either primary radiotherapy or adjuvant treatment. The exact definition of ORN has been a topic of debate in the literature, but, in general, it is a condition in which irradiated bone becomes devitalized and exposed through the overlying skin or mucosa, persisting without healing for more than 3–6 months.³⁶ This may progress to a pathological fracture, infection of soft tissues, and the formation of cutaneous fistulas, combined with severe and constant pain. The time taken to develop ORN can vary from 3 months to more than 15 years after radiotherapy. Dental extractions, surgery, or trauma frequently precede the onset of ORN, and it is probably related to impaired wound healing rather than infection, though secondary infection may be present. Factors that affect the development of ORN include primary site of cancer, proximity of tumour to bone, dentition, and type of cancer treatment and the radiotherapy dose. In addition, host factors such as age, sex, general health, cardiovascular disease, nutritional status, and continued tobacco and alcohol use may influence the onset of the disease.^{37–40}

Originally, the three factors thought to contribute to the development of ORN were irradiation, trauma, and infection.⁴¹ In the 1980s, Marx³⁶ described the first widely accepted theory of the pathophysiology of ORN. He proposed that irradiation caused an endarteritis that resulted in tissue hypoxia, hypocellularity, and hypovascularity, which in turn caused tissue breakdown and development of a chronic non-healing wound. Also, radiotherapy reduces the growth and function of bone marrow, affects periosteal and endosteal cells, and may reduce collagen production.⁴² It is now thought that osteoclasts suffer irradiation effects earlier than vascular alterations and that suppression of bone turnover via osteoclasia is another etiologic factor of importance.⁴³ It has recently been proposed that ORN arises from what is thought to be a fibroatrophic process.⁴⁴ The vascular damage concept is supposed to relate to endothelial cell reactions to radiotherapy, which range from apoptosis to permanent phenotypic changes. These vascular dysfunc-

tions help to generate the initial prefibrotic phase. A second but related theory is that of fibroblastic stromal change, which occurs as a result of reactive oxygen species' deregulation of fibroblast proliferation and metabolism.⁴⁴

Historically, the treatment of ORN has relied on a combination of conservative measures (antibiotics, debridement, and irrigation), surgical resection (sequestrectomy, marginal mandibulectomy or segmental mandibulectomy, with or without reconstruction), and HBOT. Roughly half of the patients ultimately require surgical resection of the mandible.⁴⁵ All necrotic bone should be removed, and, ideally, blood supply to the tissue be optimized. It has been proposed that the time interval between the completion of radiotherapy and the onset of ORN helps to dictate aggressiveness of surgical management,⁴⁶ but the amount of bone to be resected is a clinical decision and is based on the presence of bleeding edges of the remaining bone. For advanced stages of ORN (pathologic fractures, extraoral fistulas) segmental mandibular resection with free vascularized bone grafting has become the standard of care.

An adjunctive conservative measure that has been used since the 1960s is HBOT.⁴⁷ Using Marx's theory that ORN is a result of hypoxia, hypocellularity, and hypovascularity, HBOT is an attractive option. It increases oxygen supply in hypoxic tissue, stimulating fibroblast proliferation and angiogenesis.⁴⁸ Furthermore it stimulates collagen formation and can be bactericidal or bacteriostatic to many species.⁴⁷ For many years, the use of HBOT has been advocated for preoperative and postoperative treatment in high-risk patients undergoing tooth extractions and other surgical interventions. The beneficial effect of HBOT can be seen in the improved microvasculature of the bone.⁴⁹ In smaller lesions, it has been used in conjunction with conservative measures in an effort to avoid surgical resection of the mandible.^{50,51}

Osseointegration in irradiated tissues

Whether osseointegrated implant failures are increased after radiotherapy or not has been the subject of debate; the need for precautions such as HBOT therapy has also been debated.^{52,53} Increased knowledge today shows that a number of factors affect implant survival in irradiated bone. These include radiotherapy given before or after implant installation.^{54–56} Combined pre- and postoperative irradiation is particularly damaging to implant integration.⁵⁷ Factors that affect implant survival include irradiation dose and fractionation, chemotherapy, time from radiotherapy to implant surgery, but other factors such as fixture length and prosthetic retention also affect the results; the length of the follow-up is also relevant.⁵⁶ Despite the fact that implant survival might be affected by radiotherapy, the benefits that

the patient can gain from receiving osseointegrated implants is so high that it is often recommended. However, it is also stressed that cancer patients who are to receive osseointegrated implants should be treated at institutions well used to handling cancer patients. The risk for induction of osteoradionecrosis is always present, and it is of the utmost importance that such side effects are avoided. Adjunctive use of HBOT can reduce that risk and also improve survival of these implants.⁵⁸

Compromised skin grafts and flaps

Most modern reconstructive surgical procedures show good outcomes and no specific precautions are generally needed in normal, uncompromised skin grafts or flaps. However, in previously irradiated cancer patients, the risk for complications is significantly higher.⁵⁹ There are three distinct areas where adjunctive HBOT may be of significant value in the management of plastic surgical problems: preparation for grafting in irradiated tissue, salvage of compromised flaps and grafts, and reimplantation of facial structures.

Acutely compromised skin grafts and flaps benefit significantly from HBOT. Flap mottling after surgery is an immediate emergency indication for HBOT. In compromised flaps, HBOT can enhance flap survival and extend the margins of tissue salvage while also reducing the risk of sepsis. This has been confirmed in animals and several clinical studies. A critical review of HBOT and its application in different types of flap failure has been summarized by Zamboni *et al.*⁶⁰ Although different types of flaps and grafts have been analyzed in these studies and each flap problem is unique, the final determinant of flap necrosis was considered to be tissue hypoxia.

Old age – osteoporosis

Osteoporosis is a major health problem in societies with an ageing population. It is characterized by compromised bone strength that predisposes patients for an increased risk of fracture. Osteoporotic bone differs from normal bone in mineral composition, bone mineral content, and crystallinity.⁶¹ Poor bone quality in patients with osteoporosis presents the surgeon with difficult treatment decisions, i.e. regarding installation of osseointegrated implants. Increased failures of osseointegrated implants in older patients have been reported. Implant failures were correlated to a lower blood flow in the osteoporotic bone.⁶² When treating patients with jaw fractures, complications and slow fracture healing may occur. Fracture healing is a complex process, involving a well organized process that may be affected by both biological factors, such as age and osteoporosis, and mechanical factors, such as stability of the osteosynthesis.

Much effort has been expended to develop therapies that preserve bone mass and thus decrease frac-

ture risk.⁶³ Both manipulation of the local fracture environment, in terms of application of growth factors, scaffolds, and mesenchymal cells, and systemic administration of agents promoting bone formation and bone strength have been considered.⁶⁴ However, at present no general recommendations for the use of these treatments can be given.

Diabetes mellitus

Worldwide prevalence of diabetes is high (20% of individuals over 65 years), and is increasing.⁶⁵ Type 2 diabetes represents about 90% of the total diabetes cases. Diabetic patients have sometimes been denied maxillofacial surgical treatments such as osseointegrated implant installation, because of their increased susceptibility to infection, delayed wound healing, and microvascular complications.^{66,67} Diabetes control is critical for reducing complications of the disease. Patients with glucose fluctuations may be at more risk for complications.⁶⁸ For diabetic patients undergoing major surgery, it has been well established that absolute control of glucose plasma level by intensive insulin therapy in the perioperative period reduces the postoperative complications, i.e. deep wound infection and surgical site infection.^{69,70}

Using a similar scheme for strict control of plasma glucose, it has been shown that conventional and advanced implant surgeries, including lateral window sinus elevation, immediate loading, and guided bone regeneration can be performed without serious side-effects, and with surgical results comparable to those in non-diabetic patients.⁷¹ Thus strict control of plasma glucose in diabetic patients is recommended.

Drugs – bisphosphonates

Recently, oral and maxillofacial surgeons were the first clinicians to recognize and report cases of non-healing, exposed necrotic bone in the maxillofacial region in patients treated with intravenous (and more recently oral) bisphosphonates.⁷²⁻⁷⁴ The potential mechanism by which bisphosphonates are associated with development of osteonecrosis is still a matter of debate. Marx *et al.*⁷⁵ suspected the mechanism of osteonecrosis involved bisphosphonate-related apoptosis of osteoclasts as well as antiangiogenic effect. The rich vascularization of the jaws is believed to render them more susceptible to the deposition of bisphosphonates than other bones. Bone remodeling of the jaws can then be depressed, leading to spontaneous breakdown or poor healing after tooth extractions. Several consensus papers have been published that make recommendations for handling of bisphosphonate-induced osteonecrosis.^{76,77} The guidelines recommend today (2010) that unnecessary surgery be avoided as long as possible. If surgery must be undertaken, areas of necrotic bone should be removed and, if necessary, fractures stabilized by use of reconstruction plates. Any surgery to cure bisphosphonate-

induced osteonecrosis should only be performed in patients with extensive disease. Reconstruction by vascularized and non-vascularized bone transfer should be avoided. Further research is needed to better understand the pathogenesis of this disease, and better options for treatment are also needed.

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Part 2: **Dentoalveolar Surgery**

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Chapter 12

Extraction of Teeth

Adel Al-Asfour and Sanjiv Kanagaraja

Extraction is probably the most frequent oral surgical procedure worldwide and can be one of the simplest procedures as well as one of the most challenging technically. This is also a procedure that has profound effects on oral health status, often requiring restorative procedures to replace the missing tooth. It has also a psychological impact on the patient, both due to the fact that the patient will be losing a tooth/teeth, and the associations the patient will have with such a procedure. Extraction of teeth incorporates basic principles from physics, mechanics, and surgery, and the clinician should fully understand and master the techniques of extraction. A correct approach and performance of an extraction should lead to a tooth being gently lifted out of its socket without excessive trauma to surrounding soft and hard tissue and should prevent complications. In some situations, however, simple extraction is not possible and a surgical extraction must be carried out. This chapter gives an overview of the art of tooth extraction.

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Medical evaluation

As with many surgical procedures described in this book the medical status of the patient is crucial in the presurgical evaluation. Furthermore, the mental status of the patient is important since extraction requires the patient to be fully cooperative. The reader is referred to Chapter 1 for detailed information regarding patient evaluation.

Indications for extraction

The common position of modern dentistry is that all teeth should be treated and maintained in the oral cavity as long as possible, provided they fulfil functional and even esthetic criteria. However, it is sometimes inevitable that teeth have to be removed for various reasons.^{1,2} General indications for extractions will be discussed below, but the reader should be aware that the decision to extract has to be made individually for each case, and that recommendations are not absolute.

Caries

Severe caries and an extensive loss of tooth substance that will not permit restorative procedures is perhaps the most common reason why teeth are extracted.

The decision to extract a carious tooth instead of trying restorative procedures is something to be made by the surgeon and patient together.

Periodontal disease

Another common reason for tooth extraction is periodontal disease. Severe bone loss and irreversible tooth hypermobility is an indication for extraction (Fig. 12.1). However, bone regeneration techniques exist today and the dentist should consider this alternative before the decision to extract. The severity of periodontal disease, long-term prognosis, and even cost/benefit aspects should be considered in the decision-making process.

Pulp disease

The presence of irreversible pulpitis, pulp necrosis or internal resorption of the root canal where endodontic procedures are not possible are other indications for tooth extraction. This could be because of obliterated root canals, canals that are not accessible due to root anatomy, failure of endodontic therapy or when patient chooses not to undergo such treatment.

Pathologic lesions surrounding teeth

The most common pathologic lesion associated with teeth is apical or juxtaradicular periodontitis. If endodontic procedures are not possible, then extraction should be considered. Besides, if teeth compromise the surgical treatment of other pathologic lesions found in the tissues surrounding them, then extraction should be considered, e.g. in the treatment of osteomyelitis of the jaw.

Before radiation therapy

Careful consideration should be given to patients who are to undergo radiation therapy due to tumors

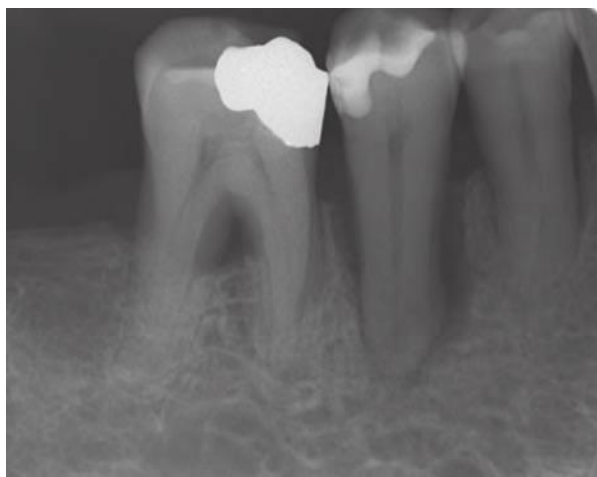


Fig. 12.1 Severe periodontal disease has led to extensive bone loss around 45 which is hypermobile and due to be extracted. Extraction in this case is easy.

in the head and neck region. Teeth associated with pathologic conditions such as periapical periodontitis should be considered either for swift endodontic procedures or extraction. Extractions should preferably be carried out before the start of the radiation therapy. A radical approach before radiation is recommended to avoid later complications in the irradiated bone. See Chapter 35 for more details.

Crown and root fractures

Crown, crown-root, and root fractures after trauma can often be successfully treated and extraction avoided, see Chapter 39. However, there are other situations where fractures of the crown and root do not allow successful restorative therapy (Fig. 12.2). In these cases extraction is the only alternative.

Teeth in bone fracture lines

Sometimes teeth in the line of a jaw fracture should be considered for extraction in order to prevent infection. Tooth luxation can almost always be treated by repositioning and fixation, but where there is severe luxation of teeth associated with complex jaw fractures, where teeth are in the line of the fractures and interfering with the repositioning, these teeth should be extracted (Fig. 12.3).

Malposition of teeth

Malposition of teeth in itself is not an indication for extraction. However, malposition associated with other conditions such as trauma to soft tissue or blockage of eruption of adjacent teeth is an indication for extraction. Where there is elongation of teeth due to the missing antagonist and where prosthetic rehabilitation is considered in the oppos-

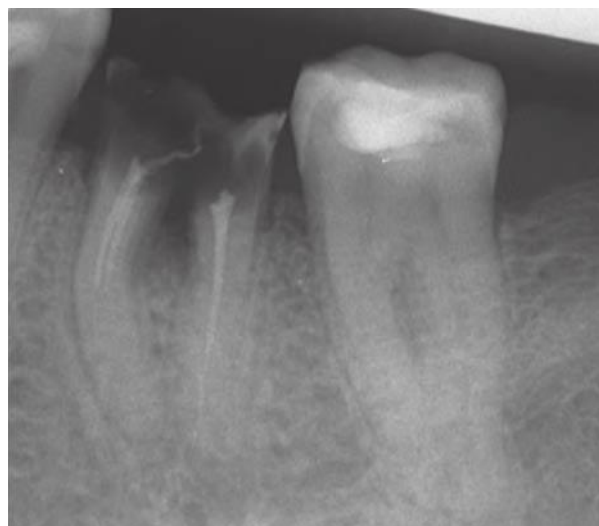


Fig. 12.2 Crown fracture of 36 leaving two delicate roots. These roots could be fragile and likely to fracture when extraction is attempted. Surgical removal of these roots should be considered.



Fig. 12.3 Tooth 48 which is root fractured and is located in the line of a mandibular fracture should be extracted. Tooth 33 is also in the line of a fracture but can be preserved if it does not interfere with repositioning of the bone fragment.

ing jaw, the elongated tooth may be considered for extraction (Fig. 12.4).

Impacted teeth

Some impacted teeth do not reach functional occlusion, often because of lack of space. These teeth should be investigated and considered for extraction if they interfere with adjacent teeth or present a potential for the development of pathology in the future. These could include the risk for root resorption of adjacent teeth, loss of bone around adjacent roots or development of other pathologic conditions such as cysts. Impacted third molars are the most common teeth considered for extraction. For more details regarding strategies for third molar extraction see Chapter 14.

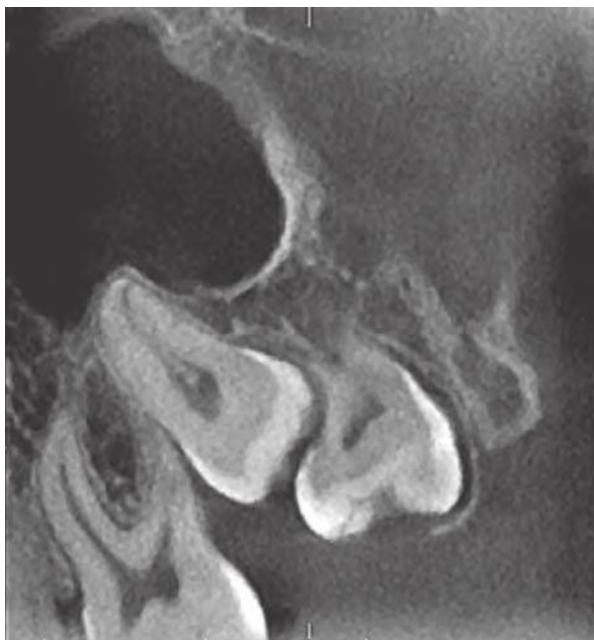


Fig. 12.4 Cone-beam computed tomography (CT) scan showing malposition of the upper left third molar blocking the eruption of the second maxillary molar.

Supernumerary teeth

Supernumerary teeth that are potential sources for future pathology should be removed (Fig. 12.5). The rules for impacted teeth apply to supernumerary teeth, which are often impacted. However, supernumerary teeth without pathology do not have to be routinely removed.

Orthodontic indications

Extraction of teeth is sometimes required to create space in order to carry out planned orthodontic treatment. The decision on which tooth/teeth are to be extracted is made by the orthodontist.

Before prosthetic extractions

Before prosthetic reconstructions it is sometimes necessary to extract teeth. Teeth could interfere with

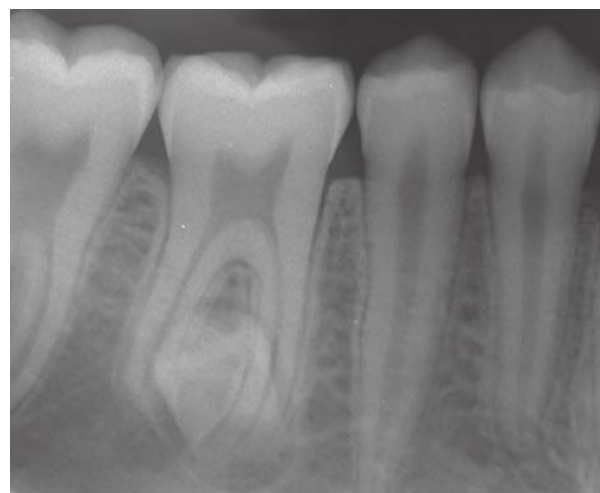


Fig. 12.5 Supernumerary tooth in the first molar region. This tooth will have to be followed radiologically and should be removed if any signs of pathology, such as progressive root resorption of the first molar, is suspected.

the proper placement of a fixed or removable prosthetic appliance and should be extracted. In the case of rehabilitation of a jaw with implants where there are one or two incisors remaining that have a dubious prognosis, it may sometimes be better to extract the remaining teeth and perform a full arch implant-supported reconstruction.

Before surgical extractions

Sometimes teeth have to be removed prior to other surgical procedures. The most common indication in this regard is the removal of impacted molars prior to LeFort-I or sagittal split osteotomies. This is done if the teeth lie in the line of the planned osteotomies or increase the risk for other complications such as undesired fractures.

Other reasons for extraction

There are other situations where extraction may be chosen, even when the extraction may be on doubtful indications. In these situations it is especially important to discuss alternative treatment and inform the patient preoperatively so the surgeon and patient agree on the choice of treatment. Extractions can be performed due to economic reasons. Patients might choose to extract a tooth rather than incur the costs of, for example, having endodontic treatment or a more expensive restorative procedure. Extractions can also be performed due to esthetic reasons. For example, the patient could choose to extract an upper malformed lateral incisor and have it replaced with an implant. Further, a patient could choose to extract protrusive teeth in the upper front and have them replaced by an implant-supported prosthesis as an alternative to undergoing orthodontic or orthognathic surgical correction. Malformed or severely discolored teeth could also be considered for extraction due to esthetic reasons where the patients choose extraction and prosthetic reconstruction over other procedures such as veneers and crowns. Teeth could sometimes also be extracted due to difficulty in maintaining adequate oral hygiene. Most commonly, the third molars are extracted due to inadequate oral hygiene and the recurrence of pericoronitis.

Contraindications for extraction

The decision to extract a tooth cannot be made entirely based on the status of the tooth and immediate surrounding tissue. The dentist should also consider the contraindications to extracting the tooth, for example with consideration of the patient's general health, which might overwhelm the indications for extraction.¹⁻³ In such a case the decision to extract is revised and the tooth left in place. Contraindications for extractions are for the most part relative and could be altered by the clinician implementing other treatments.³ When the contraindication is no longer

decisive, the previously planned extraction can be carried out.

Systemic contraindications

These constitute all general health factors and mental factors which have influence on the patient's ability to withstand the surgical procedure. Severe dental anxiety is a relative contraindication to extraction unless the procedure is planned under general anesthesia.

Patients with hemophilia or other coagulopathies should first have their disorders controlled before extraction. In general, most uncontrolled metabolic diseases such as diabetes constitute a contraindication until they are brought under control. Similarly, patients with severe uncontrolled hypertension and cardiac diseases should be treated for these conditions first before extractions are carried out. Ongoing radio- and/or chemotherapy is also a relative contraindication. In all cases, one should be aware of the medications patients are on or have had, especially those drugs that affect the immune system, delay or impair the healing process, or could interact with medication administered to manage an extraction. One should pay extra attention to patients on anticoagulant drugs, cancer medication, glucocorticoids and immunosuppressants. For a more detailed overview of medical contraindications see Chapter 3.

Local contraindications

The most common local contraindication is an ongoing acute inflammatory or infectious process. The acute infection/inflammation should first be treated before proceeding with the extraction, depending on the location of the acute process. Extraction of a third lower molar during an ongoing acute pericoronitis could lead to a life-threatening postoperative infection. However, there are also situations where an acute abscess is best drained by extraction of the tooth even in an acute phase. An acute infectious process caused by pulpal disease is resolved most quickly by extraction of the tooth. Therefore an acute infectious and inflammatory process should not be considered as an absolute contraindication for extraction. The dentist should, however, bear in mind that there could be other problems, such as severe pain, swelling, reduced mouth opening, and anxiety, which could make extractions associated with other acute conditions suboptimal. In such cases extractions should be deferred until the acute symptoms have subsided.

One of the most important contraindications to extraction is radiation therapy, past and present, involving the jaws. Delayed healing, dehiscence, and necrosis of the bone are often complications due to extractions performed in irradiated bone. Some cancer medications can also cause necrosis of the jaws

after extractions. For more details refer to Chapter 35. Finally, teeth within the area of a tumor, especially if it is malignant, should not be removed.

Clinical evaluation of teeth before extraction

The condition of the crown is assessed in order to evaluate if there is a high risk for fracture of the crown during extraction, which would thereby complicate the procedure. Surgical extraction of teeth with missing crowns or severe caries may be considered. Other factors to consider are tooth/root mobility and access to perform the extraction. Again, pathology of the pulp and surrounding soft tissues should be assessed so that the extraction can be carried out with a minimum of discomfort to the patient. Severe gingivitis or pulpitis, for example, could result in excessive bleeding or inadequate effect of local anaesthesia respectively, which could add to patient discomfort. The clinical evaluation of the tooth to be removed is done in conjunction with a radiographic assessment.

Preoperative radiographic assessment

It is imperative that a thorough radiographic examination be done prior to extraction of teeth. This helps the dentist to evaluate the degree of difficulty of extraction by assessing root anatomy, presence of pathology in the root or surrounding bone, vital structures and relation to other roots, and other factors such as ankylosis or hypercementosis of the root (Fig. 12.6). The most common and adequate radiograph is a good quality intraoral periapical radiograph. Other techniques such as panoramic radiographs, scanograms, and cone-beam radiological techniques are more valuable in the mapping of impacted teeth. For details of imaging techniques see Chapter 2.

In the maxilla, the proximity of the roots of the molars to the sinus should be assessed. In some cases the sinus membrane can rupture during extraction of maxillary molars causing an oroantral communication (Fig. 12.7). In the mandible, the position of the mandibular canal should be noted, especially in rela-



Fig. 12.6 (a) Hypercementosis of the mesial root of the first lower left molar would prompt surgical extraction of this tooth. Closed extraction in this case is impossible. The second molar has a large restoration, and the tooth could be fragile leading to fracture. The option of surgical extraction should be considered. (b) The extent of external root resorption makes extraction of the canine almost impossible due to the risk for fracture. Surgical extraction should be considered. (c) The root configuration of the second lower right molar makes it impossible to remove this tooth without a surgical procedure. (d) The lower first right molar has slender roots that taper towards each other apically. Besides, the cervical portion of the mesial root is narrower than the apical part. This will make extraction difficult and surgical extraction will have to be considered.



Fig. 12.7 (a) Note the proximity of the sinus membrane to 15. The infection around the apex of 15 can lead to destruction of the lower sinus wall and cause an oroantral communication when 15 is extracted. (b) Large maxillary sinus that extends over the apices of the molars with a risk for oroantral communication during extraction of these teeth. (c) Severe caries and loss of tooth substance will make extraction of 26 difficult. The maxillary sinus is in close relation to the apices which have apical periodontitis. This increases the risk for a rupture of the sinus membrane when extraction is attempted.

tion to third molars (Fig. 12.8). The position of the mental foramen should be noted in cases where a flap has to be raised in order to remove the premolars (Fig. 12.9).

Control of anxiety and pain

For a successful dental extraction, control of the patient's anxiety and pain is essential. Most patients have anxiety when undergoing an extraction procedure. Local anesthesia must be sufficient to eliminate pain and sensation from the involved tissues.

Sedation

Although pain associated with extractions is managed effectively through the administration of local anesthesia, patients are anxious because of the fear of pain. Anxiety control may vary from the surgeon simply showing concern and empathy for the anxious patient to the use of oral or parenteral drugs for sedation of the very anxious patients. In some patients general anesthesia has to be used to enable treatment. For further information regarding sedation and general anesthesia the reader is referred to Chapter 6.

Local anesthesia

Surgeons who are going to extract a tooth must have knowledge of the anatomy and especially the inner-

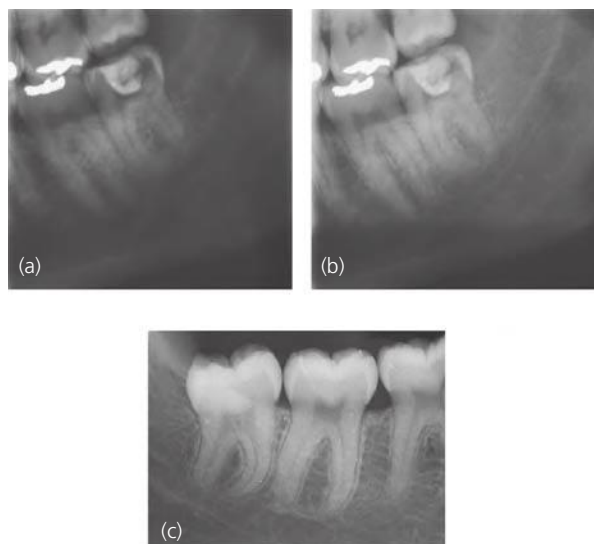


Fig. 12.8 (a) Scanogram showing the relation of the mandibular canal with respect to the apices of 37 and 38. Root anatomy makes it highly likely that the third molar would require a surgical extraction. The position of the mandibular canal must be determined before surgical extraction. (b, c) The distally angulated third molar and configuration of the root make an extraction avoiding a root fracture very difficult. Note the position of the mandibular canal in close proximity to the roots.

variations of teeth and surrounding soft and hard tissue. The surgeon must also be familiar with the techniques of local anesthesia. Profound local anesthesia results in loss of all pain, temperature, and touch sensations, but it does not anesthetize the proprioceptive fibers. The patient still may therefore feel sensations of



Fig. 12.9 (a) The mental foramen is just inferior to the roots of 45 with the anterior loop of the mental nerve visible. (b) The mental foramen is projected in contact with the apex of 45. The lamina dura of the root can be distinctly followed throughout, and this excludes apical pathology. (c) It is difficult to identify the position of the mental foramen from this single radiograph. Before surgical removal of the second premolar additional radiographs are needed to determine the relation to the mental foramen.

pressure which must be explained to the patient prior to local anesthesia and extraction.

There are various local anesthetics in use. They can be used with or without vasoconstrictor. They differ in their concentration, and duration of anesthesia. Each local anesthetic has a recommended maximum dose to ensure safety of the patient and this must be considered, especially when administered in very old or young patients. For more details regarding local anesthesia see Chapter 5.

Preparation for extraction

The concept of cross-infection control must be adopted during extraction. Patients must be regarded as possible carriers of blood-borne diseases that can be transmitted to the surgical team and the surgeon should practice the universal precautions to prevent injury or transmission of disease to their patients or themselves. Therefore, surgical gloves, surgical masks, and glasses or eyescreens with side shields should be worn by the surgeon even for simple extractions (Figs 12.10–12.12). The surgical team should wear long-sleeved gowns. For the patient, a minimal draping is necessary for performing simple extractions, with a sterile drape placed across the patient's chest. Hair should be ideally be covered both on surgeon and patient. Preoperative mouth-rinse with antiseptic, such as chlorhexidine, is recom-

mended to reduce the number of microorganisms at the surgical site.

Surgeon's position for extraction with forceps

The positions of the patient and surgeon are important for successfully performing extraction. The optimal position is when both patient and surgeon are comfortable (Figs 12.10–12.12). Dental extraction can

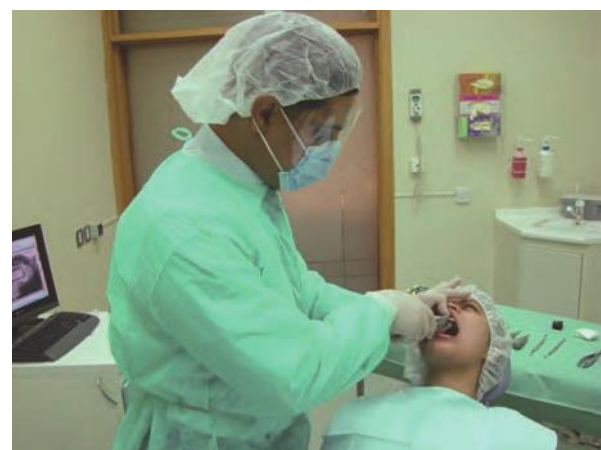


Fig. 12.10 Surgeon with proper clinical attire: gloves, mask, cap, and protective eye shield. Patient with sterile drape across the chest and a cap. The surgeon is in a standing position for extracting maxillary anterior teeth.

be performed with the surgeon sitting or standing. Most surgeons prefer the standing position. Regardless of whether the surgeon is sitting or standing, the correct positioning of the surgeon should allow him/her to deliver a controlled force to the patient's tooth through the forceps. The correct position allows the surgeon to provide stability and support and to enable the wrist to be straight with controlled force delivered from the arm and shoulder. The importance of this controlled force during extraction is to prevent any injury following sudden loss of resistance from the fracture of a root. The patient's chair should be tilted backwards. The height of the chair should be such that the patient's mouth is at the surgeon's elbow.

When extracting teeth in the maxilla, the surgeon should stand in front or by the side of the patient. For extraction of the mandibular teeth, the surgeon should approach the patient from behind during extraction of right posterior and anterior teeth. When



Fig. 12.11 Surgeon with proper clinical attire: gloves, mask, cap, and protective eye glasses. Patient with sterile drape across the chest and a cap. The surgeon is in a standing position for an extraction from the maxilla.



Fig. 12.12 Surgeon with proper clinical attire: gloves, mask, cap, and protective eye shield. Patient with sterile drape across the chest and a cap. The surgeon is in a sitting position with the patient lowered as low as possible so the mouth is level with the surgeon's elbow. The surgeon is in a sitting position for an extraction from the mandible.

extracting left posterior mandibular teeth, the surgeon should approach the patient from the front. The mandible can be supported by the surgeon's non-extraction hand. The approach from behind gives the surgeon great visibility of the extraction site and it allows the surgeon to be in a comfortable and stable position.

Principles of simple (closed) extraction

The mechanical principles applied from physics in extractions are the principles of lever, wedge, and rotating wheel and axle, where forces are transformed from long axes of the instruments and the surgeon's arms to deliver a high force in an appropriate direction at the point of application on the teeth and roots.^{1,3}

Extraction of a tooth requires expansion of the alveolar socket and separation of the attachment of the periodontal and attaching gingival soft tissues. For this reason it is important to study the anatomy of the tooth and its root, its periodontal ligament, and oral soft tissue attachment prior to extraction.

Controlled force is delivered with dental elevators and forceps to expand and, in a controlled way, fracture the alveolar socket without fracturing the alveolar process, roots or crown of the tooth. The force delivered must be carefully applied to avoid excessive damage to the supporting structures and adjacent teeth.

The tooth has to follow a certain path upon delivery from its socket. Dental forceps and elevators have been designed to provide a mechanical advantage for the delivery by a controlled force. The instruments used to extract teeth should therefore be properly selected and handled in order to deliver a controlled force in appropriate directions.

To avoid fracture of the tooth the forceps should firmly engage the tooth as apical as possible without



Fig. 12.13 The use of a periosteal elevator for releasing and expansion of gingival tissue before using elevators and forceps for extraction. This creates space for both instruments and minimizes soft tissue injury during extraction.

injuring the adjacent structures. The force delivered during removal while grasping the tooth as apical as possible can lower the center of rotation toward the apices of teeth, minimizing the chance of fracture of crowns or roots.

The first step in extraction is gently expanding the alveolar bone. This is achieved by introducing a dental elevator as a wedge as far apically as possible in the socket at the neck of the tooth to be extracted. Some surgeons prefer to start with gentle reflection of the gingival tissue around the tooth neck with a periosteal elevator to minimize the soft tissue injury and prepare for the beaks of the forceps (Fig. 12.13).

The dental elevator is used for lever transmitting from a long lever arm with moderate force into a short lever arm with high force (Fig. 12.14). A quarter turn moving the dental elevator toward the object tooth causes the alveolus to expand, loosening the periodontal ligaments, and forcing the tooth coronal-



Fig. 12.14 The dental elevator is used as a wedge with the tip being forced into the periodontal space to expand the alveolar bone and force the tooth out of the socket.

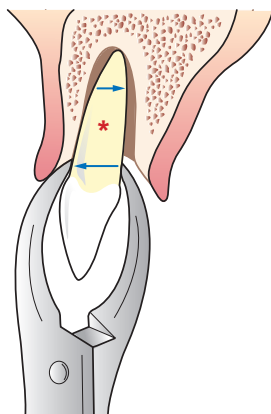


Fig. 12.15 The forceps is applied to the crown of a central maxillary incisor. The forceps is placed as far apically as possible so the center of rotation (star) is located as palatally as possible. Continued apical pressure is applied and the incisor is luxated buccally and lingually. Once some mobility is noted, the conical root allows rotational forces to be applied. Buccal, palatal, and rotational forces are used and finally the tooth is lifted out of its socket. The same technique can be used to extract maxillary lateral incisors and canines.

ly. The thumb and forefinger of the non-extracting hand of the dentist should be used to hold the alveolus, with one finger on the buccal and the other finger on the lingual aspects; in this position, the dentist can sense the amount of force being applied and the degree of movement of the object tooth. This maneuver can also protect the surrounding soft tissue and adjacent teeth from inappropriately applied accidental forces (Fig. 12.13).

The appropriate dental forceps can then be applied as far apically as possible by acting as a wedge in the socket. A steady grip must be obtained and luxation pressure is applied in lingual and buccal directions (Figs 12.15–12.17), continuing with gently applied persistent force to expand the alveolus. The center of

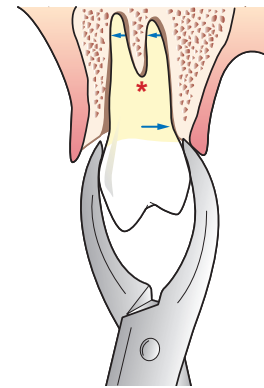


Fig. 12.16 The forceps is applied to a maxillary first premolar which has two thin roots. There is a high risk of fracture if careful technique is not applied. The forceps is placed as far apically as possible to move the center of rotation (star) as far apically as possible. The tooth is then luxated carefully buccally to expand the alveolus, and thereafter the tooth is luxated palatally. Rotational force should be avoided in teeth that have more than one root. After the tooth has been luxated it can be lifted out of its socket.

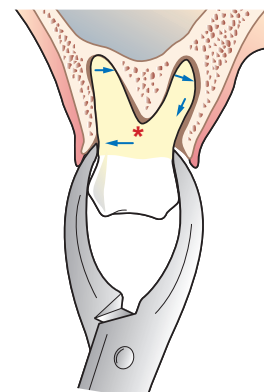


Fig. 12.17 The forceps is applied to a maxillary molar with two buccal and one palatal roots. Three-rooted teeth and root divergence will make the extraction difficult. The initial step is to seat the forceps firmly against the tooth and apply apical force to move the rotational center (star) as far apically as possible. Expansion of the sockets is achieved by careful buccal and palatal luxation. Large initial forces should be avoided and can result in uncontrolled fractures of the roots, buccal bone plate or tuberosity. Forces in buccal and palatal directions will enable bone expansion. After the tooth has been luxated the tooth is then carefully delivered out of the socket and mouth buccally.

rotation should be kept as apical as possible. One-rooted teeth can be rotated after initial luxation (Fig. 12.15). As the tooth gets luxated and loosened, the forceps should be repositioned as far apically as possible, this will increase the efficiency of the forceps, and thus lessen the chance of fracture of the roots.

Most erupted teeth can be extracted by simple extraction using the methods described.³ However it takes some time and experience to get the right feeling for luxation without fracturing roots. As the surgeon gains experience, his or her efficiency will improve, and the surgeon will also develop a sense for teeth that may present problems in their removal. If the extraction is difficult, a decision should be made early to surgically remove the tooth rather than apply excessive force. This is important, in that excessive force increases discomfort and anxiety of the patient, the risk for root and crown fractures, and the time for extraction.

Surgical (open) extraction of teeth or roots

Teeth that cannot be extracted by simple extraction require extraction by the use of a surgical flap. As a general rule, surgeons should consider performing an elective surgical extraction when they perceive a possible need for excessive force to extract a tooth. The term *excessive* means that the force will probably result in a fracture of bone, tooth root, or both. This will lead to an additional surgery and/or increase surgery time which can cause undue morbidity. The following are examples of situations in which closed extraction may require excessive force.¹⁻³ Surgical extractions for other indications are covered in Chapters 13–15.

1. Failure to remove a tooth with forceps or closed extraction. Instead of applying a force that may be not controlled, the surgeon should simply reflect a soft tissue flap, remove some bone, section the tooth or roots, and extract the tooth in sections.
2. Presence of thick dense bone, particularly bucco-cortical bone as assessed preoperatively. Surgical extraction should be considered. The extraction of most teeth depends on the expansion of the bucco-cortical plate. If this bone is thick and dense, adequate expansion is less likely to occur, and fracture of the root may be more likely. Dense bone in an older patient warrants even more caution.
3. Presence of short clinical crowns with signs of severe attrition as a result of bruxism or habitual grinding. It is likely that the teeth are surrounded by dense heavy bone. The surgeon should exercise extreme caution if removal of such teeth is attempted with a closed technique. An open

technique usually results in a quicker, easier extraction.

4. Hypercementosis. This is a condition where cementum has continued to be deposited on the tooth and has formed a large bulbous root that is difficult to remove through the available tooth socket opening. Great force used to expand the bone may result in fracture of the tooth or bucco-cortical bone and in a more difficult extraction procedure (Fig. 12.6a).
5. Teeth with long and divergent roots, especially the maxillary first molars or roots that have severe dilacerations or hooks, are also difficult to remove without fracturing one or more roots. By reflecting a soft tissue flap and sectioning the roots with a bur, a more controlled and planned extraction can be performed, with less morbidity (Fig. 12.6c, d).
6. Presence of large pneumatized maxillary sinus with the roots of maxillary molars extending into the sinus especially in the case of isolated molars (Fig. 12.7b). Extraction may result in removal of a portion of sinus floor with the tooth leading to an annoying complication known as oroantral communication. If the roots are divergent, such situation is even more likely to occur.
7. Teeth with extensive caries, root caries, or that have large amalgam restorations are candidates for surgical extraction (Figs 12.7c, 12.18). Although the tooth is grasped primarily by the root, a portion of the force is applied to the crown. Such pressures can crush and shatter the crowns of teeth with extensive caries or large restorations. Planned open extraction will result in quicker and easier extraction. Teeth with crowns already lost secondary to extensive caries, and present in the mouth as retained roots, can be considered candidates for surgical extraction.
8. Deciduous teeth occasionally present problems for the surgeon and should not be underestimated. The prime consideration in the removal of

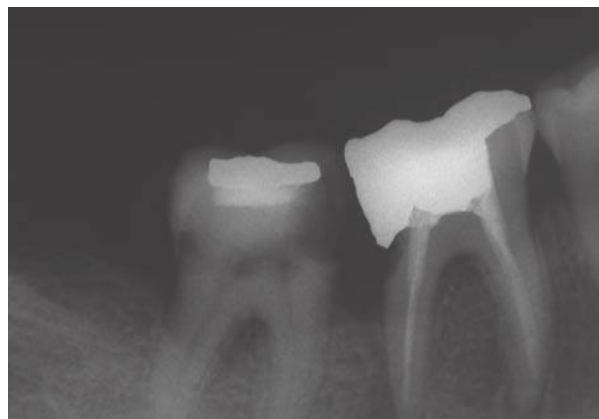


Fig. 12.18 Preoperative periapical radiograph showing severe caries in a second mandibular molar to be extracted. There is also evidence of a large amalgam filling on the first molar. Because of the high risk of fracture of crowns and filling a surgical extraction may be preferred.

deciduous teeth is to avoid injury to the developing permanent dentition. The enamel of the deciduous crowns is more brittle, and the roots are much flatter and more convex. Because the deciduous roots have already undergone resorption, seating the forceps far apically beneath the deciduous tooth may accidentally damage the developing permanent tooth. Primary teeth, especially the mandibular molars, may be submerged or ankylosed, so they should be percussed before extraction. If ankylosis is suspected, the tooth's removal should be reassessed. Radiographic changes include loss of evidence of a periodontal ligament space and apparent fusion of the bone and the tooth structure. Clinically, there is absence of mobility and a characteristic solid tone on percussion with a metal dental mirror handle. If extraction is necessary owing to infection or obstruction of an erupting permanent tooth, it is best done by a surgical procedure.

9. Erupted mandibular third molars with limited access. Although such teeth are not technically impacted, a forceps extraction is difficult due to difficulty of using the forceps properly and the presence of thick alveolar bone. Planned surgical extraction is performed by raising a soft tissue flap, and judicious removal of the buccal bone using fissure bur and surgical handpiece with copious saline irrigation to create a buccal trough to provide access for efficient instrumentation and atraumatic removal of the tooth. Surgical removal of third molars is covered in Chapter 14.
10. Endodontically treated teeth. Despite good pre-operative planning, occasionally tooth and roots will fracture during extractions. This is especially true for teeth previously treated with endodontic therapy, which often results in teeth that are brittle and may split during extraction.

Technique for surgical extraction

Surgical extractions should be carried out under high-standard aseptic conditions with the surgeon and assistant scrubbing up, wearing sterile gloves, caps, and masks, and using sterile instruments. The patient should be draped. High-speed air turbines should be avoided because of the risk for development of emphysema once a flap has been raised. Irrigation by sterile saline should be carried out when bone and teeth are cut by bur.

The flaps used with tooth extractions are envelope or sulcular flaps, which are developed along the cervical necks of the teeth.¹⁻³ If extended along the gingival margin they usually provide sufficient access to perform the necessary extraction surgery. It is best to extend this type of flap at least a minimum of one to two teeth both mesially and distally of the object tooth (Fig. 12.19). Another flap used is a modification of the envelope flap, which requires a releasing inci-

sion in either the mesial or distal end of the incision, forming a three-corner flap. Occasionally, both ends of the flap are released forming a four-corner flap.

Bone is removed by rongeur or by a surgical bur to provide access to the tooth which can be removed atraumatically in a more controlled manner. The buccal bone is usually removed and the tooth delivered from buccal aspect. Bone removal varies with the practitioner's personal experience and preference. Bone to be removed should be limited and minimized to preserve the alveolus. This has become important since the widespread use of dental implants requiring sufficient bone volume.

A straight dental elevator is used to expand the periodontal ligament space. The elevator is used as a wedge, pushing it apically to expand the periodontal space and thus displacing the tooth out of the socket. It is important in this case to use the dental elevator carefully by applying a finger rest using the index finger to control the amount of force delivered during removal of the tooth and to prevent slipping of the elevator to avoid unnecessary trauma to soft tissues (Fig. 12.19).

Teeth with two roots, e.g. mandibular molars, can be either sectioned into two halves and then removed similarly to two premolars (Fig. 12.20). In situations where the crown is missing, it is easier to separate the roots and remove them individually (Fig. 12.21).

In cases where the tooth is surrounded by thick cortical bone, bone removal is essential and important for atraumatic surgical extraction. Again a suitable surgical flap is reflected and a bone-cutting bur in a surgical handpiece is used with copious saline irrigation to remove at least one half or two thirds of the buccal bone covering the tooth root in the vertical dimension and about the whole mesio-distal dimension of the tooth in the horizontal dimension. The

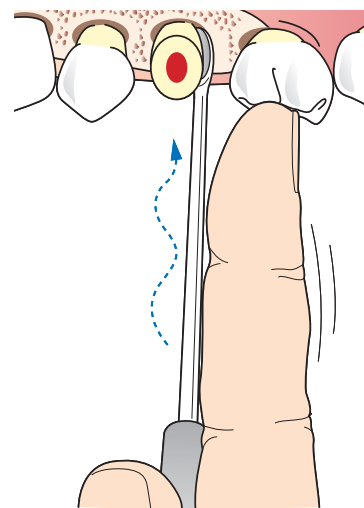


Fig. 12.19 Surgical removal of a single root after reflecting a flap. A small straight elevator can be carefully inserted into the periodontal ligament space to expand the alveolus and force the root out of its socket, using the elevator as a wedge. Note the finger support on the adjacent tooth to prevent slipping of the elevator causing unnecessary injuries.

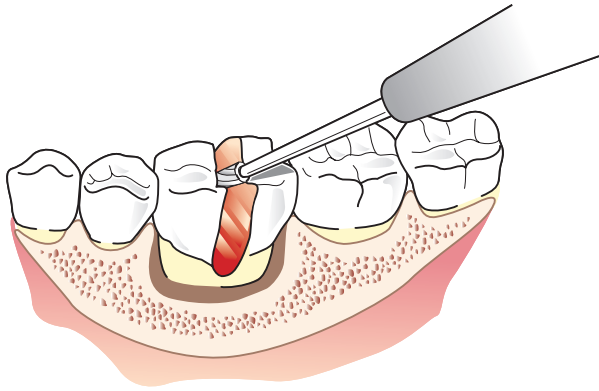


Fig. 12.20 Surgical extraction of a mandibular molar by separating the roots at the cervical level, followed by removal of the roots individually.

tooth can be then easily removed with an elevator or forceps in a buccal direction. Sometimes it might still be difficult to remove the tooth even after removal of bone. If the tooth is quite solid in the bone, a purchase point can be cut in the tooth root and a suitable elevator, e.g. a Cryer elevator, is placed into the purchase point to elevate the tooth out of the socket.

A maxillary molar is removed by separating the crown from the roots and then the tooth is sectioned at the trifurcation area to separate the roots from each other and elevate them out individually. Care must be practiced when using the elevator not to use too much force to push the roots into the adjacent maxillary sinus and to prevent slippage of the elevator injuring the soft tissues (Fig. 12.21).

It is a good practice for a surgeon to inspect the extracted tooth or root and make sure it is removed entirely. This is very important when tooth sectioning is involved. All extracted pieces should be assembled at the end of the procedure and inspected to make sure that the whole tooth or root is removed successfully. A smooth tooth apex on digital palpation also confirms a complete tooth removal.

In all surgical extractions, the base of the flap should be thoroughly irrigated with saline solution and the extraction socket should also be irrigated, especially when contaminated and when there are fragments of restorations or tooth present. Large sharp buccal bony edges should be removed with ronguers and then smoothed with a bone file. The flap is then adapted and sutured.

Postextraction care and instructions

Regardless if a tooth is removed by a closed or open surgical method, the patient should receive postoperative instructions necessary to provide proper care. These instructions should be given verbally and in writing, allowing the patient to take them home.

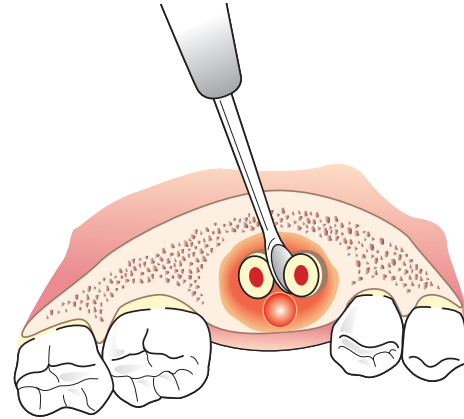


Fig. 12.21 Surgical removal of maxillary molar roots by elevator after root sectioning. The palatal root has been removed and the buccal roots are separated by an elevator before removal.

The patient and/or patient's guardian should be given the opportunity to ask any questions. After a tooth has been extracted, no debridement or curettage of the socket is necessary unless there is a pathologic process seen on a radiograph preoperatively. Obvious debris, such as tooth fragments, amalgam or calculus, may be present in the tooth socket after extraction and should be carefully removed with a curette or suction. Too vigorous curettage of the socket may lead to additional injury and may delay healing.

Gentle digital pressure with the index finger and thumb should be applied to the bucco-lingual walls of the extraction socket to compress the usually expanded bucco-lingual plates after extraction to restore the original crest dimension. The overlying mucosa should then be palpated to check for any sharp bony edges or projections. If any are present, the mucosa and underlying periosteum should be reflected and sharp bony edges smoothed with a bone file.

Postoperative instructions should include a method of applying direct pressure for initial control of hemorrhage. A moist 2×2 cm gauze should be applied directly over the extraction socket and the patient is instructed to bite down on the gauze for at least 30 minutes without interruption. It is important to place the gauze directly over the extraction site in the space occupied by the crown of the extracted tooth and not over the occlusal table of teeth to ensure that the pressure is correctly transmitted over the socket to achieve hemostasis. The patient should be informed that there may normally be slight postoperative bleeding during the first few hours; some oozing from the wound, which results in a red tinge to the saliva, can be expected for the first 24 hours following the extraction. The patient should be given extra gauze to take home and maintain pressure for another 30 minutes if bleeding resumes. If it is difficult for the patient to stop the bleeding, the patient should contact the surgeon.

The patient should be instructed to stay on a specific postextraction diet. A high-calorie, high-volume liquid diet is best for the first 12–24 hours. The patient must have adequate intake of fluid during the first 24 hours. The fluid can be juices, milk, water, or any other beverage. The patient should be instructed not to use a sucking straw; otherwise he or she will cause negative pressure that might lead to bleeding episodes. The diet should be soft and cool. Cool and cold foods help keep the local area comfortable. Hot and solid foods tend to cause local trauma and/or initiate bleeding episodes.

As with other surgeries the postoperative course also varies with patients, depending on expectations and earlier experience. Mild to moderate postoperative pain is best managed by the use of over-the-counter analgesics, e.g. non-steroidal anti-inflammatory analgesics such as ibuprofen, aspirin, or acetaminophen. A combination of non-narcotics

and narcotic analgesic drugs might be necessary for postoperative pain following extensive surgical manipulation. Whatever drug is chosen, it is important that the patient is instructed to start the pain medication before the effects of the local anesthetic have ended.

The written instruction sheet should contain a telephone number or any emergency number for the patient to contact if he or she has any question or should any complication arise.

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Chapter 13

Current Concepts and Strategies for Third Molar Removal

Leif Lysell

Surgical removal of third molars is controversial and various strategies for indications for surgical removal are applied around the world. This chapter gives an overview of current knowledge of the natural history of impacted third molars and the current indications and strategies for third molar removal, and the appropriateness of prophylactic third molar removal is discussed. Public health aspects on third molar surgery are included and relevant aspects on judgement analysis and clinical decision making based on evidence are presented to elucidate when to remove and when not to remove third molars.

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Introduction

The surgical removal of third molars has been, and still is, the most frequent operation performed by oral and maxillofacial surgeons both in private practice and in hospital settings. It has not always been a routine, safe procedure with a generally predictable outcome. In the first half of the 20th century, especially before antibiotics became available to the dental profession, severe side-effects of the operation were well recognized by the practitioners, and surgery was often delayed until evident symptoms and pathology

had developed. The second half of the 20th century marked a rapid rise in third molar surgery. This was the effect of a considerable development in technology and pharmacotherapeutics. Development of more efficient rotary cutting instruments, panoramic radiography, improved local anesthesia, outpatient general anesthesia, new chemotherapeutic agents and agents for conscious sedation, together simplified and made third molar removals a considerably safer procedure.¹

Another factor that influenced the practice of oral surgery was the growing interest in preventive dentistry that developed during the 1950s and 1960s. In a major paper on preventive oral surgery,² a “wait and see” attitude towards the asymptomatic impacted third molar was rejected. It was stated that “*current thinking advocates the taking of roentgenograms at the time teeth should normally erupt and, if they are found to be hopelessly impacted and cannot possibly erupt into the mouth and become serviceable teeth, they should be prophylactically removed at this time*”. This approach to the third molar problem was almost universally accepted during the second half of the 20th century and is still the prevailing opinion in a currently used textbook in oral and maxillofacial surgery.³

This concept of prophylactic third molar removal has, however, been questioned by some researchers in the last 20 years.^{4–7} As will be reviewed later, this discussion of prophylactic third molar surgery has been stimulated by research in medical decision making, public health analysis, cost-effectiveness studies and by incorporating patient preferences in decision making. It is well known in the practice of medicine that there is a geographical and cultural variation in diagnosis and treatment⁸ and this variation can obviously also be expected in the management of the third molar problem. This can be explained by a variation in education, treatment traditions, patients’ expectations, cultural variation to risk perception and the supply of expert providers of dentoalveolar surgery.

The aim of this chapter is to survey the current knowledge of the natural history of impacted third

molars and the current indications and strategies for third molar removal, and discuss the appropriateness of prophylactic third molar removal. To further elucidate these subjects, public health aspects on third molar surgery are included and so also are relevant aspects on judgement analysis and clinical decision analysis.

Definitions, prevalence, and public health aspects

An impacted third molar is a developmental anomaly caused by an obstruction in the eruption path or by an ectopic position of the tooth. The etiology of third molar impaction has been well reviewed by Svendsen and Maertens.⁹ From a surgical point of view, the degree of impaction and the position of the impacted third molar have been divided into different categories. Most classifications were developed for preoperative judgement of surgical difficulty and are based on radiographs.¹⁰ In this text the degree of impaction is classified as: (1) partially covered by soft tissue; (2) completely covered by soft tissue; and

(3) completely covered by bone tissue. The angular position of a third molar is classified as vertical, mesioangular, distoangular, or horizontal, according to the criteria described by Winter (Fig. 13.1).¹¹ These classifications can be used when comparing the results of different clinical studies, especially those concerned with the development of associated pathology. Winter's classification is a practical description for clinical use. For a more scientific evaluation of the third molar angulations described, the definitions of Shiller can be used.¹²

Management of an impacted third molar may include surgical exposure, transplantation, removal of the impacted molar or long-term observation in cases where a decision is made for retention of the impacted tooth. Indications for surgical exposure or transplantation are not included in this chapter. *Therapeutic third molar removals* are performed when a patient reports subjective symptoms from the wisdom tooth in combination with clinical signs of local pathology or when a patient is asymptomatic but clinical examination reveals third molar-associated pathology. In contrast to the therapeutic indications for third molar removal the *prophylactic removal of*

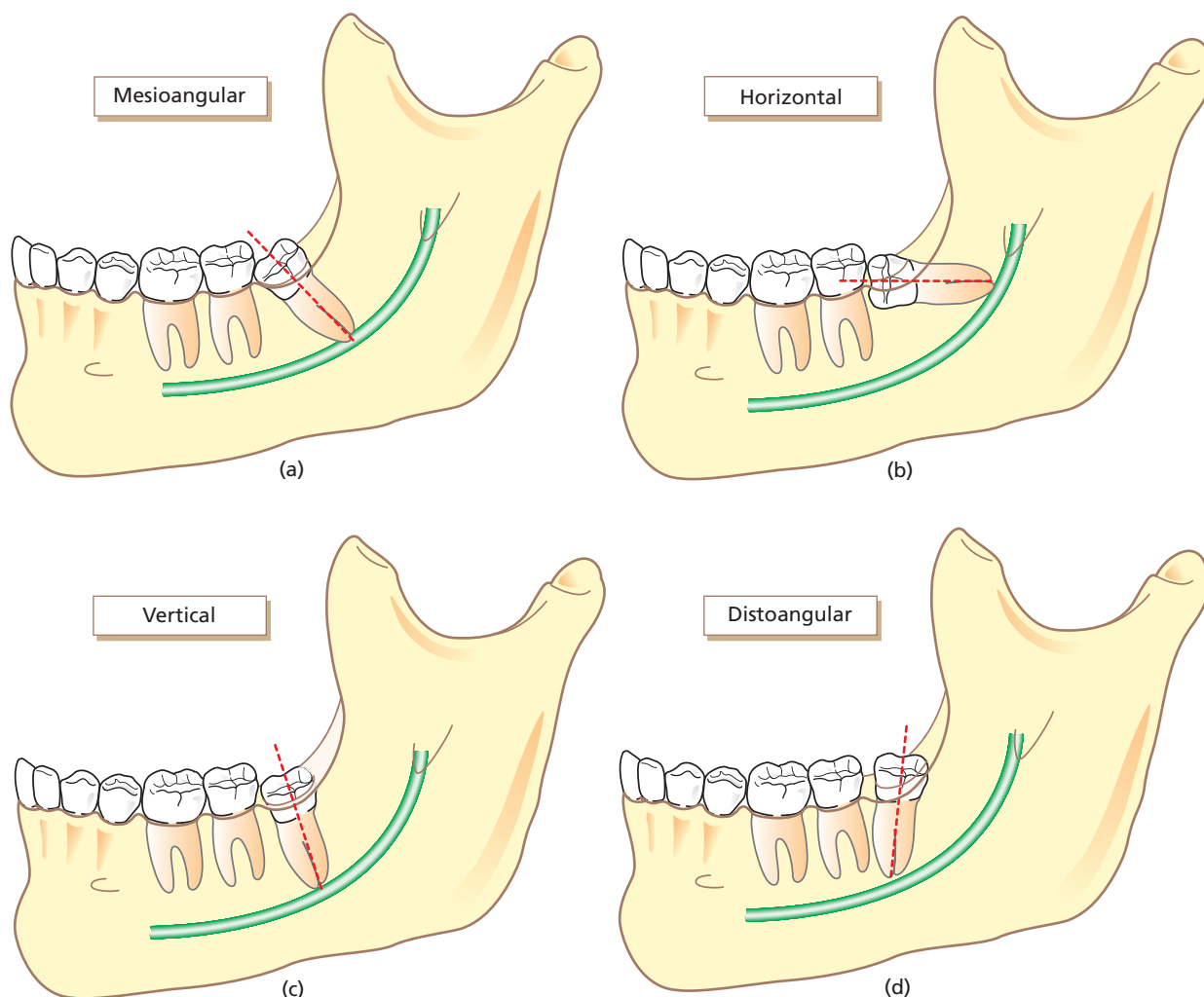


Fig. 13.1 The angular position of third molars according to Winter.¹¹

asymptomatic wisdom teeth is defined as the surgical removal of a third molar in the absence of subjective symptoms as reported from the patient as well as absence of local disease as registered by a dentist or an oral and maxillofacial surgeon.

Prevalence

Most studies on the prevalence of third molar impaction are based on patient materials from hospitals, dental schools, private practices, and army clinics.¹³ An ideal epidemiological study of disease or anomaly prevalence should be performed as a random sample of a well defined population, preferably representing the community as a whole. Such a study was performed by Hugoson and Kugelberg, and was based on 693 dentate persons aged 15–80 years from a random sample of individuals in a Swedish community.¹⁴ Approximately two thirds of individuals aged 15–80 years had between one and four third molars. Out of these wisdom teeth, 30.4% and 43.8% were partially or completely impacted in men and women respectively. In this study, the angulations of the impacted third molars were also presented and it was found that vertical angulation was most frequent (61.8%) followed by mesioangular (25.5%), distoangular (8.7%), and horizontal position (4.0%).

A suitable age for studying the frequency of third molars and their impaction should be about 20–25 years. In later age groups early extractions of third molars may escape notice and result in a too high frequency of individuals with no or lesser number of third molars. In a study of Swedish dental students¹⁵ aged 20–39 years (86% of them aged 20–29 years) it was found that 10% of the individuals lacked all third molars and half of them had all four third molars. One or more impacted third molars were found in 33% of the examined students. Calculated by number of teeth, 24% of all third molars were impacted, mostly in vertical or mesioangular position. Considering the prophylactic indication for third molar removal, these 24% of the third molars present in this age group represent primarily the third molars “at risk”.

It appears that the prevalence of third molar impaction increased during the second half of the 20th century. In a study from Finland¹⁶ it was reported that 29% of lower third molars were partially erupted in 1990 compared to 19% in 1949. Improved dental health in children and young adults, based on preventive dental programs, can be reflected in a marked decrease in first molar extractions. In modern man, with less attrition, this may result in a lack of space for normal third molar eruption. This epidemiological change in general oral health and third molar impaction has also been reflected in oral surgery practice where studies in the UK have shown a 40% decrease in routine extractions of permanent and deciduous teeth between 1974 and 1984, while surgical extractions increased by 113% during the same period.¹⁷

From the studies on the prevalence of third molar impaction one can conclude that:

- the most frequently impacted teeth are mandibular and maxillary third molars;
- at the population level, third molar impactions occur with a prevalence of 17–32%;
- in young adults, aged 20 years, 46% of the maxillary third molars were partially or completely impacted while the corresponding rate for mandibular third molars was 73%;
- females have third molar impaction more often than males;
- the prevalence of impacted third molar increased during the second half of the 20th century;
- the prevalence of impacted third molars indicates that health care resources allocated to third molar surgery are significant.

Public health aspects

Volume of surgery

Third molar removal is a high-volume surgical procedure. These operations can take place in general dental practice, both in the public health sector and in private practices, and by specialists in oral surgery in hospitals as well as in private specialist practice. In the UK, during the year 1989–90 almost 116000 patients in the National Health Service (NHS) and 22000 patients in the private sector had third molars removed.^{18,19} At this time removal of impacted teeth ranked number nine in the top ten list of procedures in the NHS and number one in the corresponding list of procedures covered by private medical insurers.²⁰ In 1989 oral surgeons in the public health sector in Sweden surgically removed 28000 third molars corresponding to 62% of all operations performed by those surgeons in that year.²¹ In Norway, with a population of 4.5 million, about 75000 impacted third molars were surgically removed in the early 1990s, about 75% removed by general dental practitioners (GPDs).²² In the US oral surgeons alone removed 2.25 million impacted teeth in 1977, the majority of which were third molars.²³ More recent statistics from the US report a marked increase in those procedures and in 1999 it was estimated that oral surgeons and GDPs removed 7 million and 3 million third molars respectively.²⁴ In a questionnaire reported in 2006, oral and maxillofacial surgeons in the US performed on average 53 third molar cases per month. With a variation of one to four third molars removed per patient and approximately 5500 practicing oral surgeons, it could well be that still at least 7 million third molars are annually removed by specialists.²⁵

On a population basis, an average of 281 third molar removals per 100000 people were performed in Sweden in 1989 by oral surgeons. The exact number of third molars removed by GDPs in Sweden is not known but has been estimated at around 1100 per

100 000 people. In Norway oral surgeons and GDPs together removed about 1600 third molars per 100 000 inhabitants. Toth¹⁸ estimated the third molar operation rate in the UK to vary from 250–680 per 100 000 people based on statistics from the South West Regional Health Authority in 1989.

A public health perspective also includes a valuation of how available health care resources are used. Regarding third molar surgery, it would be of interest to estimate the proportion of resources allocated to the removal of third molars associated with and without pathology. This is not easily assessed from the literature, mostly due to terminological inconsistencies when reporting indications for removals in surgical case series. Table 13.1 provides a summary of studies where the relative frequency of third molars removed, described as prophylactic removals, asymptomatic third molars or third molars without pathology are presented.

Costs

Large amounts are spent annually on third molar removals by public health authorities, health insurance companies, and patients. The total amount is very difficult to assess. From a public health aspect an estimation of cost for third molar surgery should include direct costs (health care facilities, professional salaries) as well as indirect costs (patients' loss of salary, travel expenses). When reported, the costs usually refer only to the direct costs. In 1989/90 the annual cost for third molar surgery in the NHS in England and Wales was estimated to be £23.3 million.¹⁸ In the US the costs for third molar removals in 1999 performed by both oral surgeons and GDPs were estimated to be \$3302 million.²⁴ Moreover, Friedman assessed that this volume of surgery involves about 5 million patients with an additional 11.3 million days of standard discomfort or disability postoperatively, an indirect cost that was not estimated in monetary terms.

Natural history of third molar impaction

Knowledge of the natural history of a disease, a condition or a symptom is a cornerstone in clinical decision making. In our case, the natural history of third molars includes descriptions of normal and disturbed eruption, prediction of third molar impaction, the prevalence of and risk for developing third molar-associated pathology, and reasons used for the removal of third molars. Most valid information of the natural history of a condition can be reached by following this condition in a well defined population for an adequate time – a longitudinal study. For an abnormal condition such as an impacted third molar, an expected follow-up time can be several years and even decades. In most circumstances this can be both extremely costly and impractical, and much of our knowledge is based on cross-sectional studies – different individuals studied at certain, different age-groups.

Eruption

If a third molar is considered erupted when any part of the crown has pierced the oral mucosa and become clinically visible, the mean age of eruption for lower third molars was 19 and 20 years of age for men and women respectively.³⁰ Completely erupted third molars in functional occlusion are very rare at the age of 20. When following the fate of non-erupted third molars in Danish dental students after the age of 20 in a 4-year clinical follow-up³¹ it was found that:

- none of the participating students had completely erupted third molars at age 20;
- 31% of the partially or totally impacted third molars at age 20 were completely erupted 4 years later;
- 38% of the third molars had been removed during the 4-year follow-up. The reason for removal was

Table 13.1 Data from surgically treated third molar patients. Relative frequency of third molars removed classified as asymptomatic, without pathology or as prophylactic extractions.

Study	Year	Country	Percentage of third molar removals classified as asymptomatic, without pathology or prophylactic extractions
Goldberg <i>et al.</i> ²⁶	1983	USA	32
Osborn <i>et al.</i> ⁴¹	1985	USA	32.9
Nordenram <i>et al.</i> ⁴²	1987	Sweden	40.3
Lysell and Rohlin ⁴³	1988	Sweden	42
Dunne <i>et al.</i> ²⁷	1995	UK (Scotland)	19
	2002		5
Knutsson <i>et al.</i> ²⁸	1996	Sweden	18
Garcia <i>et al.</i> ²⁹	1997	Spain	32.7
Bataineh <i>et al.</i> ⁴⁶	2002	Jordan	7.7
White <i>et al.</i> ⁸¹	2003	USA	78*

* To prevent future problems as stated by the patients.

mild clinical symptoms (e.g. feeling of pressure) in 39%, pericoronitis in 26%, caries in 4%, and a prophylactic indication in 31%.

In a similar study in Helsinki, Finland,³² the third molars of university students were followed clinically and radiographically for 6 years between the ages of 20 and 26 years. The main purpose was to study how many of the clinically invisible or partially impacted third molars at the age of 20 had erupted by the age of 26. It was reported that:

- at both baseline and 6 years later, upper third molars were more often erupted than lower ones;
- mandibular third molars completely impacted in soft tissue at the start of the study were more frequent in women (52%) than in men (31%);
- almost half of the third molars that were partially erupted at age 20 erupted during the 6 years' follow-up.

The findings of these two studies on university students were confirmed in a prospective cohort study in New Zealand³³ where the participants were followed from age 18 to 26. The conclusion of this study was that, with the exception of horizontally impacted third molars, a substantial proportion of other impaction types do fully erupt.

After the age of 25 there can still be some changes in eruption status of third molars, but these changes are insignificant compared to the more dynamic period for third molar development between 18 and 25 years.^{34,35} It can thus be summarized that third molars do continue to erupt after the age of 20 until 25–26 years of age. In the age group 20–26 years, in the absence of subjective symptoms or local disease, the strategy of choice for third molars seems to be observation rather than prophylactic removal.

Pathology related to third molars

The justification for routine removal of all third molars that do not reach functional occlusion is the belief that those teeth constitute an abnormality with a significant pathologic potential. Some 20–30 years ago, such a belief was supported by published case series of patients with serious pathologic consequences of third molar retention. While each case presents a convincing indication for surgery for that individual patient, the presentations were not balanced with the known or estimated prevalence of that particular condition.

There are three main kinds of studies that have shed some light on the prevalence of or risk for pathology related to non-erupted third molars. One type of study is the prospective follow-up of defined populations while other studies report data from surgically treated patient groups or otherwise defined patient populations. The most convincing evidence comes from prospective, long-term follow-up studies

of a defined population of individuals with non-erupted third molars at the baseline of the study. Preferably, persons to be included in such a study are around 20 years of age at baseline. To be sure that you will obtain relevant information, even for pathologic entities with low prevalence, the selected population has to be followed for 15–20 years and the number of individuals to be followed can be estimated to be at least 500–1000. Even if this suggested design is followed, one would certainly fail to detect those extremely low-frequency conditions which appear in middle-aged or older people. Knowledge of those entities is probably best found in data from series of surgically treated third molar patients. For many practical, logistic and financial reasons, an ideal prospective study designed as mentioned above has not yet been published.

Follow-up of defined populations

Except for the studies of third molar eruption in young adults mentioned above, the most ambitious follow-up study on clinical changes in third molar status in a defined population is that reported by Ventä and co-workers.^{36,37} They followed a population of 181 university students in Helsinki with clinical examination at the age 20, 26, 32, and 38 years. The clinical examinations were supplemented with panoramic radiographs at 20, 32, and 38 years of age. While the studies were mainly concerned with changes in third molar eruption status, it was also found that after 12 years, at age 32, 47% of the third molars were removed. At the age of 38, after 18 years' follow-up, 73% of the initially non-erupted third molars (soft tissue and bone impactions) were removed. For the initially partially erupted third molars, 64% were removed during the follow-up period. In the 12-year follow-up, it was however reported that 61% of the individuals with removals presented no symptoms while 35% of those with removals sought treatment because of symptoms. It was also shown that there was a peak in extraction frequency at 27–28 years of age. The aim of these studies did not include registrations of indications for those third molars removed, but it is quite plausible that a considerable number of those third molars without symptoms were, according to the current concept of indications at that time, removed prophylactically.

A longitudinal, 12-year follow-up was performed on a cohort of Swedish women aged 38–60 years at inclusion.³⁸ The participants were examined with panoramic radiographs at baseline as well as 12 years later. A total of 166 impacted teeth were found in 8% of the 1418 participating women. The majority of those impactions were third molars (85%). The findings of third molar-associated pathology in this study are summarized in Table 13.2. Most conditions were regarded as moderate (slightly widened follicle or resorption of the crown) and only in 1.2% of the

Table 13.2 Prevalence of third molar associated pathology in middle-aged and older women. From a 12-year follow-up study by Ahlqvist and Gröndahl.³⁸ In the baseline study, 8% of 1418 women had impacted teeth, 85% of them third molars. At the 12-year follow-up, 114 of the initially 166 examined impacted teeth could be re-examined. 18 impacted teeth had been removed during this period and 34 were found among those women not participating in the follow-up study.

	Baseline of study	12-year follow-up
Number of impacted teeth	166	114
Associated pathology		
Follicular cyst	1.2%	1.7%
Widened follicles (3–4 mm)	8.4%	7.9%
Crown resorption (third molar)	6.6%	8.7%

impacted teeth could a genuine follicular cyst be observed. At the 12-year follow-up unchanged conditions were found in 85% of the cases; 18 impactions had been removed, only two of them with associated pathology.

In a recent study from Cardiff, Wales, Hill and Walker³⁹ followed 228 persons with a total number of 427 third molars for a 5-year period. The median age at inclusion was 23 and the female:male ratio was almost 2:1. Of the 228 recruited persons, 150 remained symptomless during the 5-year follow-up. A total of 66 persons had a previous history of pericoronitis at inclusion and 23 of them had a third molar removed during the study. In addition to these 23 persons, another 48 had third molars removed during the study. The main indication for surgery in those cases was pericoronitis (30 out of 48 patients) while the other indications included caries in second molar, orthodontic treatment, food impaction, and pain.

Data from surgically treated third molar patients

Some valuable information can be gathered from studies on patients referred for third molar surgery. When one makes inferences from those data it is, however, important to recognize that they are biased for several reasons. For one thing, referral patterns differ between different countries due to varying degrees of specialization, financial organization of the health care system, and the socio-economic status of the population. The selection of patients may also vary considerably if the patients in the study represent referrals to private practitioners in oral surgery, a county hospital department, or a highly specialized university clinic. It is also evident that definitions of pathologic entities can vary, a fact which is quite obvious when you study the prevalence of follicular cysts.⁷

From the studies presented in Table 13.3 we can observe that there is a considerable variation in the prevalence of reported pathosis in surgically removed mandibular third molars.^{28,40–46} Pericoronitis as cause for removal varies from 6–53% of the cases and, in the

majority of the studies, was the most prevalent pathologic entity. Other diseases related to the third molars were less frequent, except for a deviating, high prevalence of caries on the second molar (42.7%) in the study of van der Linden and co-workers. Their findings were based on a retrospective study of panoramic radiographs and hence pericoronitis prevalence was not included. Resorption of the distal root surface of the second molar was noticed from less than 1% up to 6% of the cases. Although these studies represent third molars scheduled for removal, more serious diseases such as follicular cysts and tumors were less frequently reported. Cysts were generally found in the range of 3–6% of the operated cases. When tumors were diagnosed in relation to a third molar, the prevalence of such a pathosis was less than 1%. In the extensive surgical material from the university clinic of Oral and Maxillofacial Surgery in Ankara, Turkey, tumor was the indication for third molar surgery in 0.79% of the cases; 98% of them were odontogenic tumors and 2% were malignancies.

Other studies on dental patients

Some information on the natural history of third molar retention can be gained from cross-sectional studies on dental patients. Such patient populations differ of course from the surgical patient groups in that they are primarily not investigated clinically or radiographically because of a third molar problem or referral. It is also important to observe that patients, who have had third molars associated with clinical symptoms and obvious pathology, have already had these molars removed. Thus, symptoms and pathosis registered at various age-groups in these cross-sectional studies probably represent a minimum of possible pathologic sequelae to third molar retention.

Table 13.4 presents cross-sectional patient studies where the aim was to assess the possible complications. Stanley and co-workers⁴⁷ published a radiographic study on a large cohort of dental patients from a Veterans Administration Hospital and a university college of dentistry in Florida, USA. They reviewed the panoramic radiographs and dental and medical records of 11598 persons seeking dental treatment. The mean age of the patients was 47 years, and it was found that 1756 patients had 3702 impacted teeth (95.6% of them were third molars). Of the impacted teeth, about 8.8% showed some signs of pathology. Follicular cysts were present in only 0.31% of the impacted teeth. Presenting the radiographic changes on a per patient basis, a total of 12% had an impacted tooth with an associated radiographic lesion.

Another patient cohort was presented from the Karolinska Institute, Stockholm, Sweden by Eliasson and Heimdahl.⁴⁸ They examined radiographs of 644 randomly selected patients (mean age 42.6 years) with 1211 impacted third molars. Conditions like follicular cysts, resorption of second molar, and severe

Table 13.3 Data from surgically treated third molar patients. Prevalence of reported pathosis in surgically removed mandibular third molars.

Reference	Age of patients	Number of third molars	Pericoronitis	Caries, third molar	Caries, second molar	Resorption second molar	Apical periodontitis	Cysts	Tumors	Periodontal problems
Bruce <i>et al.</i> 1980 ⁴⁰	14–81	990	39.8	8.8	5.8	6		6	0.2	16.9
Osborn <i>et al.</i> 1985 ⁴¹	12–83	16127	6	1.9	0.3			0.3*		
Nordenram <i>et al.</i> 1987 ⁴²	16–90	2630	26	10.7		4.7		4.5		
Lysell & Rohlin 1988 ⁴³	10–>60	870	32	13	2	<1	<1	3	<1	
Van der Linden <i>et al.</i> 1995 ⁴⁴	13–75	2872		7.1	42.7	0.9	2.3	4.6	0	4.9
Knutsson <i>et al.</i> 1996 ²⁸	15–80	666	53	25.2	4.4	1		4.2	0	6.5
Güven <i>et al.</i> 2000 ⁴⁵	14–67	9994						2.3	0.79	
Bataineh <i>et al.</i> 2002 ⁴⁶	14–67	2252	46.8	23	0.5			1.6†		13.6

* Cysts and tumors.

† Cysts, tumors, and root resorption.

Table 13.4 Cross-sectional studies on dental patients. Prevalence of pathologic lesions associated with impacted maxillary and mandibular third molars.

Reference	Age of patients	Number of third molars	Resorption, second molar	Cysts	Periodontal problems	Study characteristics
Stanley <i>et al.</i> 1988 ⁴⁷	20–83	3702	3 (incl.caries)	0.8	4.5	Radiography, mandible and maxilla
Eliasson <i>et al.</i> 1989 ⁴⁸	>30, mean age 42,6	1211	0.9	4	4 (maxilla) 1 (mandible)	Radiography, retrospective

loss of marginal bone on the distal aspect of the second molars were found in 6.9% of the impacted third molars. Furthermore, 5% of these impactions showed crown resorptions.

From the three different kinds of cohort studies (follow-up of defined populations, third molar surgery patients, and cross-sectional studies on dental patients), the least reliable source of information on the prevalence of third molar-associated pathology in the general population can be derived from the surgical patient groups. The most promising result can be expected from prospective long-term follow-up studies of defined populations. Existing studies, however, lack in strength by having either too few third molars included or too short a follow-up time. From cross-sectional studies on dental patients we can observe that third molar impactions still present in middle-aged or older patients are associated with a rather limited prevalence of pathology (approx. 7–9%). There are also indications that impacted third molars without any signs of pathology in these age groups will be unchanged, with only a limited risk of becoming associated with serious pathology over a relatively long period of time. One conclusion for clinical decision making may be that relatively long intervals can pass between radiographic re-examinations of disease-free, asymptomatic impacted teeth found in those age groups. It is also important to observe that the majority of the studies were based on radiographs. Clinical findings such as chronic pericoronitis, food impaction, incipient caries, and periodontitis were not recorded.

Impaction status and angulation of the third molar

It is not an unexpected finding that the risk for developing associated disease varies with the type of impaction of the third molar. Obviously, third molars partially covered by soft tissue have a much higher exposure to local disease-inducing factors than those third molars totally covered by soft tissue or those completely embedded in bone. In a study of the mandibular third molar position as predictive criteria for risk for pericoronitis in young adults⁴⁹ it was found that 97.5% of the pericoronitis cases were partial soft tissue impactions and only 2.5% were fully impacted in soft tissue. This higher risk for pathology associated with partial soft tissue impaction was also confirmed in studies on other patient cohorts. In the studies of Ventä *et al.*⁵⁰ and Knutsson and co-workers,²⁸ third molars partially covered by soft tissue presented a much higher risk for pathology than third molars totally covered by soft tissue or bone (odds ratio 6.4 and 6.7, respectively). On the other hand, pathologic entities such as follicular cysts are only associated with impacted teeth totally covered by soft tissue or bone.

When estimating the risk for pathology, it is reasonable to combine the eruption state and the angular

position of the third molar. Leone *et al.*⁵¹ described the tooth at highest risk for acute pericoronitis as a vertical or slightly distoangular positioned mandibular third molar that is in contact with the second molar, at or above the occlusal plane, and partially covered by soft and bone tissue. In contrast, Ventä *et al.*⁵⁰ found that the most common mandibular third molars that gave rise to acute problems (mostly pericoronitis) were distoangular-oriented and partially embedded in soft tissue. The odds ratio for development of pathology was, according to Knutsson *et al.*²⁸ about 5–12 times higher for distoangular third molars compared to other angular positions. They found that mesioangular molars presented the lowest odds ratio, 0.5, compared to 5.8 for the distoangular-oriented third molar.

General aspects on clinical decision making

The quality of health care in every clinical specialty depends mainly on two factors: the quality of the judgements and decisions on what to do, and the quality of how these interventions are performed practically. Interventions in this context may be a decision to perform a diagnostic test, a surgical procedure, administration of a medication, or a decision to “wait and see” the natural outcome of a symptom or clinical finding. Thus, it is easy to appreciate that everyday clinical work involves a lot of judgements and decisions. For surgeons, it is important to recognize that in spite of an excellently performed surgical operation, if it is based on a poor decision, the outcome for the patient may be suboptimal or even harmful. One also has to acknowledge that clinical decisions create costs: (1) monetary costs for health care providers, society, insurance companies and patients; (2) for patients, in addition to the financial burden of the intervention, there are also the costs of the morbidity of that particular disease and intervention. Therefore, in an era of limited health care resources, research that promotes the quality of clinical judgements and decisions is of importance for clinicians as well as for health care administrators, who all are under pressure to provide medical and dental care cost-effectively. Research on and implementation of clinical decision making is an expanding field which demands contributions from different fields of science such as medical and dental specialists, psychologists, economists, decisions theorists, and statisticians. For those readers who want to develop their interest in this area of health care, a review edited by Chapman and Sonnenberg⁵² is recommended.

Judgement analysis

A clinician's decision on a specific diagnosis or recommendation of a specific intervention is preceded by judgements on a number of cues characterized

by uncertainty. Such judgements can be interpretation of laboratory data, radiographs, findings on a physical examination, and soft data from interview of the patient. Excellence in clinical work is to combine the judgements of different cues to a decision with a high accuracy to a "true state", e.g. the outcome of the intervention or the confirmation of the diagnosis with a recognized "gold standard". It has, however, been shown that there is a substantial variation in judgements in almost every aspect of clinical practice. In a review article, Eddy states that *"in general, observers looking at the same thing will disagree with each other or even with themselves from 10% to 50% of the time"*.⁵³

Social judgement theory (SJT) as described by Brehmer⁵⁴ in 1988 has inspired a lot of studies on how clinicians and other professional decision makers combine and integrate probabilistic information. Judgement analysis is a descriptive approach to analyze medical decisions and Brunswik's lens model (Fig. 13.2) is a conceptual model commonly used in such studies as described by Wigton in 1988.⁵⁵ Brunswik used the analogy of rays of light passing through a convex lens to describe the relationship between the interpretation of information (cues) and the actual relationship of those cues to the true state. Judgement analysis has been applied in several areas, such as medicine, psychology, education, social work, and business. In the medical domain, several aspects of clinicians' judgement have been studied, both in the process of making a diagnosis or prescribing an intervention. Reviewing some of these studies Wigton⁵⁶ concludes that a most important finding was the surprising degree of variation in the judgement of experts in medicine. He also concludes from the policy-capturing studies that cues given considerable weight by some clinicians may not be recognized

as important by other physicians or by textbooks. A series of important papers on professional judgement, many of them on clinical decision making, has been assembled by Dowie and Elstein.⁵⁷

Judgement analysis and third molar surgery

With the Brunswik lens model as a conceptual model, Brehmer, Knutsson, Lysell, and Rohlin^{58–65} published a series of papers on dentists' judgement in the removal of asymptomatic mandibular third molars without associated pathology. The studies were based on 36 mandibular third molars referred for removal and were selected with the aim of representing an equal distribution of males and females, of different age groups of patients, as well as of different angular positions and degree of impaction of the third molars. Each case was presented to the clinicians participating in the study as a radiograph and a questionnaire. Next to the radiograph a short text informed the participant about the sex and age of the patient and the degree of impaction of the third molar. The clinicians were asked to decide whether or not to remove the third molar in question, and furthermore to establish the degree of confidence with which they judged the need for removal (named the indication index) on a 100mm visual analog scale (VAS). For each third molar presented the participants had to estimate the probability for development of pathology in general and for six specified pathologic entities, also on a 100mm VAS.

Initially, 40 clinicians from Sweden were included in the study, 30 general dental practitioners (GDPs) and 10 oral and maxillofacial surgeons (OMFS). In a later part of the study another 18 GDPs and 10 OMFS from Wales were included for a comparative study between dentists' judgement on preventive third

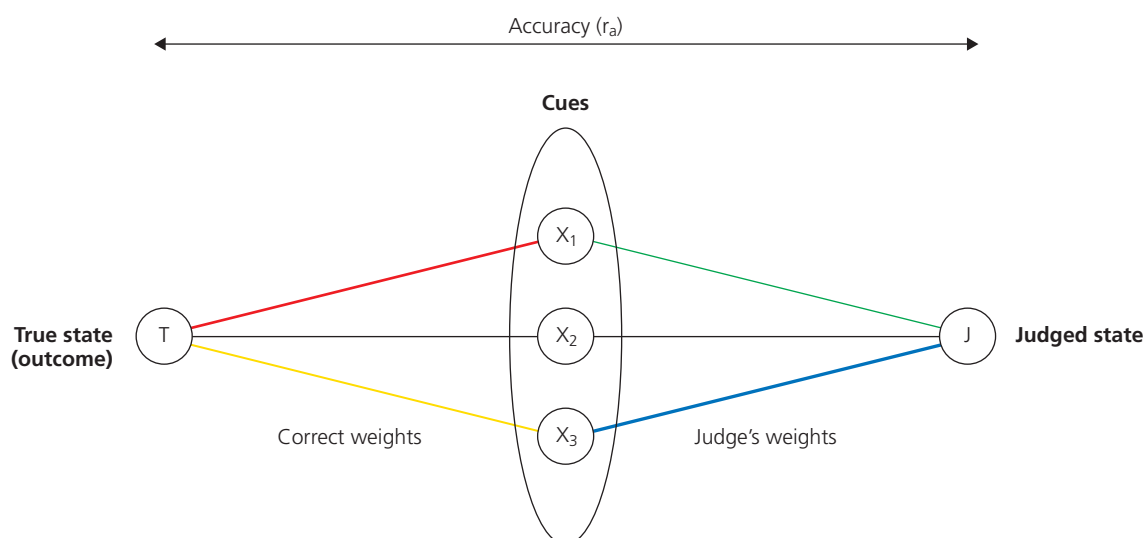


Fig. 13.2 Brunswik's standard lens model, copied and adapted from the papers by Brehmer,⁵⁴ Wigton *et al.*,⁵⁵ and Knutsson.⁶⁵ The cues can be clinical symptoms or signs in a diagnostic or therapeutic judgement. A linear model of the judges (clinicians) is calculated from repeated judgements on the right side of the lens, while a model of the true state (or the outcome) is calculated from repeated observations of real outcomes on the left side. Accuracy (r_a) is represented by the correlation between the judgements and the actual outcomes.

molar surgery in Sweden and Wales. The paragraphs below summarize what we have learned from those studies:

1. There were substantial variations, both among individual GDPs and OMFS, in the decision whether or not to remove asymptomatic mandibular third molars. The number of molars the 30 GDPs proposed for removal varied between 0 and 26 of the 36 presented cases. For the OMFS the corresponding figure varied between 3 and 21 third molars. There was no third molar among the 36 cases that all observers in the two groups agreed should be extracted. However, the OMFS were unanimous in the judgement *not* to remove 11 of the 36 third molars.
2. There was no significant correlation between the level of professional experience and the judgement whether or not to remove asymptomatic third molars either among GDPs or OMFS as a group.
3. The VAS is an appropriate tool for rating an indication index in judging the need for removal of asymptomatic third molars. There was no overlap of the 95% confidence limits for third molars proposed for removal versus third molars not proposed for removal.
4. As part of the lens model concept, multiple linear regression analysis was performed to determine the relative weights of the specific pathologic entities given by the participants in their judgement of the perceived likelihood of development of pathology in the asymptomatic third molars. The GDPs estimated the highest weights to cyst formation and pericoronitis, whereas the OMFS gave pericoronitis the highest weight and considered caries in the second molar as important as development of a follicular cyst. The risk for neoplastic changes and root resorptions on the second molar received low weightings. For certain entities, such as cyst formation, the weights given by individual participants varied widely from almost zero to very high weightings.
5. The influence of the three cues, age of the patient, angular position of the third molar, and impaction status of the third molar, on the judgement to remove an asymptomatic third molar, showed a considerable variance regarding individual judgements. Third molars partially covered by soft tissue were given the highest indication index for removal by both groups of clinicians. As a group, GDPs estimated the need for removal to be higher for patients below 40 years than those above this age. OMFS, however, estimated the need for removal to differ for the three different age groups (19–25 years, 26–40 years, and 41–60 years), giving the highest indication index to the youngest age group and the lowest for the oldest age group. Overall, the mean proportion of variance explained by the three cues was high, indicating that the clinicians used the three cues and the combination

of them to a great extent in their judgement of the risk for development of pathology that can be associated with an asymptomatic third molar.

6. Studying how the participants integrated the cues as compared with existing evidence in the literature showed a wide variation in judgement strategies. Only one person, a GDP, integrated the three cues according to the evidence.
7. In a comparison between clinicians in Sweden and Wales no evidence was found in mean numbers of third molars scheduled for removals by the GDPs. The Swedish oral surgeons, however, scheduled significantly more third molars for removal than the oral surgeons in Wales. This difference was interpreted as a result of the development of authoritative guidelines in the UK.

Judgement analysis does not bring new information on *why* and *when* a third molar should be removed. This method can be used to characterize how dentists weigh clinical information in reaching a judgement of intervention as well as non-intervention. The result of those studies can also be of advantage in undergraduate as well as postgraduate education to give students awareness of the difficulties in probabilistic judgements. The results can also reduce an often prevalent overconfidence in holistic judgements and encourage students to compare their own judgement strategies with those of their fellow students and with published results and existing evidence.

Decision analysis

Decision analysis is an approach that combines clinical data mathematically for developing an explicit decision making under uncertainty. As written by Dowie and Elstein:⁶⁶

Decision analysis decomposes any decision problem into discrete components and provides a procedure for synthesizing these components into an overall measure of the attractiveness of each possible action – its expected utility – so that the *optimal* strategy can be selected.

Decision analysis has been used for diagnostic as well as for treatment and prognostic decisions in medicine for decades^{67–70} and its use in dentistry has been thoroughly reviewed by Rohlin and Mileman.⁷¹ Decision analysis is described in detail elsewhere⁷⁰ but for those readers not familiar with the method a brief review will be given.

A decision analysis starts with a description of the decision problem faced by the decision maker. Currently, it is unrealistic to make separate decision analyses for individual patients and for that reason the decision problem has to be relevant for groups of patients with similar clinical features. The major components of decision analysis are the structure of the model of the decision problem (commonly using

a decision tree, Fig. 13.3, as a visual representation of the decisions available), assigning probabilities to the outcome events incorporated in the model and assigning utility values to each outcome. A utility value represents the strength of a patient's preference for one outcome over others. Utility values can reflect several dimensions of an outcome, for example life expectancy, quality of life, morbidity of the intervention, and costs. In this way it has been suggested that decision analysis reconciles evidence-based medicine with patients' preferences. In many clinical decision problems the assignment of utility values is *per se* a complex research process where people or patients have to assess the different dimensions of an outcome in order to arrive at a single index value for a future health state. Often the best health outcome is given the index value "1" and the worst possible outcome is given "0". In some more basic decision analyses patients' preferences are substituted with the decision makers' own estimation of morbidity and costs. Finally, when the expected value of each decision option has been calculated, a sensitivity analysis is used to determine the robustness of a choice made using this method. This is done by repeating the analysis and at the same time varying the outcome probabilities and utility values within clinically realistic limits to see how easily a decision would change – in other words to see how sensitive it is.

Even if decision analysis has not yet been incorporated in the face-to-face consultation between doctors and patients it is an increasingly used research tool, at least in medicine, and it also helps the clinicians to inform, in a more explicit way, patients of possible choices and their associated benefits and harms.

Decision analysis and third molar surgery

Two research-groups, Tulloch *et al.*^{7,23} and Brickley *et al.*^{6,73} have used the decision analysis approach to calculate the optimal strategy for the management of the mandibular third molar. The design of their studies varied in how probabilities of outcomes were estimated and also in their assessment of utilities. The conclusions of their studies did not, as we shall see, differ.

Tulloch *et al.* considered what treatment recommendations should be made by clinicians to healthy young adolescents with an incompletely developed or erupted mandibular third molar. In the study they compared three alternatives: (1) extract each third molar prior to complete root formation; (2) delay the decision to extract until the third molar has no further potential for eruption, then extract only those third molars that remain totally or partially impacted; (3) extract only those third molars that become associated with some type of pathology. The outcomes they considered are the disability and costs associated with the pathology, surgery and complications of each strategy. The probability of different pathologic entities was obtained from the literature, while the probability of complications with surgical extractions related to the type of impaction, and presence or absence of pathology were estimated by nine oral surgeons in a so-called Delphi process. Each complication was assigned an equivalent number of "days of standard discomfort" (DSD) and the valuation of the DSD was performed by 46 randomly selected dentists. A detailed calculation of costs was performed, including surgeons' fees, radiographic examinations, operating room facilities, anesthesia, postoperative medication, and follow-up visits.

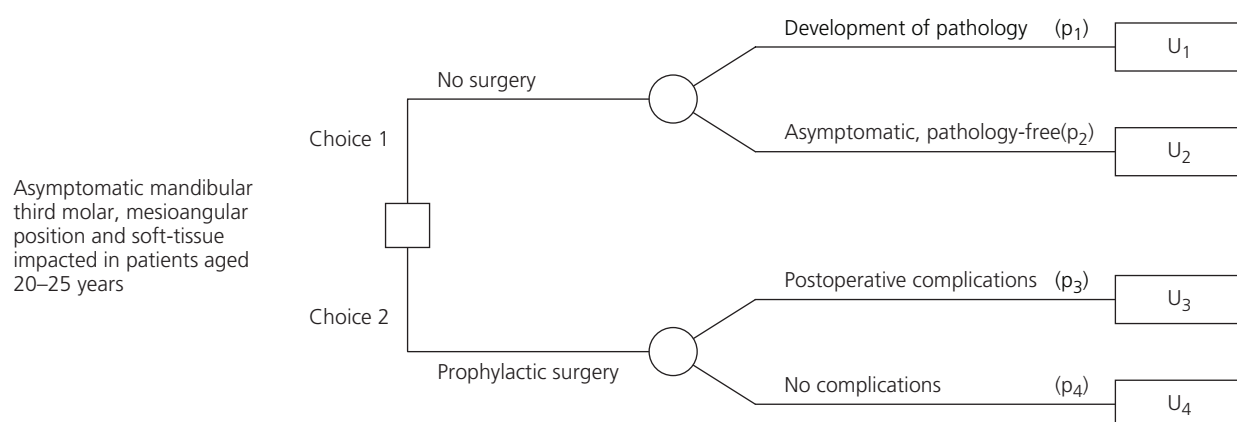


Fig. 13.3 Structure of a decision tree. A basic decision model starts on the left-hand side with a *decision node* (square). This node indicates the fundamental decision to be made, in this example prophylactic third molar removal versus watchful waiting. *Chance nodes* (circles) describe various events that are subject to chance and are not under the control of the decision maker. Each possible outcome is associated with a certain probability that it will occur (p_1 through p_4). All of the probabilities at a specific chance node must sum up to 1. Each branch in the decision tree ends with a *terminal node* (boxes) representing the *utilities* (U_1 through U_4) of ending up in a particular outcome state. In a decision problem with only two choices, each with two possible outcomes, the *expected value* of Choice 1 is simply the sum of the possible outcomes of that choice weighted by the probabilities of each outcome: $p_1U_1 + p_2U_2$. Consequently, the *expected value* of Choice 2 is: $p_3U_3 + p_4U_4$. The branch with the highest value at the decision node is the recommended choice.

Finally, the expected outcome for each of the three treatment strategies was expressed as the sum of all possible costs and disabilities under that specific strategy. The result was an expected cost (in 1985 US dollars) and DSD for each strategy and should be interpreted as the cost per person if that strategy was universally applied. They found in this calculation, including a sensitivity analysis, that strategy 3, only extractions of third molars associated with pathology, was the optimal strategy with the lowest expected costs and disability.

Brickley *et al.*⁷³ performed a decision analysis with the alternatives: extract or leave the third molar *in situ*. The probabilities of the outcomes were estimated from a comprehensive literature review and a clinical audit. The utility values were based on data from 104 individuals who rated on a VAS their perceptions of different health states resulting from extraction of as well as leaving a mandibular third molar *in situ*. The vignettes that were used to describe the health states included descriptions of possible complications of surgery and different pathologic entities that could develop in association with third molars left *in situ*. They did not, as Tulloch and co-workers, include the monetary costs for the two strategies.

When performing the decision analysis they found that the maximum expected utility of prophylactic third molar surgery was lower than that for non-intervention. Sensitivity analyses showed that the probability for recurrent pericoronitis has to be greater than 0.52 (52%) before the optimal decision would alter from non-intervention to surgical removal. The corresponding threshold value, e.g. the probability value at which the optimal decisions changed from non-intervention to surgery, was found to be 0.34 (34%) for a cystic change.

The important contribution of the study by Brickley and his team is their inclusion of patients' value when assessing the expected utilities. Both research groups integrated what was then the current knowledge of impaction prevalence, disease prevalence, probability of complications after surgery, and risk for future pathology in case of non-intervention as reported in the literature or by some kind of clinical audit. Some of those estimations were impaired by great variations. Because decision analysis is laid out in a formal and explicit way, the method has at least the advantage of identifying areas where evidence is absent or scarce.

Patient preferences

Decision making in medicine and dentistry involves evaluating outcomes in relation to both patients' and clinicians' preferences and values. An evaluation must be made whether the benefits of a certain intervention outweigh the physical, social, and emotional discomfort as well as the risk of serious complications. The last 20–30 years have witnessed a radical change of power in clinical decision making from a

more paternalistic approach from health care professionals to a situation of shared responsibility between patients and professionals for treatment decisions. At one extreme, patients have been described as informed consumers making decisions on what treatment to purchase, reducing health professionals to technical providers of certain services. A more common and realistic scenario is that patients want to be well informed and have an active role in the discussion of possible treatment choices. This demands an improvement in the quality of information given to patients but also a deeper understanding by clinicians of patients' preferences for the various outcomes of different interventions. Individual decision making, and also development of guidelines for specific disease entities and formulation of health care policies on a societal level, have created a need for a more explicit assessment of patient preferences.

Utility assessment is the technique most often used to evaluate patient preferences in clinical decision making. In medical decision making the most frequently used methods of assessing utilities are the Standard Gamble, the Time Trade-off, and VAS. The pros and cons are well reviewed by Stiggelbout.⁷⁴ Utility values are often generated by groups of patients with a similar clinical condition, and are preferably used for decisions at the group level, i.e. for development of clinical guidelines. When making decisions for the individual patient, the purpose is not to use absolute utility scores, but they can, however, be useful to help a patient to clarify his or her values. Utility assessment is a complex cognitive process as it involves rating different health states when faced with uncertainty. It is also important to remember that a certain health state involves physical as well as social and emotional components. In the case of third molar surgery, it means that clinicians should not only consider the probabilities of well known complications, such as postoperative alveolitis, nerve injuries, and bleeding complications, but also incorporate the influence on patients of the effects of a normal postoperative course such as pain, swelling, and trismus. Furthermore, Stiggelbout states that "*the elicitation of utilities for transitory, non-chronic health states is more complicated than for chronic, stable states*".

One methodology, the multi-attribute utility (MAU), as described by Torrance *et al.*⁷⁵ allows incorporation of both medical and non-medical factors in utility assessment and has been applied in studies of patient preferences on the removal and retention of mandibular third molars.^{76–78} The process is time-consuming and comprises three stages:

1. Elicitation of domains. These are main areas, such as general well-being, social life, work or studies, and economic situation, which patients generally consider to be important in their lives in relation to the intervention or disease under investigation.
2. Intra- and interdomain weighting.

3. Rating of outcomes. Different outcomes are often presented in short vignettes describing various health states related to a particular disease or intervention.

A preference value (or utility) for each vignette/health state and each patient is calculated using a special formula incorporating the result of each of the three stages. For the interested reader, the MAU-process is presented in detail in relation to third molar removal or retention by Liedholm and co-workers.⁷⁷

Using the MAU methodology, patient preferences were assessed by similar groups of patients in Sweden and Wales referred for removal of one or both mandibular third molars. The participants were recruited after a consultation with an oral surgeon but before

any intervention was performed. None of these patients had any earlier experience of third molar surgery. The preference or utility values and the relative ranking of the 18 different health states are presented in Table 13.5. There was an overall agreement between patient preferences and their ranking in the two countries. Generally, the results indicate that patients estimate that third molar removal had a more negative impact on their lives than a strategy of non-removal. Of interest for the practicing surgeon is the negative impact of postoperative pain on the preference valuation. When third molar surgery has to be performed, a sufficient analgesic regimen must be prescribed to obtain optimal patient satisfaction. The results also indicate, e.g. in the case where the vignette suggested a follicular cyst and patients in Sweden

Table 13.5 Patients' preferences in Sweden and Wales when 55 patients in Sweden and 100 patients in Wales were asked to imagine the outcomes of 18 vignettes. Ranking (R) 1 represents the least annoying and ranking 18 the most annoying outcome. There was a high degree of correlation between the two countries ($r_s = 0.93$, $P < 0.001$). From Liedholm *et al.*⁷⁸ Reprinted with permission from Macmillan Publishers Ltd, copyright 2005.

Vignette	Wales		Sweden	
	Mean	R	Mean	R
1 After the extraction you experience moderate pain and your face and mouth are very swollen	51.7	11	55.5	10
2 After the extraction you experience severe pain which keeps you awake for a few nights	37.3	17	38.8	16
3 After the extraction your jaw is stiff and painful. You have difficulty opening your mouth as wide as normal	55.9	9	43.5	15
4 After the extraction you experience minor discomfort and abnormal bleeding from the socket for a few hours	60.8	7	56.2	9
5 After the extraction you experience minor discomfort and also tingling and prickling sensations in your lower lip or tongue for up to 2 weeks	56.3	8	59.9	7
6 After the extraction you have minor discomfort. Your tongue is completely numb on one side for up to 6 weeks	50.5	12	52.5	11
7 After the extraction you have minor discomfort. Your tongue is completely numb on one side permanently	44.1	14	46.1	13
8 After the extraction you have minor discomfort. Your lower lip (but not your tongue) is completely numb on one side for up to 6 weeks	55.0	10	56.9	8
9 After the extraction you have minor discomfort. Your lower lip (but not your tongue) is completely numb on one side permanently	48.2	13	49.2	12
10 After the extraction you have minor discomfort. Your lower lip and your tongue are completely numb on one side for up to 6 weeks	42.8	15	45.9	14
11 After the extraction you have minor discomfort. Your lower lip and your tongue are completely numb on one side permanently	40.5	16	38.3	17
12 After the extraction the surgeon tells you that your jaw is broken and that you need your teeth wired together for up to 4 weeks	35.9	18	15.5	18
13 A tooth comes up at the back of your mouth. On one occasion this gives you a few days of aching pain and around the tooth the gum is swollen	85.0	2	78.8	2
14 A tooth comes up at the back of your mouth. Every six weeks or so you experience a few days of aching pain and around the tooth the gum feels swollen	73.8	5	70.4	3
15 The position of the wisdom tooth causes "gum disease" in the tooth in front which will make losing it more likely	71.8	6	64.9	6
16 You have a new tooth, which comes up, behind your last lower tooth. This results in the tooth in front becoming decayed and needing extraction	82.7	4	68.5	5
17 You notice that the appearance of your lower front teeth has changed slightly. The teeth appear to be more "squashed" together	83.7	3	80.5	1
18 After having an X-ray to find out if your wisdom tooth is present, the dentist tells you that there is a fluid-filled sac around your wisdom tooth and that this tooth must be removed	85.1	1	69.6	4

gave this health state the highest ranking (that is least negative effect on patients' life), that patients and professionals may interpret the seriousness of a certain health state quite differently.

In a shared decision on third molar removal an important aspect to discuss is the preferences of patients who had experienced third molar surgery. Such information is of value for the patient deciding on third molar removal, especially in cases of asymptomatic third molars or when prophylactic third molar removals are considered, and of course also to clinicians providing informed consent. Postoperative morbidity presented as biomedical measurements of swelling, trismus, and pain relief from different analgesic regimens has been extensively covered in the literature, while the issue of quality of life after third molar surgery has only started to attract researchers during the last decade. In 1996, when Shugars *et al.*⁷⁹ presented an instrument to measure patients' perceptions after third molar surgery they found that patients "*consistently talked about how their social activities were limited during the first 3 days after surgery*". The authors concluded that some kind of scale for ranking social activity and work have to be included when assessing the quality of life after this kind of surgery. They developed and modified this health-related quality of life (HRQOL) designed for the short-term outcome of third molar surgery and conducted a study with 249 patients.⁸⁰ The patients had to record their perceptions on four main areas each postoperative day for 14 days. These main areas, or domains, were pain, oral function (mouth opening, chewing, and talking), general activity (routine daily activities, recreation, sleeping, and social interaction), and other symptoms (bleeding, bruising, swelling, nausea, food impaction in surgical sites, and halitosis).

Using the same HRQOL instrument White *et al.*⁸¹ reported clinical as well as HRQOL data on the recovery of 603 patients who all had four third molars removed. This was a prospective study where patients were enrolled both in academic clinical centers and in community specialist practices. The follow-up for recovery was 14 days. Another research team in Dundee, UK⁸²⁻⁸⁴ developed a similar third molar outcome scale, PoSSe (postoperative symptom severity scale), which was validated by comparing it with a well validated measure of health, the anglicized version of the SF-39 health survey questionnaire. They found that the PoSSe-instrument was a reliable, valid measure of postoperative symptoms after third molar removal and of the impact of those symptoms on the perceived health of the patients. A brief summary of those studies reveals that:

- Social life was substantially affected during the first 3 postoperative days. A return to a more normal function was registered at postoperative day 5.
- Recreation and daily activities (work/studies) followed closely the development of social activities and was near normal 1 week after surgery (Fig. 13.4).
- Almost half of the patients experienced some pain 1 week postoperatively. One can expect almost all patients to take analgesic medication the first postoperative day and 1 week after surgery almost half of the patients were still in need of pain medication (Fig. 13.5).
- Bleeding and nausea were only a concern to patients during the first day after surgery.
- Chewing, mouth opening and swelling required 6, 5, and 4 days to attain minimal or normal levels.

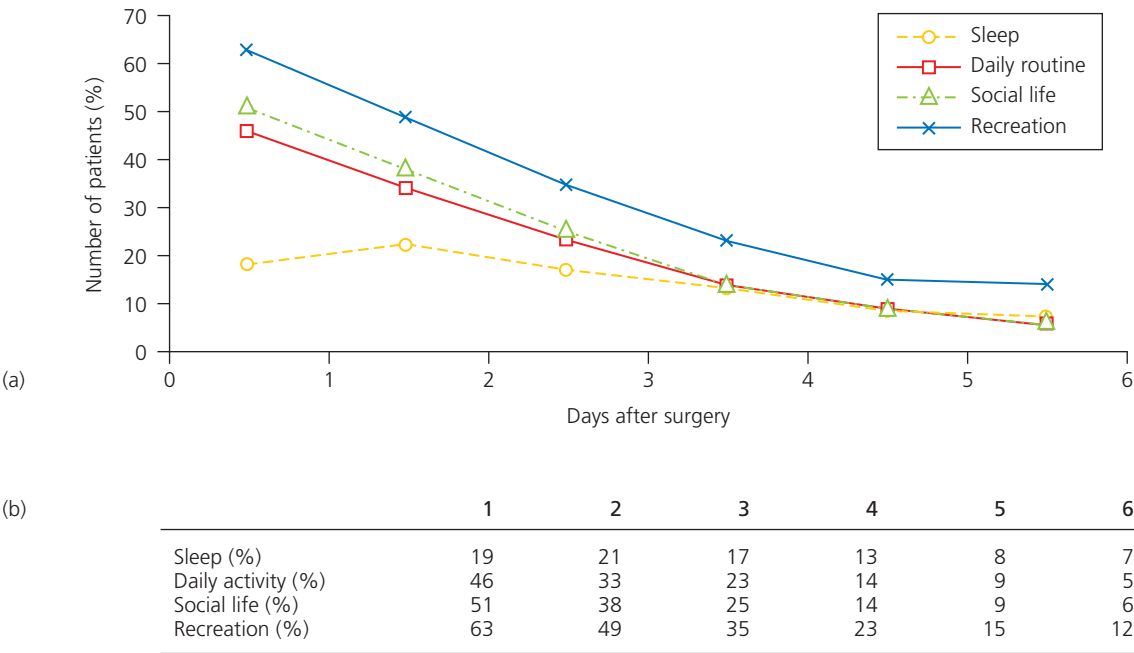


Fig. 13.4 (a, b) Percentage distribution of patients ($n = 547$) indicating that third molar surgery interfered "quite a bit/lots" with lifestyle. From White *et al.*⁸¹ Copyright © 2003 Elsevier.

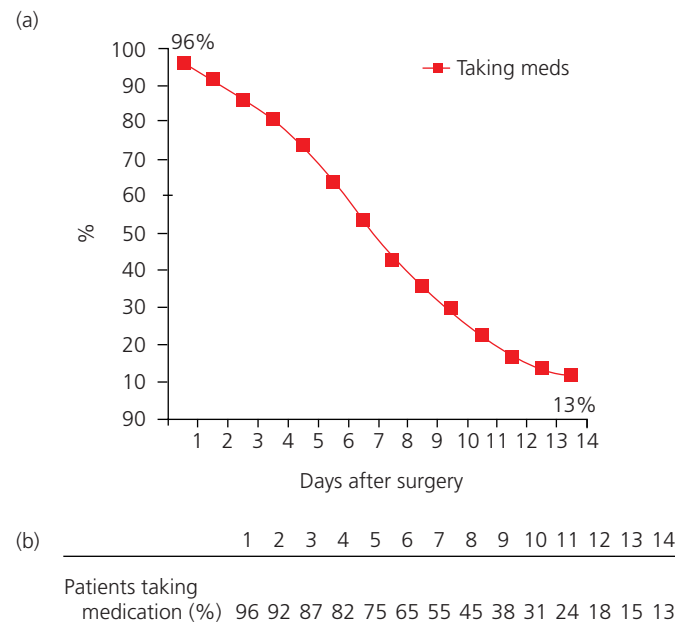


Fig. 13.5 (a, b) Percentage of patients taking analgesic medication after third molar surgery. From White *et al.*⁸¹ Copyright © 2003 Elsevier.

- Postoperative swelling was at its maximum during postoperative day 1 and 2 and had almost disappeared on postoperative day 5 to 6.
- Food collection and halitosis were still a problem for 15–20% of the patients on postoperative day 7.
- When comparing the perceptions of the impact of third molar surgery on the quality of life between patients, GDPs, and oral and maxillofacial surgery consultants, they were found to differ in some aspects. GDPs appeared to overestimate the impact of pain while patients ranked interference with daily activities and problems with chewing and enjoyment of food as more disturbing in the first postoperative period. Oral surgeons were in closer agreement with their patient; however, like the GDPs they also overestimated the impact of pain.

Of equal, if not of more importance, is the question of whether third molar surgery can improve the quality of life as perceived by patients in the long term. This is of course a more serious question when asymptomatic third molars or prophylactic removals are discussed. Is it realistic to assume that patients appreciate that the disability of the postoperative 7–14 days, together with estimated risk of complications, can be compensated by the assurance that third molar-associated pathology is eliminated? It is not an easy question from a researcher's perspective, as it includes the use of valid and reliable quality-of-health measures, both general and oral health-related, as well as cultural perceptions of health, risks and preoperative characteristics such as age, medical status, and severity of third molar pathology. There are indications, as presented in a study by McGrath *et al.*,⁸⁵ that patients perceive an "oral health gain", an improvement in oral health 6 months after third molar removals. This was more pronounced in patients having preopera-

tive symptoms (previous pericoronitis) compared to patients with asymptomatic third molars.

There are certain problems and limitations to extracting and analyzing relevant information of patients' perception of the postoperative quality-of-life after third molar surgery. Not only do outcome scores differ, so do surgical protocols. One study included patients where all four third molars were removed⁸¹ while patients in another study⁸³ had from one up to four third molars removed at the same surgical session. While one study reported only on patients operated on under general anesthesia⁸² another included procedures performed under local as well as intravenous anesthesia.⁸⁰

Indications for third molar removal

Therapeutic indications

There is a general agreement on well defined criteria for third molar removal that were the result of the consensus conference held by the National Institutes of Health (NIH) in 1979.⁸⁶ It was recommended that fully or partially impacted third molars should be removed when there is evidence of pathological changes. The criteria included infection, non-restorable carious lesion, cyst, tumor, and destruction of adjacent tooth and bone. Beyond those criteria it was also agreed that impaction or malposition of a third molar is an abnormal state and may justify its removal and that such treatment is not considered "prophylactic". Although there was no consensus on removal of asymptomatic impacted third molars without any evidence of pathology, the conference stressed the need for long-range studies on this subject. Furthermore it was agreed that enucleation of third

molar buds in young age groups was not acceptable and that there was little rationale for the removal of third molars solely to minimize present or future crowding of lower anterior teeth.

Following the NIH consensus conference, and inspired by its conclusions and suggestions for further research, quite a number of research papers and review articles have been published. Reports on clinical decision making, public health aspects on third molar surgery, and research aiming to assess the prevalence of third molar associated pathology and the natural history of third molar retention, referred to earlier in this chapter, are good examples. The reviews of Stephens *et al.*,⁴ Mercier and Precious,⁸⁷ Daley,⁸⁸ and Adeyemo⁸⁹ are also helpful. Since there is a general agreement on the indications for surgery when a third molar is associated with pathology, the controversy that still remains is over the treatment of asymptomatic third molars and the practice of prophylactic third molar removal.

The National Institute for Health and Clinical Excellence (NICE) is an independent organization in the UK established by the government to encourage evidence-based practice in medicine and to produce guidance on treatment and care of people with specific diseases and conditions within the NHS. In 2000 NICE published a "Guidance on the Extraction of Wisdom Teeth" with recommendations that are more or less in line with the criteria from the NIH consensus conference in 1979.⁹⁰ Summing up these two publications produces a list of appropriate indications for third molar removal:

- recurrent or severe pericoronitis;
- periodontal disease with a pocket depth of 5 mm or more distal to the second molar;
- non-restorable caries in the third molar;
- resorption of the third molar or adjacent tooth;
- caries in the second molar where the third molar removal would render restoration possible or more simple;
- apical periodontitis;
- cysts or tumors associated with the third molar (or adjacent tooth);
- when required for orthognatic surgery;
- removal of third molar in a fracture line.

While the NICE guidance clearly states that "*the practice of prophylactic removal of pathology-free impacted third molars should be discontinued in the NHS*", it was agreed in the NIH conference, "*after lengthy discussion*", that impaction or malposition of a third molar is an abnormal state and *may* justify its removal. These two documents present a slightly different view on whether a single episode of pericoronitis is appropriate indication for third molar removal. The NIH conference was in favor of referring to the well known "potential for repetitive infection and morbidity". From a patient perspective, Slade *et al.*⁹¹ noted that 37% of 480 patients who had pain or swelling

indicating an episode of pericoronitis 3 months before surgery, found the symptoms sufficient to seek surgical expertise for third molar removal before the symptoms recurred. This clearly emphasizes the importance of an open dialog between patients and dentists/oral surgeons where patients' preferences can be evaluated and the clinician presents his or her judgement of the benefits and risks for removal or retention for that particular third molar. One should also remember that a document such as the NICE publication is guidance, and as it is stated in this paper the guidance "*does not, however, override the individual responsibility of health professionals to make the appropriate decisions in the circumstances of the individual patient, in consultation with the patient and/or guardian/carer*". Guidelines for health care procedures are at their best an evidence-based view of eminent scientists and clinicians at a certain time and will thus form an excellent basis for ethical practice. However, new research findings, new treatment options, changes in patients' preferences, and changes in resources allocated to health care require that clinical guidelines be reviewed and updated regularly.

Prophylactic third molar removal

Prophylactic removal of a non-erupted tooth is by definition a surgical intervention to prevent future disease. This practice is often referred to as "removal of asymptomatic third molars". It is important to define this subject more precisely. A *symptom* is defined as an indication of a disease noticed by a *patient*, such as pain, swelling, restricted mouth opening, foeter ex ore, difficulty in chewing or swallowing food or drink. *Clinical signs* are observations by a *health professional* indicating a disease or disorder such as bleeding on probing, pathologic pocket depth, tenderness on palpation, and radiographic signs of pathology. Thus, it is obvious that a patient can present with an asymptomatic third molar but with clinical signs of pathology. It is not uncommon that a third molar completely impacted in bone and associated with a follicular cyst can be totally asymptomatic to the patient (Figs 13.6, 13.7). There is, however, no controversy in the professional recommendation for surgical removal of a third molar with clinical signs of disease even if this molar is asymptomatic to the patient. Surgery on such indications is regarded as therapeutic. What prophylactic surgery specifically refers to is the removal of asymptomatic and pathology-free non-erupted or impacted third molars. In other words, the indication for surgical removal in such circumstances is the mere presence of a non-erupted or impacted third molar even if not associated with pathology.

The practice of prophylactic third molar removals that emerged in the middle of the 20th century was almost universally accepted in the industrialized world. This concept was the leading norm, or gold standard, for treatment of impacted third molars in

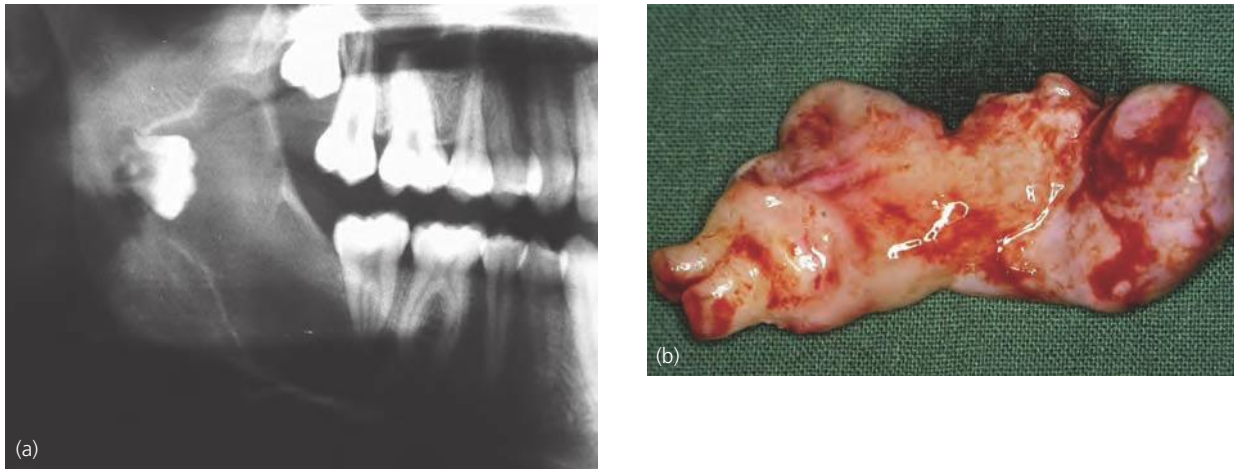


Fig. 13.6 Sixteen-year-old male examined with a panoramic radiograph (a) as part of a preorthodontic evaluation. There were no subjective symptoms and no extra- or intraoral findings suggesting the presence of this cystic lesion. The histopathologic examination (b) revealed an odontogenic keratocyst. The patient was followed radiographically for 6 years without any signs of recurrence.

textbooks on oral and maxillofacial surgery. As a good example of this view, Larry J. Peterson stated as late as 1998⁹² that, “If impacted teeth are left in the alveolar process, it is highly probable that one or more of several problems will result. To prevent this, impacted teeth should be removed.” He then argues, using radiographic illustrations, that third molar removal can prevent periodontal disease, dental caries, pericoronitis, root resorption, odontogenic cysts and tumors, and fractures of the jaw. Third molar removal is also indicated to facilitate orthodontic treatment and in some cases of unexplained pain in the retromolar region of the mandible. Furthermore, impacted teeth in areas later to be restored with a prosthodontic appliance should be removed before such treatment is performed.

Recent research on the “third molar problem” has focused on periodontal problems related to the retention of third molars. A research team headed by Raymond P. White and supported by the American Association of Oral and Maxillofacial Surgeons in

concert with the Oral and Maxillofacial Surgery Foundation has made a progress report on a very ambitious and comprehensive project called “Third Molar Clinical Trials”.⁹³ They have enrolled 356 patients for a longitudinal study with a follow-up time of at least 4 years. The median age at inclusion was 26 years and it was required that the subjects have no third molar symptoms at enrolment. From their preliminary data, they conclude that a considerable number of young adults documented substantial periodontal pathology affecting the third molar region. They also conclude that “data from the subjects followed over time suggest that in a considerable number of young adults, periodontal pathology initiates in the mandibular region, spreads forward in the lower jaw, up to the third molar region of the maxilla, and subsequently forward in that jaw”. One of their conclusions also suggests that third molar periodontal pathology is a major contributor to chronic oral inflammation with a potential for a systemic inflammatory response with



Fig. 13.7 Panoramic radiograph of a 64-year-old female. A follicular cyst was found in association with the impacted left mandibular third molar. There was no subjective or clinical finding in relation to this finding. Her other three third molars were totally impacted covered by bone and/or soft tissue, asymptomatic, and pathology-free. Only the left mandibular third molar with its associated cyst was treated surgically.

possible negative effect on cardiovascular and obstetric health outcomes. To a great extent based on these Third Molar Trials, Assael,⁹⁴ in an editorial in 2005, concludes that *“all third molars should be considered for removal in young adults in order to mitigate the risks of systemic inflammation and local progression of emergent periodontal disease”* and that patients with retained third molars *“should be informed of research regarding increased risks for systemic disease”*. One should, however, remember that patients, including young adults, with evidence of periodontal disease (pathologic pockets >4mm) in their third molar region have an accepted therapeutic indication for surgery. The treatment and prevention of periodontal problems in other regions of the jaws is of course the responsibility of their GDP.

In the last 20–25 years the practice of prophylactic third molar removal has been questioned for several reasons. Public health experts have questioned the effectiveness of allocating limited health care resources to preventive surgery. Data from decision analysis as well as from studies on patients' preferences suggest that prophylactic third molar surgery is not an appropriate procedure. Those studies have been reviewed earlier in this chapter. To this, an ethical dilemma can be added. If we admit that removal of an asymptomatic pathology-free third molar eliminates possible risks for future disease, we also have to acknowledge that such a procedure has its costs, and so also has a non-surgical management with intentional retention of the third molar. The costs for surgery include not only direct and indirect monetary costs as reviewed earlier, but also individual personal costs of postoperative disability, influence on social life, time lost from work or studies, and complications, both temporary and permanent. The costs for intentional retention of a third molar are the monetary costs for regular dental check-ups which of course also must include the third molar region, costs for removal should pathology eventually be diagnosed, and individual costs for disability related to symptoms from the disease. From an ethical point of view, it is reasonable to suggest that an invasive procedure with certain well known risks on an asymptomatic pathology-free condition requires stronger evidence than performing the same intervention with the same risks when a disease is finally diagnosed. Furthermore, it is also probably easier for a patient to accept a permanent complication or disability of an intervention if it was based on a therapeutic indication than if surgery was performed to prevent a possible disease.

Systematic reviews and guidelines

The appropriateness of prophylactic third molar removals has been assessed by independent health care organizations as well as professional associations. The number of reviews and guidelines published during the last 10–15 years confirms the impact

of third molar management as an important public health item. In the early 1990s Ben Toth¹⁸ at the Health Care Evaluation Unit at the University of Bristol summarized a literature survey stating that *“on the basis of these studies it is concluded that prophylactic surgery is not an appropriate management strategy for asymptomatic impacted third molars”*.

In 1999 the Scottish Intercollegiate Guidelines Network (SIGN) published an important and comprehensive national clinical guideline on the *“Management of Unerupted and Impacted Third Molar Teeth”*.⁹⁵ Although not specifically addressing the subject of prophylactic third molar removals, there are several statements in this document that contradict prophylactic surgery. Referring to third molars completely covered by soft tissue and/or bone and without evidence of pathology, the recommendation is to leave these teeth *in situ*. The NICE guidelines from 2000⁹⁰ are very clear in their recommendation that *“the practice of prophylactic removal of pathology-free third molars should be discontinued in the NHS”*.

Song *et al.*⁹⁶ from the NHS Centre for Reviews and Dissemination, University of York, UK, published a report in 2000 based on an extensive literature survey where published articles were assessed according to inclusion criteria on their scientific evidence (for review, see Goodman⁹⁷). From more than 4500 references of possible relevance, they examined 290 papers and finally included 40 studies in their review: two RCTs (randomized controlled studies), 24 literature reviews, and four decision analysis studies. They conclude that *“there is no reliable research evidence to support the prophylactic removal of disease-free impacted third molars. Available evidence suggests that retention may be more effective and cost effective than prophylactic removal, at least in the short to medium term.”*

The 2003 report on prophylactic third molar surgery from a Norwegian national center for health technology assessment, SMM,⁹⁸ emphasizes that the evidence is based on studies that use small selected patient groups and it is therefore difficult to give recommendations. The authors conclude, however, that *“Norwegian dentists recommend prophylactic removal of third molars when the likelihood of third molars causing problems in the future is high and the incidence of postoperative complications is low. This includes partially erupted wisdom teeth. Removal of asymptomatic fully retained wisdom teeth is not recommended.”*

The Cochrane Collaboration is an independent global network aiming to improve health care decisions through evidence-based systematic reviews. As part of this network, Mettes *et al.*⁹⁹ published a review on *“Interventions for treating asymptomatic impacted wisdom teeth in adolescents and adults”* in 2005. They selected all randomized or controlled clinical trials comparing the outcome of prophylactic removal of third molars versus their retention (no treatment). They found only three publications that satisfied their inclusion criteria and found no evidence to support

or refute routine prophylactic removal of asymptomatic third molars in adults. On the other hand, there is some reliable evidence that removal of asymptomatic third molars in adolescents neither reduces nor prevents late incisor crowding.

In a statement by the American Association of Oral and Maxillofacial Surgeons in 2007 concerning “The management of impacted third molar teeth”¹⁰⁰ there are several arguments supporting the removal of asymptomatic non-erupted third molars. The document states that “*aside from obvious indications for removal of impacted teeth such as overt pathology, removal is also the preferred option for teeth if there is insufficient anatomic space to accommodate normal eruption*” and that “*removal of such impacted third molar teeth at an early age is a valid and scientifically sound treatment rationale based on medical necessity*”.

It is evident that there are geographical differences in how dental professionals judge the scientific base for or against prophylactic third molar removals. While oral and maxillofacial surgeons in the US generally recommend patients to routinely have their third molars removed prophylactically, their colleagues in many European countries discourage their patients from having impacted pathology-free third molars removed. This may reflect differences in the health care systems, in the available manpower of surgical specialists in dentistry, but also differences in the interpretation of risks and benefits for individual patients and the need to assess priorities in a limited health care economy.

From the literature on the natural history of third molars referred to earlier, it is obvious that not all non-erupted or impacted third molars run the same risk of developing pathology. When patients ask for guidance whether or not to remove an asymptomatic pathology-free third molar it is important to present a realistic assessment of the risk for the individual third

Table 13.7 The scores from the eight variables in Table 13.6 are added together and form the “Risk Index M3”. The table indicates the level of risk with an index score ranging from 0 to 8. Of clinical importance is that the higher the score, the higher is the risk of a deep intrabony defect subsequent to third molar removal. From Kugelberg *et al.*^{101,102} Copyright © 1991 Elsevier.

Risk Index M3	Index score
No risk	≤1
Low risk	2
Moderate risk	3
High risk	≥4

molar in question. Implementing the facts on third molar-related disease prevalence one can conclude that:

- Third molars with complete root formation and totally covered by bone tissue are associated with a very limited risk for development of pathology and can thus be retained.
- Third molars partially covered by soft tissue have different risk for pathology depending on their position. A vertical or distoangular position presents a higher risk for development of associated pathology than other positions.^{28,50}
- There is no evidence at present of a significant increase of third molar-associated pathology with age.^{38,47}
- Periodontal problems on the distal surface of the second molar can result both from retaining or removing a third molar as has been shown by Kugelberg *et al.*^{101,102} The Risk Index M3 as designed by Kugelberg (Tables 13.6, 13.7) can serve as guidance when discussing the indication for third molar removal with patients at risk of remaining periodontal bone defects. An index score of 3–4 rather indicates a therapeutic indication as earlier described than a prophylactic approach.

Table 13.6 Variables included in Kugelberg’s Risk Index M3. From Kugelberg *et al.*^{101,102} Copyright © 1991 Elsevier.

Variable name	Symbol	Classification	Value
Preoperative plaque index (second molar distal)	(PLI)	Not visible Visible	0 1
Preoperative probing depth (second molar distal)	(PD)	≤6 mm >6 mm	0 1
Preoperative intrabony defect (second molar distal)	(IBDP)	≤3 mm >3 mm	0 1
Sagittal inclination third molar	(INCL)	≤50° >50°	0 1
Contact area second and third molar	(AREA)	Small contact Large contact	0 1
Resorption distal root of second molar	(RESO)	No Yes	0 1
Pathologically widened follicle of third molar	(FOLL)	No or distal Mesial ≥2.5 mm	0 1
Smoking habits	(CLG)	Non-smoker Smoker	0 1

Medical and surgical indications for prophylactic third molar removal

There are certain medical and surgical conditions or situations when removal of an asymptomatic pathology-free third molar is an appropriate treatment. Most of these indications are relatively uncommon and, although there is a general agreement on some of these interventions, others represent relative indications. In oral and maxillofacial surgery such indications are:

- the presence of a third molar in a fracture line;
- prior to orthognatic surgery;
- when a third molar may be considered for autogenous transplantation, usually to a first molar region;
- when the third molar region is involved in tumor resection or jaw reconstruction surgery.

Medical conditions that require a serious consideration of prophylactic third molar removals are:

- prior to radiation therapy for head and neck malignancies;
- prior to organ transplantation;
- chemotherapy;
- bisphosphonate therapy.

The indication for prophylactic removal is especially relevant for third molars partially or totally covered by soft tissue. The strength of the indication in those situations is often discussed in cooperation with the physician responsible for the treatment of the medical condition, but the oral and maxillofacial surgeon, however, must have a great impact on risk assessment and the decision on whether to remove or retain the third molar(s).

For 5–6 years, data has been accumulating on a rare but serious complication of bisphosphonate therapy. Our present knowledge of this condition – bisphosphonate-induced osteonecrosis of the jaws – has recently been presented by Robert E. Marx.¹⁰³ The complication presents clinically as exposed necrotic alveolar bone in the mandible or maxilla for more than 8 weeks in a patient who has taken or currently is taking bisphosphonate medication. There should be no history of radiation therapy to the maxillofacial region. The risk for developing this side-effect of the drug is correlated with its administrative route. Intravenous administration of bisphosphonates, usually prescribed for bone metastasis from breast cancer or multiple myeloma, results in a much faster accumulation in bone in contrast to the oral administration of bisphosphonates where the primary indication is treatment of osteoporosis. A high number of osteonecrotic complications (75%) develops as a result of surgical dental trauma or uncontrolled odontogenic inflammatory diseases so it is recommended that medical oncologists refer all patients with an indication for intravenous bisphosphonate therapy to an experienced dentist or an oral and maxillofacial surgeon for consultation. Dental treatments that are invasive in character, and are deemed necessary for control of odontogenic infections, such as tooth extractions or periodontal therapy, should be performed before the start of intravenous bisphosphonate therapy. That includes a recommendation of removal of non-erupted third molars with oral communication.

For patients taking oral bisphosphonates the accumulation rate in bone tissue is much slower, and there is evidence that oral surgical procedures like tooth removals, sinus augmentation grafts, and implant placements can be performed if the patient has taken this medication for less than 3 years. For patients with a regular use of oral bisphosphonates for a period longer than 3 years, there is a steady increase for risk of osteonecrosis of the jaws following invasive dental procedures with the length of use. For this group of

patients a judgement of the need for prophylactic removal of non-erupted third molars with oral communication should be made during the first 3 years of medication.

Complications associated with third molar surgery

As with all surgical procedures there is a risk for operative or postoperative complications when an impacted third molar is surgically removed. In any elective intervention the judged benefit, both in a short- and a long-term perspective, must be outweighed by the consequences of estimated complications. Besides the impact on social life, work, and studies, as well as on oral function as described earlier in this chapter, there is a number of transient or permanent complications after third molar surgery that should be considered. The prevalence, prevention, and treatment of those complications is covered elsewhere in this book (Chapter 14). There is an extensive literature on complications following third molar removal. A summary of frequencies reported in the last 20–30 years as reviewed by Liedholm¹⁰⁴ indicates the following possible complication rate:

- postoperative alveolitis 0.5–32.5%;
- postoperative infection 0.9–4.2%;
- postoperative bleeding 0.2–1.5%;
- transient dysfunction of the inferior alveolar nerve 0.6–5.5%;
- permanent dysfunction of the inferior alveolar nerve 0.1–0.9%;
- transient dysfunction of the lingual nerve 0.004–11.5%;
- permanent dysfunction of the lingual nerve 0.2–0.6%.

When giving advice to a patient whether or not to have a third molar removed, the surgeon must inform the patient of his or her judgement of risks associated with the particular third molar in question. There are several factors to consider when such a risk assessment is made. The age of the patient, his or her medical history, the degree of impaction and angular position, as well as the relation of the third molar roots to anatomical structures, such as the inferior alveolar nerve, are all factors that may influence the risk for complications associated with third molar removal. Thus, the information of operative risks must be adapted to the individual third molar, e.g. the slightly mesoangular third molar partially covered by soft tissue and with a short conical root presents a lower risk for complications than a horizontally deep bone-impacted third molar with divergent roots with the mandibular canal in close proximity to the roots. When a patient considers the benefits of prophylactically removing an asymptomatic pathology-free third molar it is especially important to make sure that this patient comprehends the



Fig. 13.8 Panoramic radiograph of a woman at age 56 years. The right mandibular third molar is totally covered by bone, asymptomatic, and without associated pathology. The decided strategy was a long-term observation.

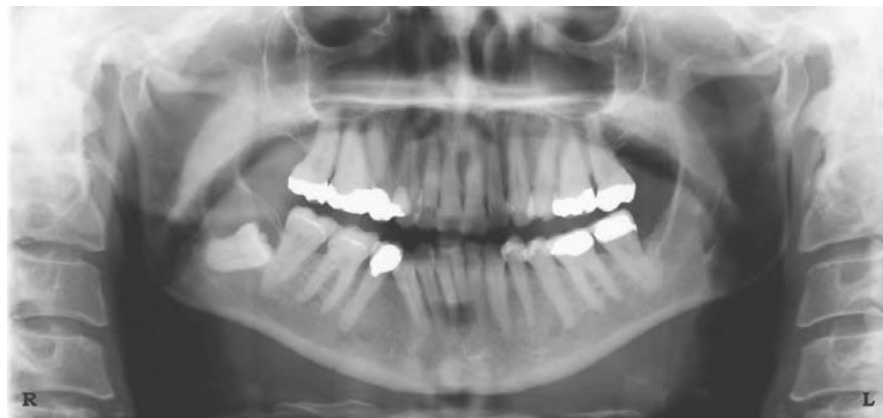


Fig. 13.9 Panoramic radiograph of the same person as in Fig. 13.8, now at age 63. A follicular cyst has developed during these 7 years, now indicating removal of the impacted third molar and a cystectomy.

judged risks for complications. It may also be relevant to inform the patient that in a number of professional reviews and guidelines this indication for third molar removal is discouraged. Furthermore, studying patients' preferences about third molar surgery, patients ranked the described outcomes of non-removal higher than those outcomes describing the outcomes of removal.

Contraindications for third molar removal

As a general rule, no teeth should be removed without an appropriate indication. Summarizing our current knowledge on the natural history of third molar impaction and professional judgement published in reviews and guidelines it is recommended that:

- Third molar buds in young people should not be enucleated.
- Asymptomatic and pathology-free third molars totally covered by bone should not be removed.
- Routine removal of asymptomatic pathology-free third molars totally or partially covered by soft tissue is not recommended. Specific medical and surgical conditions as well as a high index score on

the Kugelberg Risk Index M3 may prove a prophylactic approach appropriate.

- Third molar surgery is contraindicated in patients whose medical history or conditions expose the patient to an unacceptable risk to their overall health.

When an impacted third molar is diagnosed and a decision is made to retain that molar, the patient should be informed of the reason for non-intervention. Regular examination of that third molar to check the development of possible pathology should be recommended (Figs 13.8, 13.9).

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Chapter 14

Surgical Management of Third Molars

Tara Renton

Third molar removal is one of the most common surgical procedures worldwide. This chapter provides an overview of presurgical assessment and management and describes principles for the surgical management. The intention of this chapter is to provide some useful assessment and surgical tips for use in third molar surgery. It is also a specific aim to introduce the idea of minimal access third molar surgery in order to minimize trigeminal nerve injuries where possible. Coronectomy is discussed as a viable alternative to tooth removal in high-risk third molar teeth.

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Introduction

The failure of eruption of third molars is a very common condition.^{1–3} Extraction of impacted third molar teeth is one of the most frequent surgical procedures carried out in the National Health Service (NHS) in the UK.³ It has been reported that a significant proportion of those on oral surgery waiting lists are awaiting third molar removal.^{2,3}

Removal of third molars remains the “mainstay” of activity for oral surgery. Third molar surgery has been one of the ten most common procedures undertaken in the NHS and the cost of removing wisdom teeth in the UK has been calculated to be in the region of £50 million per annum, although this figure may well have decreased significantly since the publication of guidelines by the National Institute for Health and Clinical Excellence.⁴ Conversely over 95% of oral surgeons in the USA regularly undertake prophylactic third molar surgery.⁵ As a profession we must be cognizant of various geographical differences in oral surgical practice as the pressure and outcomes will vary accordingly. Third molar surgery (TMS) is a source of considerable income for some surgeons.⁶ It comprises the main costs for some USA insurance companies^{6,7} and for all those involved TMS provides a daily challenge in patient care provision. Guidelines for the indications for third molar surgery are discussed in Chapter 13.

Surgical procedures for extraction of third molar teeth are associated with significant morbidity. These include expected sequelae, including pain and swelling, and potential complications, including temporary or permanent nerve damage, resulting in permanent numbness, pain or altered sensation of lip or tongue.⁸ There appears to be substantial variation in management of third molars and it has been reported that conservative treatment with more rigorous adherence to specific indicators for removal would reduce surgical cases by up to 60%.⁹ A recent review by the NHS Centre for Reviews and Dissemination¹⁰ concluded that “*there appears to be little justification for the removal of pathology-free impact-*

ed third molars". However in the USA prophylactic removal of third molars is routine⁶ and debate continues on this practice.^{6,7,9,11–13} Despite this transatlantic difference a consensus exists that the keys to successful surgery are good training, planning, patient selection, and care provision (preoperative, operative, and postoperative). The main complications related to third molar surgery are alveolar osteitis (fibrinolytic alveolitis, dry socket) and temporary or permanent nerve injury.¹⁴ These injuries often cause permanent neuropathic pain in and around the mouth, resulting in significant difficulties for the patient with regard to eating, drinking, speaking, kissing, shaving, applying makeup, and often sleeping (just about every social interaction we take for granted). As a result lingual and inferior alveolar nerve injuries remain one of the main causes of litigation related to dentistry, often resulting in significant financial compensation depending on the country and legal system. Prevention of these injuries must be foremost in the surgeon's mind when planning this type of surgery. This section aims to provide a basic framework and useful tips for third molar surgery.

Definitions and terminology

For the purposes of this chapter guideline, minor modifications of previously reported definitions^{1,5,8,9} have been introduced:

- An unerupted tooth is a tooth lying within the jaw bone, entirely covered by soft tissue, and partially or completely covered by bone.
- A partially erupted tooth is a tooth that has failed to erupt fully into a normal position. The term implies that the tooth is partly visible or in communication with the oral cavity. Pericoronitis, the most common indication for removal of third molars, can only occur if the tooth is partially erupted and in communication with the mouth.
- An impacted tooth is a tooth which is prevented from completely erupting into a normal functional position. This may be due to lack of space, obstruction by another tooth, or an abnormal eruption path. The tooth may be soft tissue impacted or hard tissue impacted (bone or adjacent tooth) and may be unerupted or partially erupted. Impaction per se is NOT an indication for removal, it is a clinical description of the position of the tooth.

Throughout this chapter the term **third molar** refers to a partially erupted third molar tooth which may or may not be impacted. In general the term applies to both **upper** and **lower** third molar teeth, but surgical management of upper third molars is generally simpler compared to that of lower third molars. Upper wisdom teeth cause less discomfort, are more likely to erupt, and are simpler to remove unless unerupted and encased in bone (when perhaps removal is not

indicated). Removal of upper third molars results in far less postoperative morbidity.

Presurgical assessment of third molars

Clinical assessment

Clinical assessment should be carried out with the aim of assessing the status of the patient and their third molars and excluding other causes of the symptoms. Initial assessment should include:

- patient complaint;
- patient age;
- social history;
- a full medical history;
- a full dental history;
- extraoral clinical examination;
- intraoral clinical examination.

Positive findings from this examination, which suggest that treatment of the third molar or related structures may be indicated, require that a more detailed examination is carried out. This should determine whether removal is indicated and/or advisable (see Chapter 13) and should include radiological assessment.

A complete examination should include assessment of:

- willingness and ability of the patient to cooperate with care;
- the eruption status of the third molar (Figs 14.1, 14.2);
- caries in, or resorption of, the third molar and the adjacent tooth;
- periodontal status (Fig. 14.3);
- occlusal relationship;
- temporomandibular joint function (identify existing limited access or pain which may be exacerbated by third molar surgery);
- mouth opening, as trismus will hinder access for surgery;
- regional lymph nodes;
- any associated pathology.

Radiological assessment

The purpose of a careful radiological evaluation is to complement the clinical examination by providing additional information about the third molar, the related teeth and anatomic features, and the surrounding bone. This is necessary in order to make a sound decision about the proposed surgical procedure, the most appropriate location for this to take place, and to highlight aspects of management which may require specific discussion with the patient.



Fig. 14.1 Clinical photograph of partially erupted lower left molar with no local inflammation.



Fig. 14.2 Clinical photograph of partially erupted lower left with acute pericoronitis of 38. Notice the "imprint" of 28 occlusal surface.

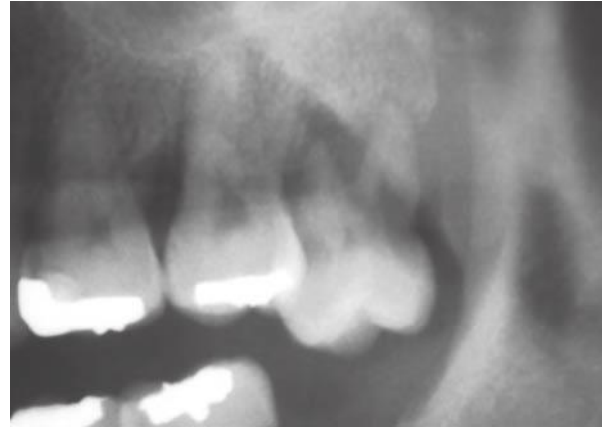


Fig. 14.3 An overerupted upper 28 with periodontal disease.

Prior to the age of 13, radiographic examination is not normally indicated for the assessment of third molars¹⁵ and films taken from the age of 20 are most useful in assessing the likelihood of eruption.¹⁶ When more than one third molar requires assessment, the radiographic examination of choice is a panoramic radiograph as the radiation dose of a panoramic radiograph is lower than from four periapical views and the diagnostic yield is higher.¹⁷⁻¹⁹ Doses from panoramic radiography can be further limited by using field size limitation to prevent exposing areas not required in the field of view.²⁶ Periapical or oblique lateral radiographs may be taken as an alternative. All radiographs should be of a diagnostically acceptable standard.^{19,20}

The effectiveness of axial computed tomography (CT) with coronal and sagittal reformatting in establishing the three-dimensional relationship of the inferior alveolar nerve (IAN) and the third molar has been shown.²¹⁻²³ CT permits: localization of the IAN canal in the superior-inferior and mediolateral positions; detection of an intraradicular path; determination of the distance between the tooth and IAN canal; and root angulation. In the setting of high-risk findings on the panoramic radiograph where the mandibular third molar requires removal, CT can provide valuable information to facilitate management.²³ If the IAN is entrapped within the substance of the tooth, coronectomy or monitoring may be indicated. Knowing the IAN canal position relative to the third molar may decrease the frequency or severity of IAN injury.

Overall the combined clinical and radiologic assessment of third molars is intended to enable the clinician to make a diagnosis which may be concordant with indications for removal of the tooth leading to surgical planning. Factors that are important in planning the surgical approach are shown in Table 14.1.

Application depth

The alveolar bone level and tooth position will dictate the application depth (Fig. 14.4). The depth of applica-

tion and the point of elevation will dictate the amount of bone removal that is required to gain access to the optimal application point. This factor is the most predictive of difficulty of surgery.^{24–26}

Table 14.1 Dental factors that may be important in planning surgery.

Factor	Illustration
Depth impaction	Fig. 14.4
Angulation	Fig. 14.5
Crown width	Fig. 14.6(a)
Crown condition of 8 – caries, gross caries, heavily restored	Fig. 14.6(b–e)
Root width	Fig. 14.7
Root morphology	Fig. 14.8
Root surface area compared with adjacent tooth	Fig. 14.9
Enlarged follicular size	Fig. 14.10
Associated cyst	Fig. 14.11
Periodontal status 8 and 7	Fig. 14.12
Restorative condition of adjacent 7	Fig. 14.13
Proximity to inferior alveolar canal	Fig. 14.14
CBCT scan of IAN close to lower 8	Fig. 14.15
Long rooted lower third molars or atrophic mandible	Fig. 14.16
The relationship or proximity of upper third molars to the maxillary antrum and of lower third molars to the inferior dental canal	Fig. 14.17

Angulation

The distinction between vertical, mesoangular, horizontal, and disto-angular orientation may affect the surgical approach, in particular with regard to the requirements for bone removal in order to gain access to point of application. These positions have been classified by Archer (1975).²⁷ The incidence of the position of the tooth is:

- Vertical impaction: 40% of all impacted mandibular third molars and usually the least difficult to remove.
- Mesoangular impaction: 45% – can be of moderate difficulty.
- Horizontal impaction: 10% – are generally of intermediate difficulty.
- Distoangular: 5% – the most difficult to extract and frequently underestimated.

The application point is not only dictated by the angulation of the tooth but also root morphology and proximity to adjacent structures (adjacent tooth, antrum, and nerve). In association with identifying the tooth angulation, the potential space for application points must be identified. Often distoangular lower third molars are in close proximity to the adjacent 7 and have limited space for mesial application point. Several examples of radiographs of third molars illustrating angulation of teeth and a summary of potential application points and planned sectioning are provided in Figs 14.4 and 14.5.

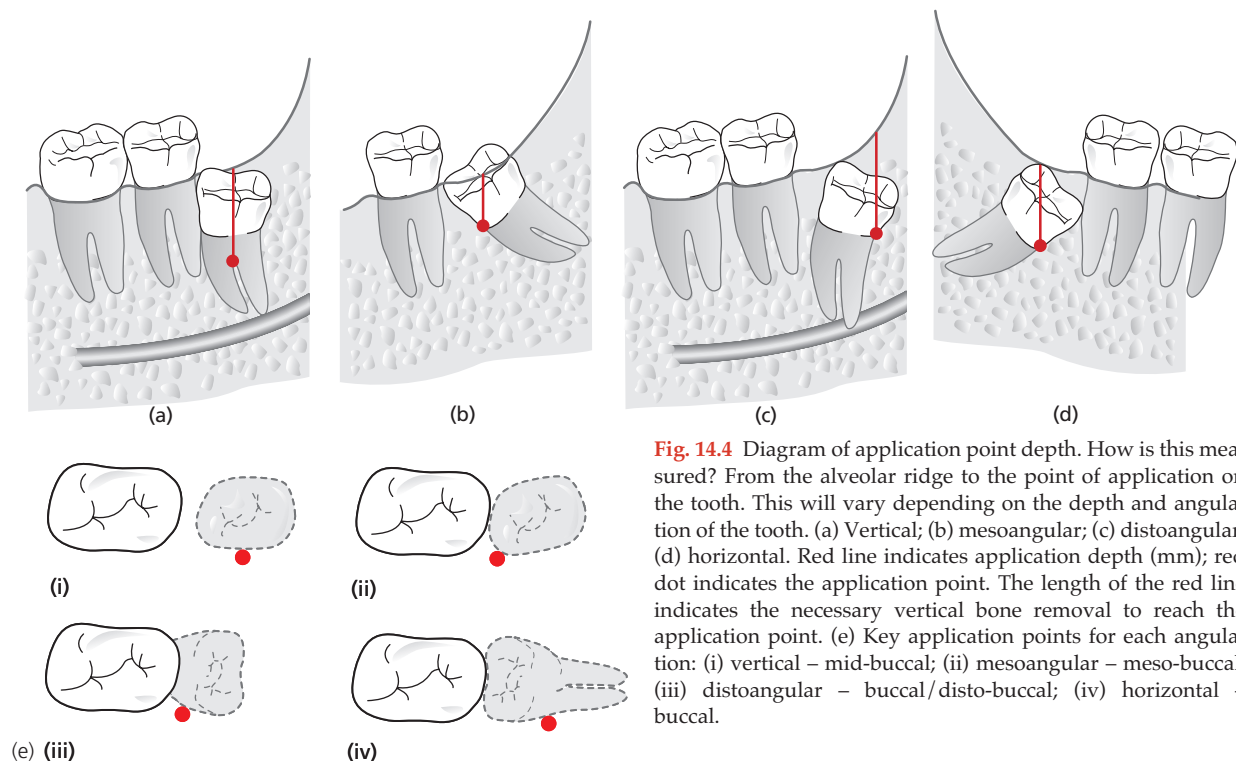


Fig. 14.4 Diagram of application point depth. How is this measured? From the alveolar ridge to the point of application on the tooth. This will vary depending on the depth and angulation of the tooth. (a) Vertical; (b) mesoangular; (c) distoangular; (d) horizontal. Red line indicates application depth (mm); red dot indicates the application point. The length of the red line indicates the necessary vertical bone removal to reach the application point. (e) Key application points for each angulation: (i) vertical – mid-buccal; (ii) mesoangular – meso-buccal; (iii) distoangular – buccal/disto-buccal; (iv) horizontal – buccal.



Fig. 14.5 The orientation of the tooth and the proximity to the adjacent tooth are important to assess, particularly with regard to distinction between vertical and distoangular orientation which may affect the requirements for bone removal. (a) Maxillary buccally erupted mesoangular 28 with no application space distal to 27. (b) Maxillary buccally partially erupted impacted distoangular 28. (c) Mandibular non-impacted erupted 48 in vertical position. (d) Vertical position, soft tissue impacted, partially erupted 48 with evidence of chronic inflammation with distal bone loss. (e) Vertical position, unerupted, bone impacted. (f) Horizontal position, partially erupted, tooth impacted. (g) Horizontal position, unerupted with oral communication. (h) Horizontal, unerupted below bone, no oral communication. (i) Mesoangular position, partially erupted, tooth impacted. (j) Distoangular, partially erupted, bone impacted. Diagrammatic summary of tooth angulation: classification of impaction of (k) maxillary third molars and (l) mandibular third molars (according to Archer, 1975²⁷): 1, mesoangular; 2, distoangular; 3, vertical; 4, horizontal; 5, bucco-angular; 6, linguo-angular; 7, inverted.

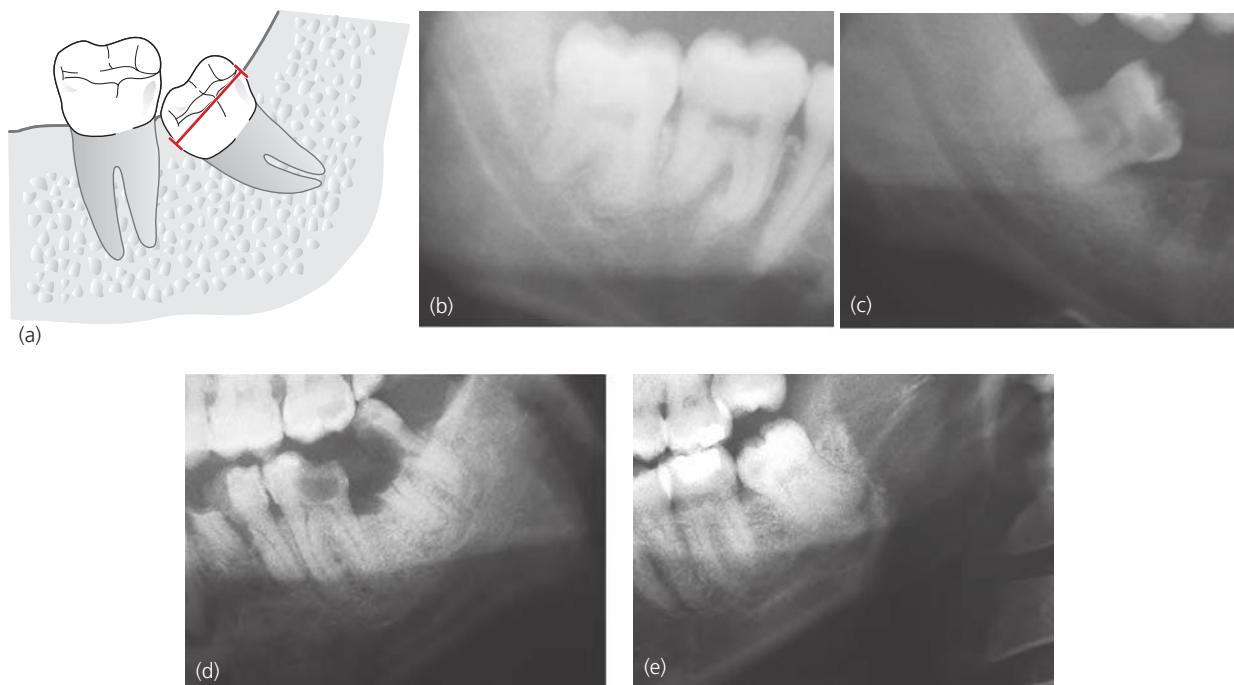


Fig. 14.6 Size of the crown. (a) Diagram of measurement of crown width. Red line drawn across maximum width of crown. If this is more than bony window in alveolar ridge (black line) then crown section will be indicated. (b) Crown wide. Wide crown width will increase surgical difficulty. (c) The crown condition – caries. (d) Gross caries of 38. (e) Crown heavily restored.

The crown size and condition

The crown width of the third molar can be an important factor in making surgery more difficult. If the tooth is impacted and the crown is wide, sectioning of the crown will be required to minimize bone removal thus reducing potential pain and swelling for the patient. If the third molar is carious or heavily restored or indeed non-vital, this may render the tooth more brittle on elevation thus impacting on surgical difficulty. See Fig. 14.6.

Distance across roots is larger than width of crown–root junction

If the root width is greater than the width of the tooth at the alveolar crest, the difficulty of surgery will increase and root sectioning will be indicated (Fig. 14.7). Multiple roots or hooked roots will also contribute to surgical difficulty and are more commonly seen in certain ethnic groups. Hypercementosis or ankylosis of third molar roots will also increase surgical difficulty and must be identified on radiographic evaluation.

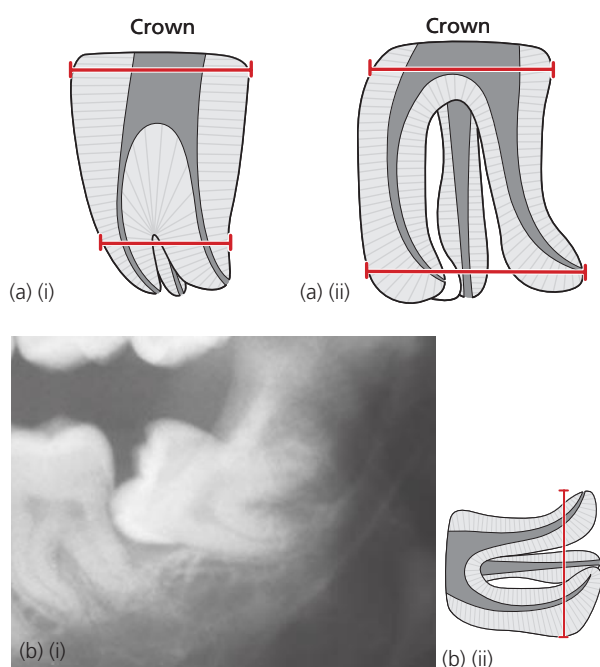


Fig. 14.7 (a) Diagram of measurement of root width. (i) Roots convergent when root maximum width is less than that of the crown base. (ii) Roots divergent when maximum root width is greater than base of crown (indication of increased difficulty of surgery and probable root section). (b) (i and ii) Splayed roots. Distance across roots is larger than width of crown root junction. (c) Lower third molar with four splayed roots.

Root number and morphology

More complex root morphology is associated with increased difficulty of surgery (Fig. 14.8).²⁸ Identification of such hooks is important, as they may fracture during removal of the tooth and a decision is then

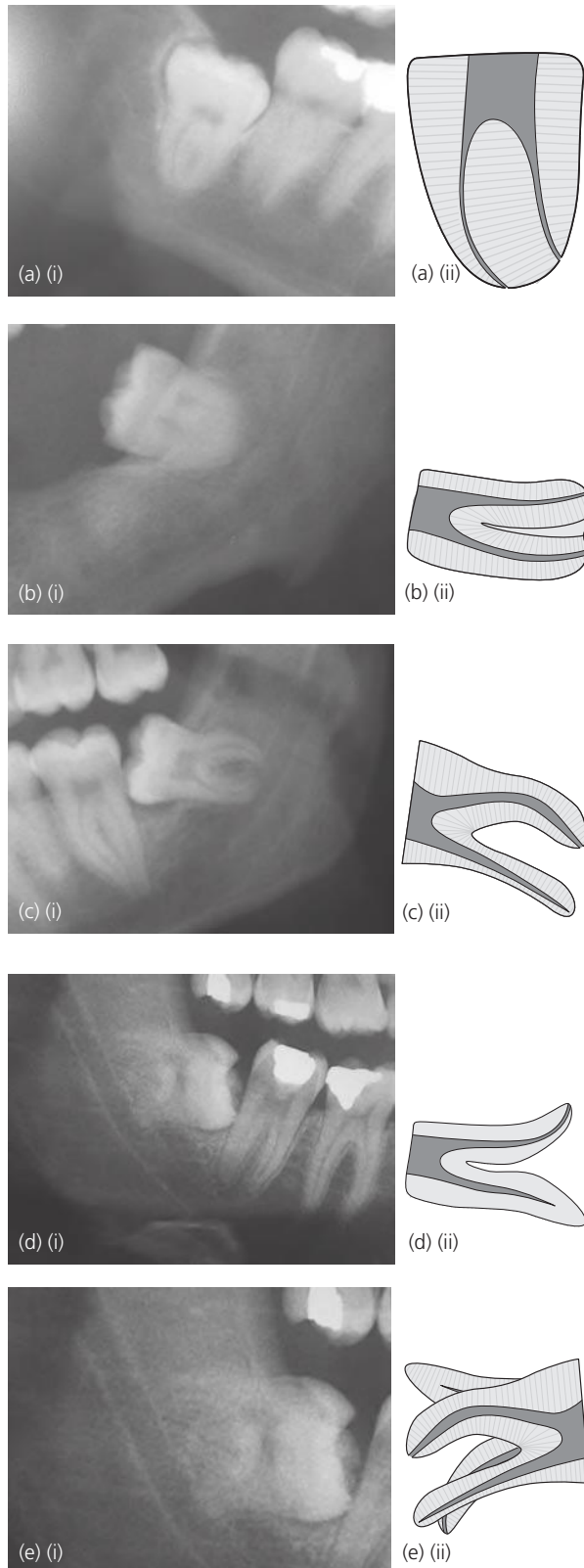


Fig. 14.8 (a) Conical/convergent roots. (b) Club-shaped roots. (c) Bifid roots. (d) Bifid divergent roots. (e) Multiple roots (may be penetrating lingual plate).

required as to whether to attempt their removal. Hooked, dancing or bent roots may provide a “challenge” to routine elevation. Even after sectioning of the crown and roots, if the root morphology is complex the roots will often require further sectioning and patience is often required to identify the “sweet spot” with which the resistant root will comply with elevation.

Occasionally lower third molar roots can perforate the lingual plate and this is often not evident until part way through surgery. Every effort must be made to ensure these roots do not get lost through the perforated lingual plate, one of the rare indications to raise a lingual flap for third molar surgery.

Root surface area

Assessment of the relative root surface area of the tooth to be extracted and the adjacent tooth must be estimated (Fig. 14.9). If the third molar is multirrooted adjacent to a conical root lower 7, the lower 7 is more easily mobilized during elevation of the 8. If the root surface area of the adjacent tooth is significantly less than that of the intended extractee then the surgical approach must be adjusted accordingly to prevent any undue application of pressure near the adjacent tooth.

If the adjacent tooth is subluxated it should be immediately replaced and a figure-of-eight suture placed across the occlusal surface to “restrain” the tooth. Antibiotics and oral hygiene should be initiated, and, of course, the patient advised.

Follicular width

There is no substantive evidence as to the dimension of follicular space which clearly indicates that cystic change has taken place.^{29,30} Where doubt exists as to the likelihood of cystic change and there are no other positive indications for removal of the tooth, radiological review at between 6 and 12 months is the recommended course of action.

An enlarged follicle is usually considered to be non-cystic where the diameter is less than 1 cm. Radiographic radiolucency adjacent to the crown of a partially erupted third molar may be due to a

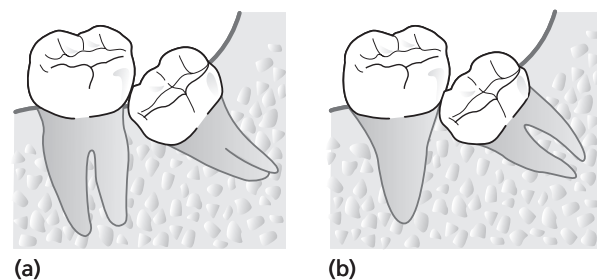


Fig. 14.9 Root surface area depends on length, width and number of roots. (a) Root surface area of 8 less than that of 7. (b) Root surface area of 8 greater than that of 7 (risk of displacing 7 on mesial application to 8).



Fig. 14.10 Radiographic radiolucency adjacent to the crown of an impacted third molar may be due to enlarged follicle.

previously enlarged follicle or chronic local infection (Fig. 14.10).

Pathology associated with third molars

Cysts are the most common pathology associated with mandibular third molars. If the cyst is associated with the crown of the tooth, the first differential diagnosis should be dentigerous cyst followed by keratocyst or ameloblastoma. Keratocysts are more likely to be associated with missing teeth. Surgical removal of third molars in association with pathology may be facilitated. However the surgeon must ensure the pathology is correctly diagnosed and treated accordingly. Some examples of cysts associated with third molars are shown in Fig. 14.11.

Periodontal status

The periodontal status of the adjacent tooth and that of the third molar (Fig. 14.12) will influence surgery. Bone loss due to periodontal disease will often facilitate the removal of the third molar tooth but care must be taken to prevent inadvertent removal of the periodontally involved 7.

The restorative condition of the adjacent 7

If the adjacent tooth to the third molar is carious or heavily restored or indeed non-vital (Fig. 14.13), this may render the tooth more brittle and more at risk of damage on elevation of the third molar, thus impacting on surgical difficulty. It is important that the patient is warned of possible damage to the adjacent 7 during surgery.

Proximity to the inferior alveolar canal

There are several radiographic signs assessed on panoramic radiographs that are associated with an increased risk for IAN injury (Fig. 14.14).^{26,31–36}



Fig. 14.11 Cystic lesions associated with third molar teeth. (a) Paradontal cyst associated with 38. (b) Monocystic lesions associated with crown of mandibular third molar likely to be a dentigerous cyst but could be mimicked by keratocyst. (c) Multicystic lesion associated with impacted 38. This proved to be an ameloblastoma.



Fig. 14.12 The periodontal status of 48 is severe.

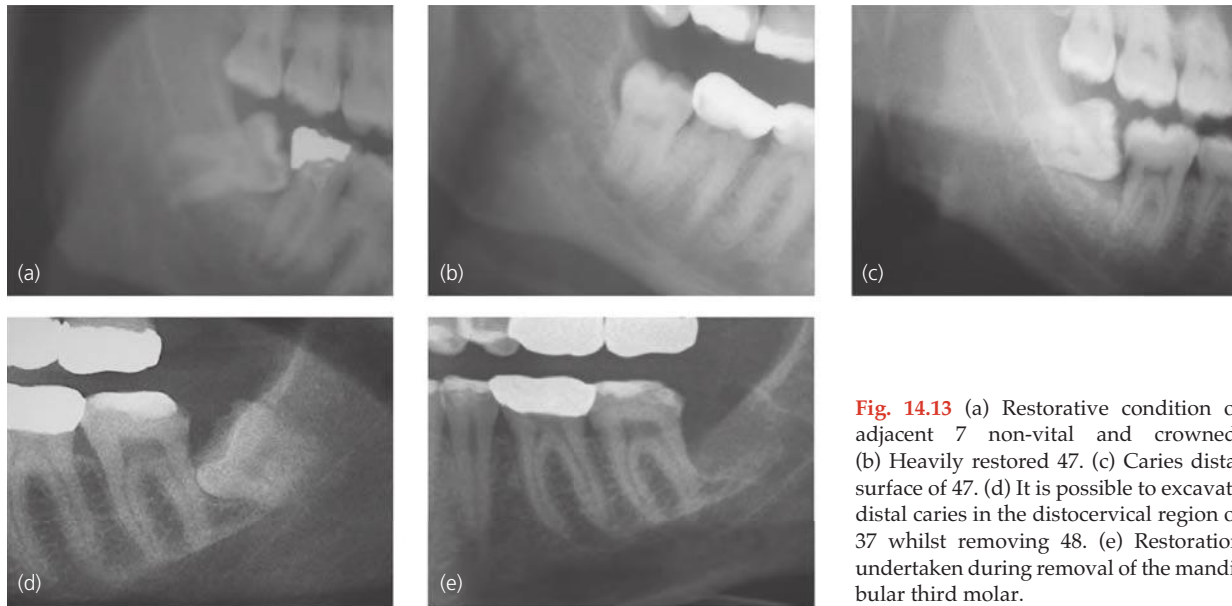


Fig. 14.13 (a) Restorative condition of adjacent 7 non-vital and crowned. (b) Heavily restored 47. (c) Caries distal surface of 47. (d) It is possible to excavate distal caries in the distocervical region of 37 whilst removing 48. (e) Restoration undertaken during removal of the mandibular third molar.

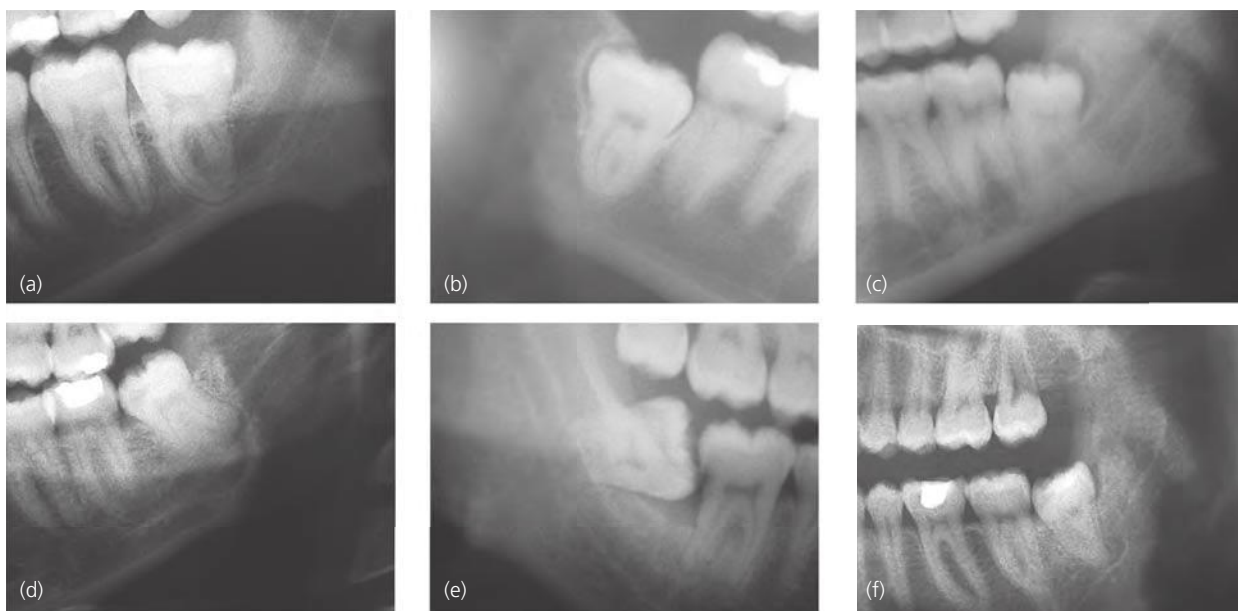


Fig. 14.14 (a) Superimposition of the IAN canal and third molar. (b) Diversion of the IAN canal. (c) Darkening of the root where it is crossed by the canal and widening of canal. (d) Interruption of the white lines of the canal. (e) Darkening of roots with associated widening of the canal. (f) Juxta apical area. This apical area on a vital third molar may reflect a confluence of the periodontal membrane and the IAN canal; however thus far neither CT nor MRI scan have been able to detect specific cavities. This radiographic feature was the main radiological risk factor for IAN injury.²⁶

Cone-beam CT scanning

Cone-beam CT (CBCT) scanning for mandibular third molars is indicated if on the initial panoramic radiograph, there is a suggestion of a relationship between the roots of the lower third molar and the IAN canal. This is a relatively new technique and caution is required to only use this additional scanning when the lower third molar is found to cross the IAN canal on plain films. It will be interesting to see how CBCT scanning will influence surgery. For many surgeons, high-risk vital third molars are planned for coronectomy and CBCT will not alter the planned surgery in these cases. However if the high-risk third molar is non-vital or the patient requests removal of

the third molar, CBCT scanning may assist the surgeon in planning surgery depending on the relationship of the IAN canal and the tooth. Examples of lingual and buccal relationship of the IAN to the lower third molar tooth are shown in Fig. 14.15.

The sensitivity and specificity of CT imaging to panoramic imaging in predicting IAN exposure at the time of third molar removal was 70 and 63% respectively.²² This suggests that in the absence of any high-risk findings on panoramic radiograph, the risk for IAN injury is unlikely and has a high negative predictive value (>90%). Other studies have reported that the presence of two or more radiographic signs, depth of impaction, and horizontal angulation were

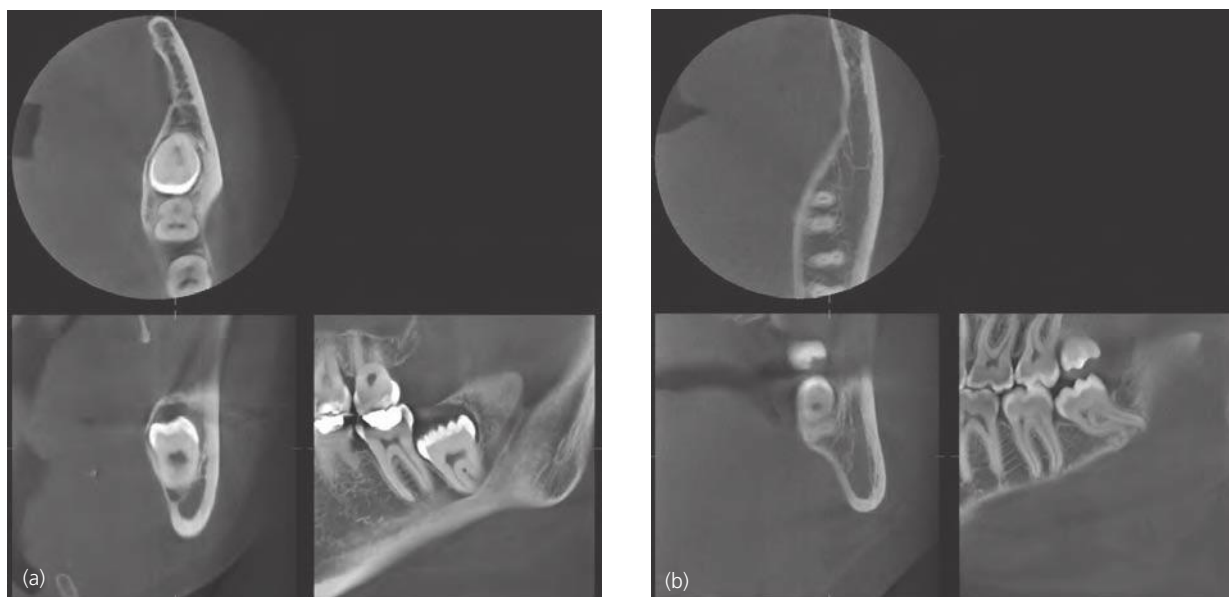


Fig. 14.15 Cone-beam CT (CBCT) scan of lower third molar. (a) CBCT section illustrating close proximity of IAN to the third molar tooth root on the lingual aspect. (b) CBCT section illustrating distant proximity of IAN to the third molar tooth root on the buccal aspect.

associated with an intimate anatomic contact between the third molar and IAN canal on CT.³⁶ Likewise another study noted several clinical and panoramic radiographic predictors associated with an increased risk for IAN injury.³⁷ For the CT findings, the sensitivity and specificity were 93 and 77%, respectively. In another study similar findings were noted with the surgeon's prediction of IAN injury on reviewing the panoramic radiograph to have a sensitivity of 72% and specificity of 91%.³⁵ The American Association of Oral and Maxillofacial Surgeons (AAOMS) White Paper⁵ concludes that the exact role and indications for CT imaging for the management of impacted third molars is unclear and evolving. In the presence of any of the above findings the decision to treat should be carefully reviewed. The patient should be advised of the increased risk (20% temporary or 2% permanent IAN injury). Alternative surgical tech-

niques may be indicated (coronectomy of the tooth if vital, splitting of the roots if multirrooted).

Atrophic mandible or very long roots of third molar

Exceedingly long roots or atrophic mandible may place the patient at higher risk of mandibular fracture during removal of the lower 8 (Fig. 14.16).

Proximity of other structures

The relationship or proximity of upper third molars to the maxillary antrum and of lower third molars to the IAN canal should be assessed (Fig. 14.17).



Fig. 14.16 Exceedingly long roots or atrophic mandible may place the patient at higher risk of mandibular fracture during removal of 38.



Fig. 14.17 The relationship or proximity of third molars to the adjacent unerupted teeth. Maxillary antrum to 18 and 48 and 19 to the inferior dental canal.

Risk assessment

Evaluating the difficulty of third molars has been the aspiration of many authors (Table 14.2). The primary factors that influence the difficulty of third molar surgery (length of procedure and related complications) will depend on patient factors, the depth of application, and proximity to vital structures.³⁸

A study of 4004 patients showed a 1.5 times likelihood of a complication if the patient had third molars removed at over 25 years of age, with generalized increasing risks with age through to age 65.⁴⁸ A consensus of the literature supports the concept that postoperative risks from third molar removal increase with age.²⁸

Table 14.2 Risk factors.

Factors	Complication risk	Reference
Patient factors		
Age <25 years	Reduced	Mercier 1991 ³⁹
Age >25 years	Increased	Bruce 1980 ⁴⁰ Phillips 2003 ⁴¹ Valmaseda-Castellón <i>et al.</i> 2000 ⁴² Gbotolorum <i>et al.</i> 2007 ²⁸
Ethnicity	Non-Caucasian – longer operating time	Renton <i>et al.</i> 2001 ³⁸
Body mass index	Increased difficulty	Gbotolorum <i>et al.</i> 2007 ²⁸
Gag reflex	Risk of aspiration	Elgazzar ⁴³
Anxiety	Increased difficulty	Phillips 2003 ⁴¹
Gender	Males increased complications	Renton <i>et al.</i> 2001 ³⁸
Dental factors		
Root morphology	Increased difficulty	Gbotolorum <i>et al.</i> 2007 ²⁸
Depth application point		Susarla & Dodson 2004 ²⁵ Renton <i>et al.</i> 2001 ³⁸ Gbotolorum <i>et al.</i> 2007 ²⁸
Operative factors		
Lingual flap	Increases lingual nerve injury	Yuasa & Sugiura 2004 ⁴⁴ Robinson <i>et al.</i> 2003 ⁴⁵ Pichler & Bierne 2001 ⁴⁶ Valmaseda-Castellón <i>et al.</i> 2000 ⁴²
Anesthetic	General anesthetic increased complications	Brann <i>et al.</i> 2006 ⁴⁷
Junior surgeons	Junior surgeons increased complications	Jerjes <i>et al.</i> 2006 ³⁷

Grade of difficulty

1. Pell and Gregory classification.⁴⁹ Describes the relationship of tooth to anterior border of ramus (1, 2, 3) and relationship of tooth to occlusal plane (A, B, C) (Table 14.3). The level of impaction was determined using Pell and Gregory classification. In another study Pell–Gregory classification has been found to be an unreliable predictor of difficulty in extracting impacted lower third molars.⁵⁰
2. Winters lines⁵¹ (Fig. 14.18).
3. Pederson method of assessment of difficulty (Table 14.4).
4. Yuasa classification of difficulty²⁴ (Table 14.5).
5. Renton *et al.* 2001.³⁸ The main factors that prolonged operative time for third molar surgery in 1400 patients were in order of importance:

- depth of impaction;
- density of bone;
- age of patient/ethnicity of patient;
- proximity to IAN canal;
- surgeon.

There are many types of grading methods for the assessment of difficulty of third molars. The author believes that the most important factors are:

- patient factors (cooperation, age, ethnicity, and mouth opening);
- dental factors (application depth, root morphology, and condition of teeth);
- surgical factors (surgeon technique and training).

The suggested methods for difficulty assessment may assist the less experienced surgeon.

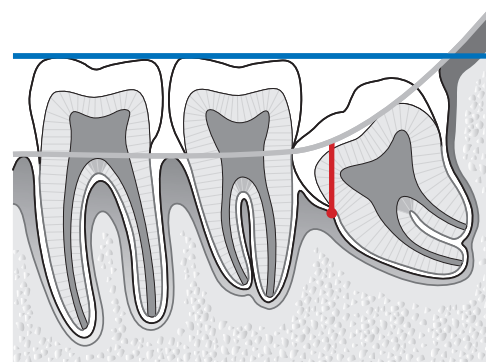


Fig. 14.18 Winter's lines. The most important factors may be remembered by using the mnemonic WHARFE: W, angulation using Winter's lines; H, height of mandible; A, angle of second molar; R, root form and development; F, size of follicular sac; E, exit path of tooth to be extracted.

Table 14.3 Pell and Gregory classification.

(1) Position A	The highest portion of the impacted mandibular third molar is on a level with or above the occlusal plane
(2) Position B	The highest portion of the impacted mandibular third molar is below the occlusal plane but above the cervical line of the second mandibular molar
(3) Position C	The highest portion of the impacted mandibular third molar is below the cervical line of the second mandibular molar
(4) Position I	None of the crown is in the ramus of the mandible
(5) Position II	Less than half of the crown is in the ramus
(6) Position III	More than half of the crown is in the ramus

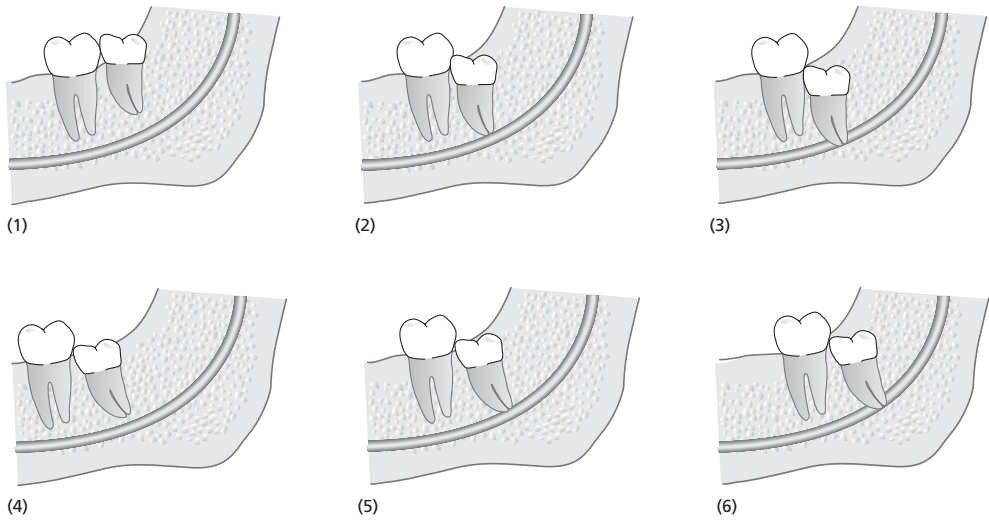


Table 14.4 Difficulty index for removal of impacted mandibular third molars, as described by Pederson.⁵²

Classification	Value
Spatial relationship	
Mesoangular	1
Horizontal/transverse	2
Vertical	3
Distoangular	4
Depth	
Level A: high occlusal level	1
Level B: medium occlusal level	2
Level C: deep occlusal level	3
Ramus relationship/space available	
Class 1: sufficient space	1
Class 2: reduced space	2
Class 3: no space	3
Difficulty index	
Very difficult	7–10
Moderately difficult	5–6
Slightly difficult	3–4

Table 14.5 Yuasa’s classification of difficulty of surgical removal of impacted third molars. The key factors indicating highest degree of difficulty are listed.

Depth or ramus relationship/space available or width of root on rotational panoramic images predicts difficulty	
Depth is deep occlusal level	Level C
Ramus relationship/space available is no space	Class 3
Width	Bulbous

Note: abnormal root curvature is not seen in some radiographs.

Presurgical management

Once it has been decided that a third molar should be removed, consideration should be given to the appropriate treatment setting. Patient factors, both local (spreading infection) and systemic (bleeding tendency), will dictate possible treatment strategies. Pretreatment may be necessary for persistent local infection or a medical condition. Essentially the practitioner must assess the patient's demeanor. If the patient is unable to cooperate with routine dental care, or too anxious for care under local analgesia, patient management will need to be modified accordingly.

Referral

Many general dental practitioners will feel suitably competent to manage routine or even complicated third molars, although this does not preclude direct referral to a department of oral surgery or a specialist practitioner. The basis of this decision should take account of the general suitability of the facilities and training for operative procedures, the recovery facilities, and the competence of support staff. In addition, each case should be assessed with regard to the patient's medical history, patient preferences, and the expected degree of difficulty of surgical treatment.

The referring clinician should provide information as to the clinical findings on presentation, medical history, and any radiographs pertinent to the case. Ideally all preoperative radiographs should be shared between clinicians concerned with the assessment and treatment of the patient. The operating surgeon should retain a duplicate in situations where the level of morbidity raises concern.

A discharge letter should always be sent to the referring clinician outlining the treatment plan, specific information provided to the patient, the form of anesthesia, and what follow-up arrangements are required.

Consent

Obtaining informed consent is a general legal and ethical principle that must be undertaken before surgical intervention. Valid consent must be obtained before starting treatment or physical investigation, or providing personal care, for a patient. This principle reflects the right of patients to determine what happens to their own bodies, and is a fundamental part of good practice.⁵³ Patients have a right to choose whether or not to accept a dental professional's advice or treatment.

In the UK the General Dental Council (GDC) has set standards for dental professionals⁵³ and recommends the following principles in getting consent:

1. Informed consent – the patient has enough information to make a decision.

Table 14.6 Types of NHS consent forms.

Consent form 1	Patient agreement to investigation or treatment
Consent form 2 Under 16 years of age	Parental agreement to investigation or treatment for a child or young person under 16 years. If a child is under 16 years but deemed to be competent they can consent to their treatment
Consent form 3	Patient/parental agreement to investigation or treatment (procedures where consciousness not impaired)
Consent form 4	Form for adults who are unable to consent to investigation or treatment

2. Voluntary decision-making – the patient has made the decision.
3. Ability – the patient has the ability to make an informed decision.

The Department of Health (2009)⁵⁴ in the UK has recently introduced four consent forms for NHS patient interventions (Table 14.6).

Patient information

It is recognized that good communication is central to the clinician–patient relationship and to good clinical care. Patients require information about the options available for management of their third molars, together with an explanation of the operation/procedure itself.

At the preoperative appointment, the potential outcome of any chosen course of action, adverse or otherwise, should be explained to the patient in terms that they can easily understand. Details should be noted in the patient's records and should include aspects relating to the patient's quality of life. In addition, care should be taken to explain to the patient the consequences of not having the tooth removed and other treatment options which may be required in this event.

The information provided should be sufficient to enable the patient or their carer to make a valid informed decision and give consent (a minimum of 24 hours prior to surgery).⁵⁵ The US National Institutes of Health recommend that patients should be informed of potential surgical risks including any transitory condition that occurs with an incidence >5% and any permanent condition with an incidence >0.5%.^{4,5} If the patient is at increased risk of inferior alveolar nerve injury based on radiographic assessment then the patient must be warned of the increased odds.^{26,55,56}

At the time of surgery, the patient should be reminded of the possible complications and side-effects of the operation. The operator should ensure that consent has been obtained, that the patient still wants to go ahead with the procedure, and a note should be made in the patient's records.

At the time of the operation the patient should know:

- How to contact the surgeon in case of emergency.
- How to look after their mouth postoperatively (sutures, etc.).
- Possible complications and side-effects of the operation in general and any problems specific to the operation undertaken.
- Any drug therapy required.
- Whether a review appointment is required and if so, when.
- That postoperatively the referring practitioner will receive a letter detailing the treatment undertaken.

The medically compromised patient

There are some medical conditions which have to be taken into consideration. Medically compromised patients are covered in more detail in Chapter 3. Table 14.7 presents some medical conditions which are especially important to consider for third molar surgery.

Every effort should be made at the time of the operation to minimize or avoid complications and side-effects of the operative procedures.

Medications commonly used in oral surgery

Anxiety and pain relief

General anesthesia

Methods of anxiety and pain relief include local analgesics, local analgesia with intravenous sedation, and general anesthesia. It is common practice to use local analgesia. Appropriate selection of patients for local analgesia versus local analgesia plus sedation, or outpatient or inpatient general anaesthetic is often confusing for those new to the specialty. In essence surgery taking longer than 40 minutes should be limited to patients with sedation-augmented procedures or outpatient general anesthesia. Medical conditions may restrict the provision of day case anesthesia and lead to the necessity of general anesthesia (Fig. 14.19). In general dental practice, the former two methods are considered appropriate, but still require suitable facilities to be available.⁶⁵ General anesthesia may be needed for complex and lengthy procedures but it must be recognized that local anesthesia carries less risk.⁶⁵ Recent General Dental Council guidance emphasizes that general anesthesia is a procedure which is never without risk and that *"in assessing the needs of an individual patient, due regard should be given to all aspects of behavioural management and anxiety control before deciding to prescribe or to proceed with treatment under general anaesthesia"*.⁶⁶

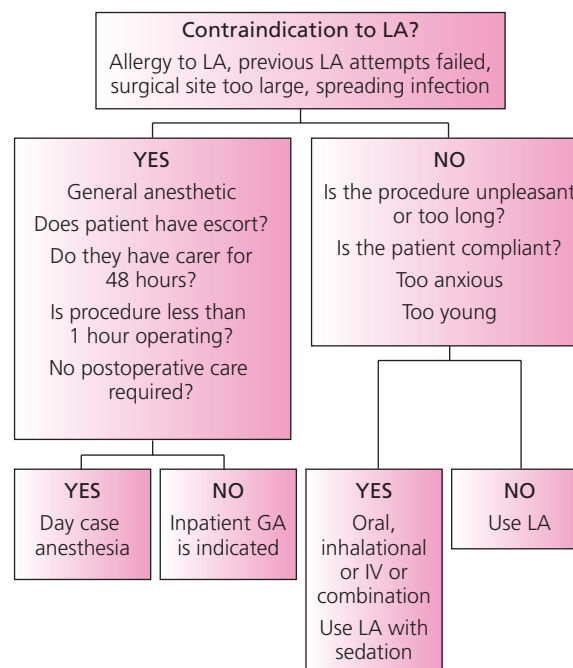


Fig. 14.19 Local analgesia/anesthesia/sedation flow chart.

Sedation

One must be aware of differences between sedation practice in the UK and the US. UK conscious sedation is limited to single drug use, commonly midazolam, and maintenance of verbal communication with the patient.⁶⁷ US sedation is nearer a UK-defined general anesthetic with use of multiple drugs (commonly propofol and midazolam) and laryngeal mask airway. Day cases by definition in the UK are cases admitted and discharged within a day (12 hours), however day cases in the US are allowed to stay for one night and are generally discharged within 24 hours. Most oral surgeons in the US undergo anesthesia training for provision of non-conscious sedation in their surgeries. Conversely all general anesthetics and multiple drug sedations are undertaken in the hospital setting in the UK.

Local analgesia

There have been several recent reports of successful routine removal of upper third molars with placebo palatal local anesthesia (21 patients, split-site design using buccal lidocaine)⁶⁸ and without palatal analgesia (root canal treatment, 53 patients using buccal articaine).⁶⁹ The author routinely elevates maxillary third molars with buccal lidocaine infiltration as the discomfort of the palatal injection appears to be significantly greater than that perceived during extraction.

Many practitioners already undertake routine dental and third molar extractions with articaine-only infiltrations. Currently there is no evidence base for this practice, however in the author's department a prospective randomised study is underway evaluating the scope of articaine infiltration only for mandibular dentistry.

Table 14.7 Common medical problems influencing management of dental patients.

Medical condition	Recommendations	Reference
Infective endocarditis (IE)	Antibiotic (AB) cover for dental treatment not recommended Patients at risk of IE should maintain high standard of oral health Chlorhexidine mouthwash is not recommended Dentists must warn patients about IE symptoms	NICE draft 2007 ⁵⁷ American Heart Society (1997) ⁵⁸
Steroid	No evidence base for steroid cover Patients should take their normal dose Cover is required for surgery under GA or if patient has missed their routine steroid dose unless patients receiving <10 mg of prednisolone or equivalent in which case they do not need steroid cover Steroid cover may be required if steroid cover has been taken within 3 months Moderate surgery – preoperatively 25 mg of IV hydrocortisone followed by 25 mg IV every 8 hours for 24 hours. Usual preoperative dose then continued Major surgery – usual dose of steroids preoperatively, then a 50 mg of IV hydrocortisone followed by 50 mg IV 8 hourly for 48–72 hours. Continue this infusion until the patient has started light eating, then restart normal preoperative dose	EMIS 2007 ⁵⁹
Warfarin Coumadin	Check on day of surgery or 3 days beforehand No adjustment of warfarin is required unless international normalized ratio (INR) >4 for routine extractions Multiple or difficult surgical extractions may require heparin management undertaken by hematologist	North West Medicines Information Centre 2007 ⁶⁰ FDA 2009 ⁶¹
Tranexamic acid	Mouthwash is not recommended	North West Medicines Information Centre 2007 ⁶⁰
Bisphosphonates	Prior to treatment with an IV bisphosphonate a dental examination with appropriate preventative dentistry should be considered The surgeon must complete all necessary invasive procedures prior to IV bisphosphonates. Prophylactic extractions should be undertaken with a view to short arch strategy in order to prevent necessary future extractions and surgery. Regular dental check-ups should be arranged, with necessary oral hygiene, chlorhexidine mouthwashes, and topical fluoride treatments After/during treatment avoid extractions/surgery On development of osteonecrosis of the jaw while taking a bisphosphonate, dental surgery may exacerbate the condition If an invasive dental procedure cannot be avoided there are no data to suggest whether discontinuing bisphosphonate treatment reduces the risk of osteonecrosis of the jaw	Marx <i>et al.</i> 2007 ⁶² Mehrotra & Ruggiero 2006 ⁶³
Previous radiotherapy involving the jaws	Similar recommendations to IV bisphosphonates No evidence that antibiotics improve outcome Pentoxifyline 400 mg q12h, vitamin E 1000 IU, chlorhexidine 10 ml q6h Hyperbaric oxygen may have an effect? No evidence base Ultrasound therapy no evidence Short arch (leave 5–5 and remove molar teeth) prior to radiotherapy Oral fluoride supplements before, during radiotherapy and afterwards	Katsura <i>et al.</i> 2008 ⁶⁴
Patient with human immunodeficiency virus (HIV)	Viral load HIV RNA 50 k/ml high infectivity CD4 count: >200 cells/ml Rx as normal <200 cells/ml check blood profile Check for neutropenia, thrombocytopenia, liver function, coagulation screen	

There is continued debate about high concentration local anesthesia being related to increased incidence of inferior alveolar and lingual nerve injuries during block injections but there is a limited evidence base. The nerve injury may be related to concentration of articaine rather than the agent itself. The use of 5% lidocaine was abandoned many years ago due to its neurotoxicity. The AAOMS white paper⁵ states that there was no difference in nerve injuries with articaine in a longitudinal study in the US but there was an increased incidence with prilocaine.⁵ Some reports state that 4% articaine is no more effective than 2% lidocaine⁷⁰ and routine 2% lidocaine IAN blocks are generally recommended. Increasingly practitioners are undertaking articaine buccal infiltration-only extractions in adults and children and the author believes this will soon become standard care with use of top-up lidocaine 2% IAN blocks when necessary.

Antibiotics

The use of prophylactic antibiotic therapy following third molar surgery is a common, if not universal, practice. There is very little evidence to support its routine use and there is, however, a growing body of data to suggest that this practice wastes resources with very little prospect of health gain. The use of broad-spectrum, third-generation antibiotics seems completely unacceptable;^{71–73} however, in severe cases, where there is acute infection at the time of operation, significant bone removal, or prolonged operation, antibiotics should not be withheld. The bacteriology of the wisdom tooth socket and the research that was related to it was beautifully described by MacGregor.⁷⁴ He concluded, “*Antimicrobial drugs appear to have a marginal benefit in third molar surgery when clinically uninfected teeth are removed.*” Over 30 years later, little appears to have changed.

Analgesia

Third molar surgery is the gold standard used for the evaluation of analgesics for the management of moderate to severe acute postsurgical pain.⁷⁵ Peak pain levels occur after the local analgesia has worn off at 4–6 hours and lasts for up to 12 hours.^{76,77} Normal practice is to prescribe or advise oral analgesics such as paracetamol or ibuprofen for outpatients.⁷⁶ For inpatients a number of options including non-steroidal anti-inflammatory agents and opiates are commonly prescribed. Pre-emptive analgesia may be considered, however evidence base does not support this practice.⁷⁸ The most effective analgesia is diclofenac (number needed to treat (nnt) 2.3) followed by Ibuprofen 400 (nnt 2.6) 600mg every 6 hours.⁷⁸ Many authors routinely recommend combined non-steroidal anti-inflammatory agents (if not contraindicated) with paracetamol as the best combination for acute postsurgical pain,

avoiding complications associated with opiate and opioids.⁷⁸

Steroids

Where there is a risk of significant postoperative swelling, pre- or perioperative administration of dexamethasone or methylprednisolone has been shown to reduce swelling and discomfort.^{78–80} For nerve injury, the window of opportunity for treatment with high-dose prednisolone is within the first 72 hours (some even say the first 48 hours). The protocol based on treatment for facial palsy may have a role in minimizing inflammatory injury to peripheral nerves. This involves a step-down prednisolone treatment of 20mg for 5 days, 10mg for 5 days then 5mg for 5 days.

Chlorhexidine

There is sufficient evidence base for the routine prescription of chlorhexidine prior to or after third molar surgery in order to reduce alveolar osteitis (dry socket).⁸¹

Surgical procedure

The procedure is variable and is influenced by patient factors, including the type of impaction and anatomy of surrounding structures, for example proximity of the inferior alveolar and lingual nerves. Generally surgery involves the raising and protection of soft tissue flaps and bone removal with either chisel or bur with water cooling irrigation. There is conflicting evidence as to the most appropriate form of protection for the lingual nerve.^{82,83} However the evidence base highlights that temporary lingual nerve injury is reduced when lingual access is avoided.^{42,46}

The whole tooth should be removed and wound toilet completed. Any suspected pathological material should be sent for a histopathology report.

Principles of exodontia of third molars without using a flap

Suitable instrumentation for surgical extractions of third molars is shown in Fig. 14.20. The surgeon should scrub in using sterile gloves, mask, and head cover and draping of the patient should be carried out (Fig. 14.21). A mouth prop on the contralateral side allows the patient to relax with an open mouth (Fig. 14.22). It is important to support the patient's head and mandible (Fig. 14.23).

Simple forceps extraction of erupted third molar is similar to any other extraction using forceps and is covered in Chapter 12. Elevation of a mandibular molar is possible when the tooth is erupted and vertically impacted. If the tooth is minimally meso- or



Fig. 14.20 Suggested instrumentation for routine dentoalveolar surgery.

From lower right to left:

Toothed forceps

Needle holders (Olsen Hagar needle holders come with cutting surface so that additional scissors can be dispensed with)

4/0 Vicryl Rapide suture

Couplands 1–3

Warwick James

Lax tongue retractor

Minnesota buccal retractor (with Cawood modification with 'Kilner' end)

Mitchells trimmer

Wards perisoteal (or Freer) elevator

Blade handle and #15 blade

From top right to left:

Saline (or chlorhexidine solution)

10–20ml syringe for regular irrigation

5 pack of gauze

Mackesson mouth props

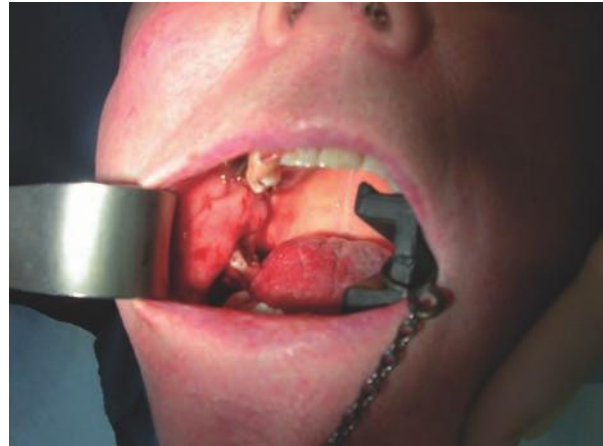


Fig. 14.22 Prop in mouth (allows patient to 'relax' with mouth open and reduces the strain on the TMJ).



Fig. 14.21 Patient positioning for surgery.



(a)



(b)

Fig. 14.23 (a) Position of surgeon's hands to support patient. (b) Support of mandible whilst retracting with Minnesota retractor.

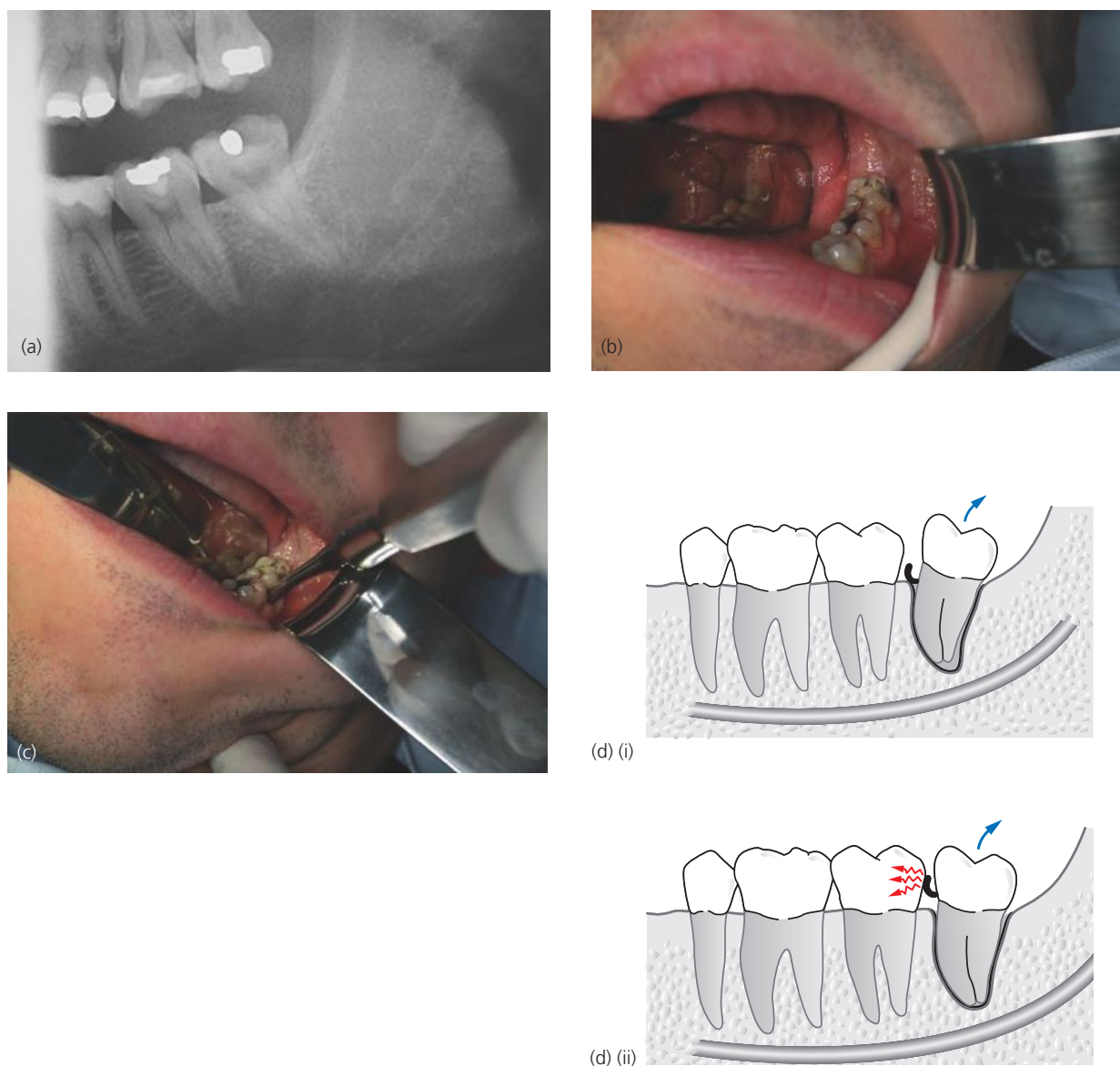


Fig. 14.24 Elevation of mandibular vertical, non-impacted, lower left third molar. (a) Preoperative radiograph. (b) Clinical view of non-impacted 38. (c) Placement of straight Warwick James elevator either at the mesial or buccal aspect of the lower left 8 in order to elevate. (d) (i) Ideal path of elevation of a single rooted erupted lower 8; (ii) when placing the elevator at the mesial aspect, avoid pressure on the distal aspect of the adjacent second molar. This may result in fracturing of the crown of the second molar, particularly if it is heavily restored or non-vital.

disto-angularly impacted, elevation may be possible, particularly in younger patients and with favorable root morphology (Fig. 14.24).

If the tooth is partially erupted, a relieving incision and elevation (Warwick James method) is carried out (Fig. 14.25). This is a very useful method that allows the surgeon to place a small (less than 3 mm) incision through the disto-buccal aspect of the gingival cuff overlying the partially erupted tooth.

After the cuff is relieved (similar to an episiotomy) a Wards periosteal elevator can be used to establish if any bone is overlying the distal aspect of the crown of the tooth. If there is no overlying bone, the vertical or mesoangular tooth can be elevated with a straight Warwick James elevator as the mucosal impaction has been relieved; often no further mesial relieving mucosal incision or bone removal is necessary.

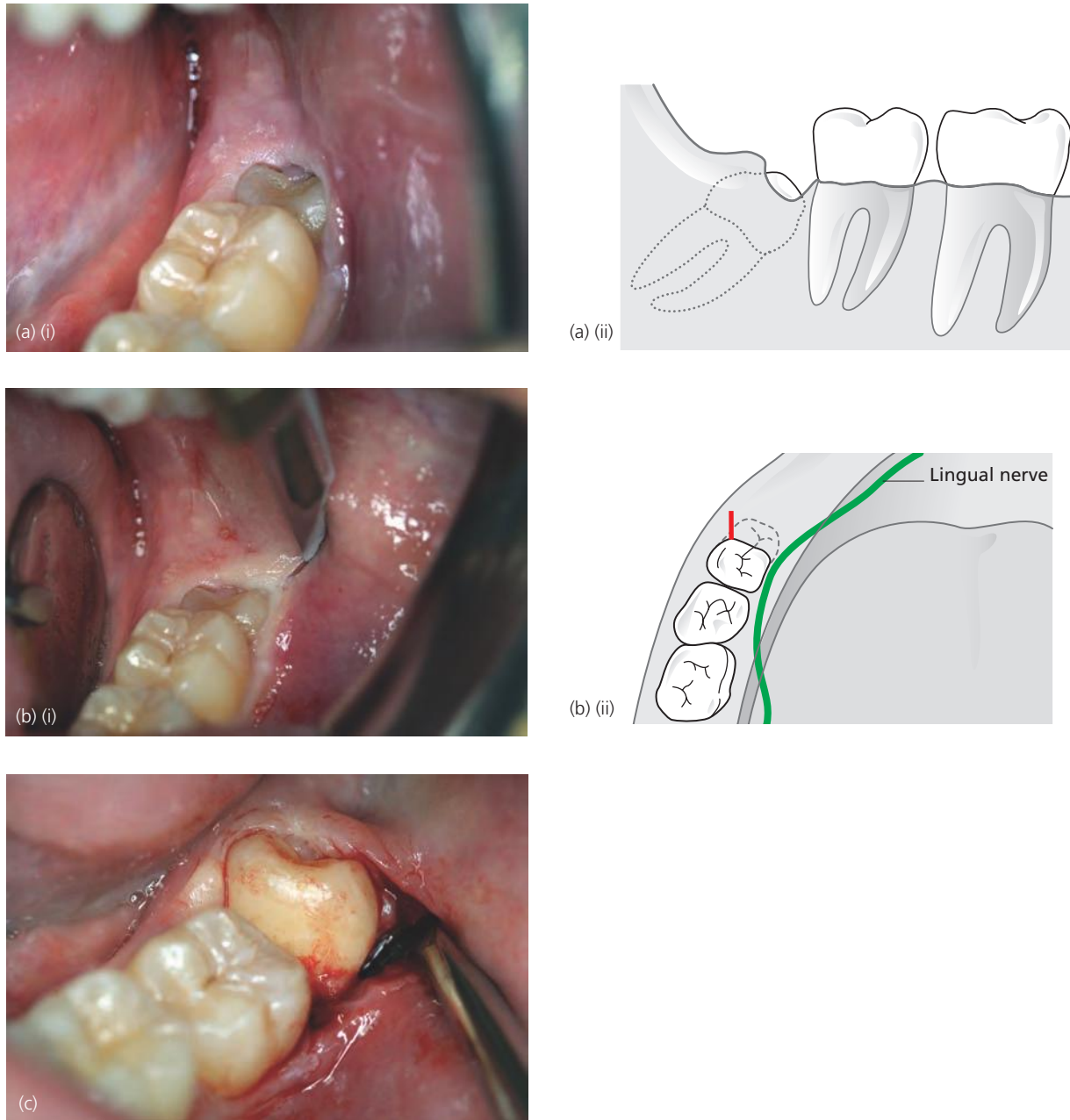


Fig. 14.25 (a) Partially erupted 38 before incision. (b) Relieving incision and elevation for partially erupted third molar. Incision using #15 blade. Essentially the blade is placed buccally at the distal aspect of the gingival cuff and the incision extended to the periodontium of the 38 (length of incision is usually 4–5 mm). (c) Right-curved Warwick James is placed at the mesial aspect of the lower left 8 and the tooth is elevated superiorly and buccally. Once the tooth is mobilized and superiorly displaced the same Warwick James can be placed in the furcation of the lower left 8 and the tooth elevated.

Surgical extraction of third molars

Flap design

A single “slash” is commonly used to access unerupted maxillary third molars (Fig. 14.26). Maxillary third molars rarely require sectioning because the overlying bone is thin and relatively elastic. Usually extrac-

tion is accomplished by removing additional bone rather than by sectioning. For maxillary third molars, bone removal is done primarily on the lateral aspect of the tooth down to the cervical line to expose the crown. Frequently, the bone is thin enough that it can be removed with a periosteal elevator or a chisel with hand pressure.

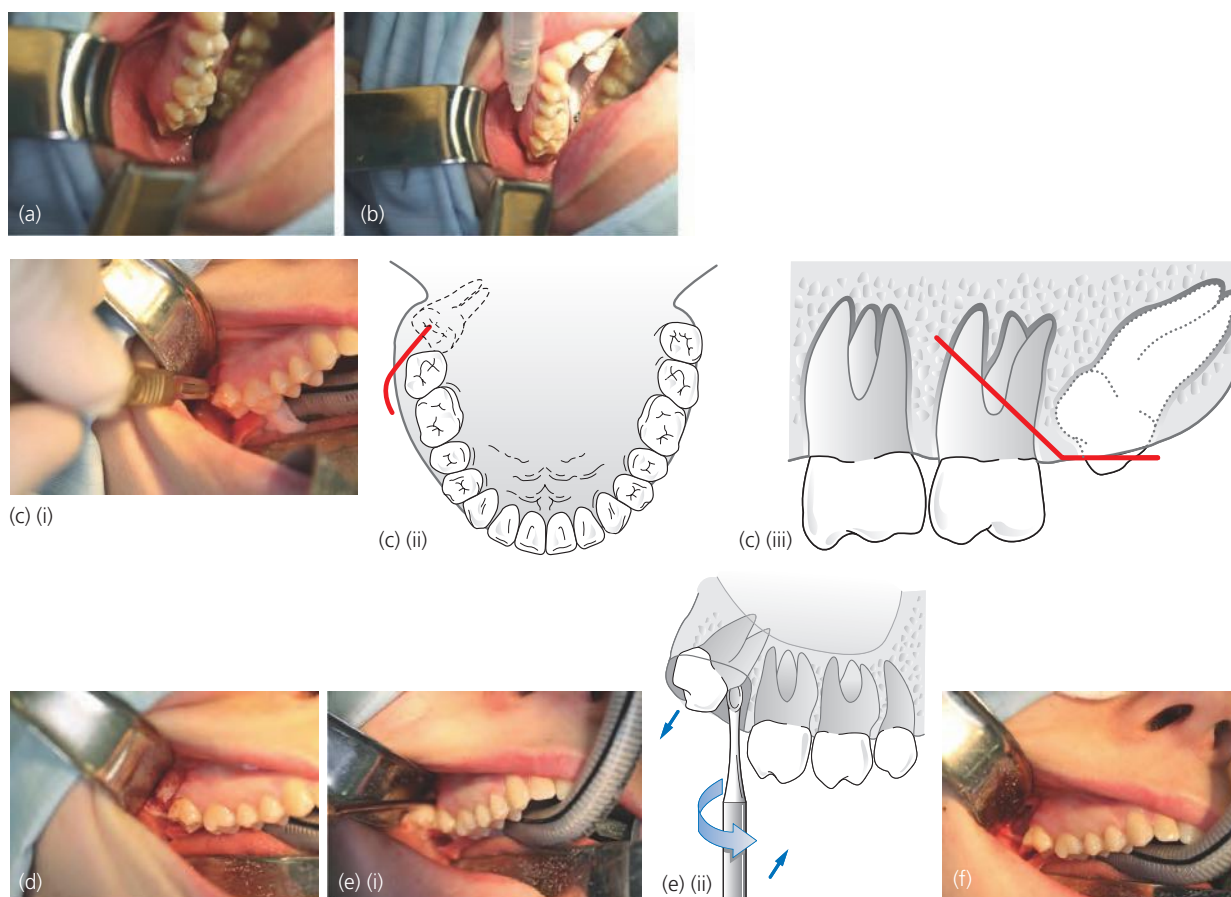


Fig. 14.26 Flap design for removal of maxillary third molar. (a) Preoperative view of 18. (b) Local anesthesia – buccal infiltration. (c) Placement of Minnesota retractor and incision with #15 blade. (d) Elevation of buccal flap, 18 retracted with Minnesota retractor. (e) Elevation upper root 18 with right-curved Warwick James elevator. The tooth is delivered with curved elevators applied on the mesio-buccal with rotational and lever types of motions. The tooth is always delivered in a disto-buccal and occlusal direction. (f) Socket 48 after extraction. Irrigate socket and use gloved finger to ensure no bony spicules are present and that the buccal plate is not fractured. No suture is necessary as the buccal flap remains sealed by the pressure of the cheek.

Mandible envelope flap

See Fig. 14.27a. A disadvantage is that the long distal incision to allow reflection of the flap is more likely to expose distal bone of the 8 and thus tempt the surgeon to drill distal bone and place the lingual nerve more at risk.

Mandibular buccal triangular flap

See Fig. 14.27b. The advantage of this minimal access flap is that there is sufficient exposure for buccal bone removal and tooth section without extensive periosteal stripping of the buccal aspect of the mandible or temporalis muscle attachment, resulting in significantly less postsurgical pain and swelling. The triangular flap is best used with a Minnesota retractor providing buccal and flap retraction in one.

Mandibular bone removal

See Fig. 14.27c. An air-driven or electric hand piece with round or fissure burs is used. If using the preferred buccal approach to minimize lingual nerve injury, a fissure bur should be used to get clean sec-

tions for tooth splitting for elevation. For mandibular teeth, bone on the occlusal and buccal (**NOT distal**) aspects of the impacted tooth is removed beyond the cervical line down to the roots of the tooth. It is advisable not to remove bone on the lingual aspect due to the likelihood of damage to the lingual nerve.

Sectioning the tooth

See Fig. 14.27d. Using a fissure bur allows the surgeon to get a narrow clean section into the dental pulp then extend the “incision” within the confines of the tooth. At no point should the bur breach the mesial, distal or lingual surfaces of the tooth. Thus one achieves a “partial” section of the tooth covering about 40% of the split surface.

Planning tooth section

Planning potential tooth sectioning is important and saves operative time (Fig. 14.27e). Depending on the depth of application point and the angulation, the crown or root will need to be sectioned. When sectioning the roots the surgeon can use the dental pulp

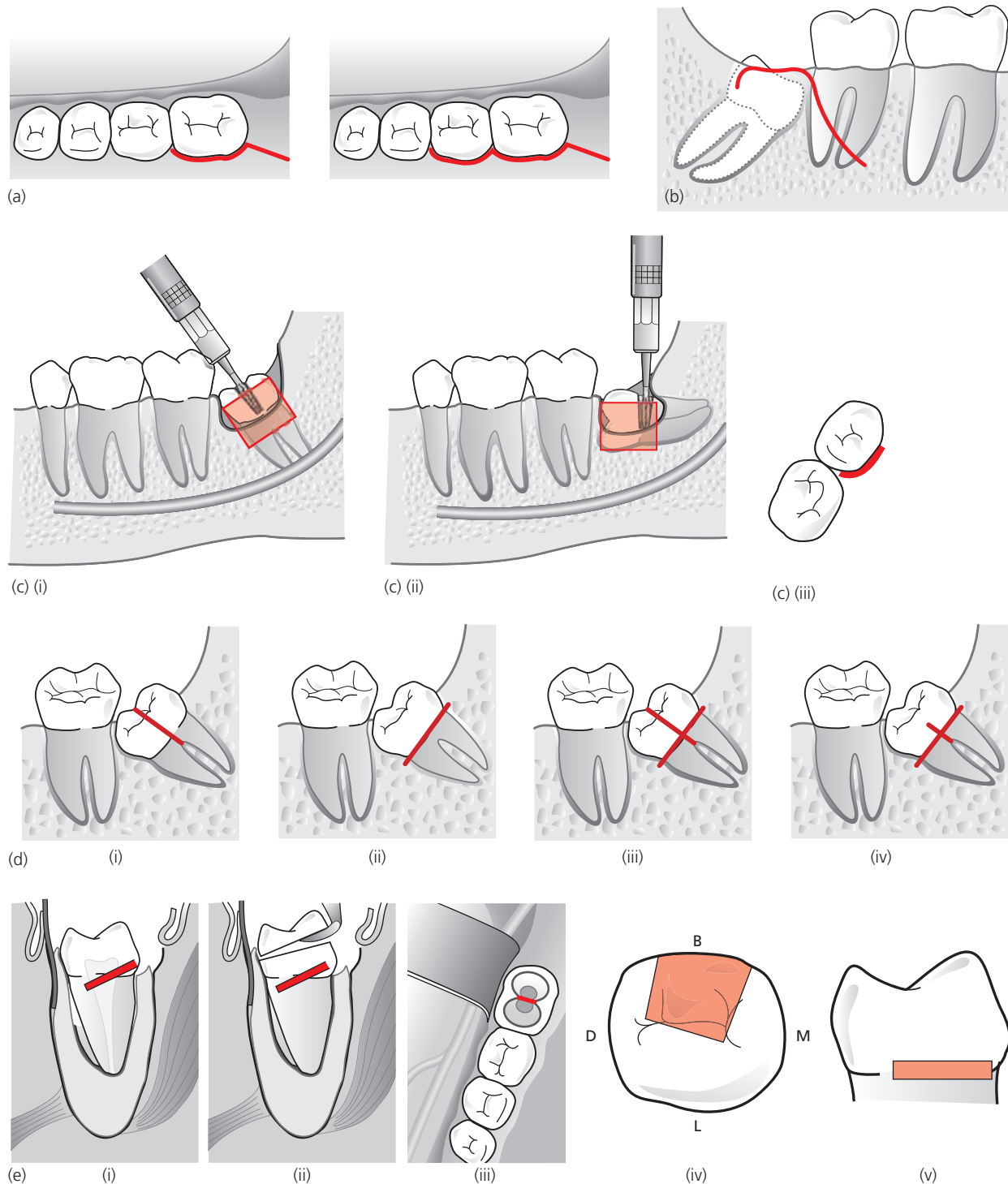


Fig. 14.27 (a) Mandibular envelope flap, not recommended due to the long distal extension required. (b) Triangular flap. (c) Bone removal is performed using a fissure bur at full speed adjacent to the enamel of the crown. The gutter or trough should be kept narrow (bur's width) and extend **inferiorly only** to the furcation of the tooth to allow elevation of the roots later without further bone removal. The bone cut should *not* extend beyond the mesial aspect of the crown *or* the buccal aspect of the tooth distally (see diagram). (d) Surgical approach to sectioning of the tooth for vertical, mesial, horizontal, and distoangular angulation. (i) Sectioning options for mandibular mesoangular third molar impactions. This view shows a cut longitudinally through the furcation. (ii) Same as (i), but with an additional cutting of the mesial crown, which is wedged under the second molar. (iii) Crown and root separation. The crown is removed and then the root is delivered into the original crown space. (iv) Crown removal as in (iii), but then the roots are divided longitudinally through the furcation and moved into the crown space for removal. Mesoangular – no section, i, ii, iii, iv; vertical – none, i; distoangular – none, iii, iv; horizontal – none, ii, iii, iv. (e) Using a fissure bur as near to the crown root junction of the 8 and as near to 90 degrees to the tooth (i). The tooth should be perforated until one feels the pulp chamber (ii). Then the cut should be lateralized or verticalized depending on whether you intend to decoronate the tooth or vertically section it. Using a straight Warwick James elevator the crown should be "factured off" (iii). If the roots require sectioning the fissure bur cut should *not* extend outside tooth material (iv) and the ideal underside of the sectioned crown should be cut only from the buccal aspect covering about 40% of the split surface (v). From the buccal aspect the crown section cut should be confined to the buccal surface and *not* extend through distal, mesial or lingual surfaces of the tooth.

chambers to estimate where to split the roots. Again at no point should the drill cut extend through the lingual aspect of the roots. Often drilling a small hole large enough to engage the tip of a curved Warwick

James elevator is useful in gaining a “purchase point” to elevate the roots. Figs 14.28–14.31 illustrate surgical extraction of third molars.

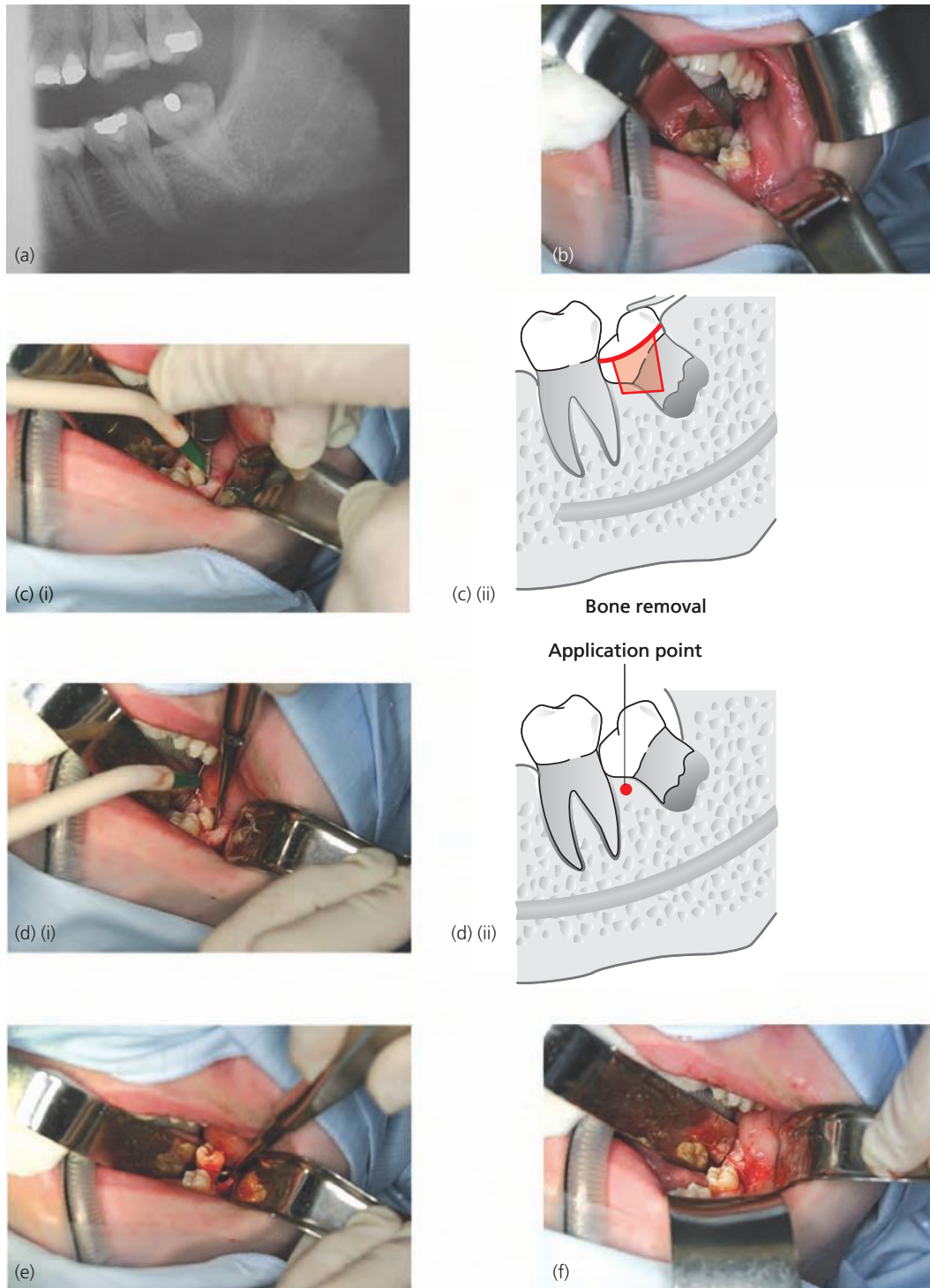


Fig. 14.28 Buccal flap with buccal bone removal for partially erupted third molar with deeper depth of impaction. (a) Preoperative film, 38 erupted non-impacted. (b) Clinical picture, 38 erupted, non-impacted. (c) Buccal bone removal with fissure bur to achieve narrow gutter. (d, e) Tooth elevated with contralateral (right) Warwick James elevator at mesial application point. This elevates the conical rooted tooth in a vertical and buccal direction. As the tooth moves superiorly the same Warwick James can be placed in the now exposed furcation of the tooth and final delivery is controlled. (f) After irrigation of the socket the buccal triangular flap is secured with a single 4/0 Vicryl Rapide suture.

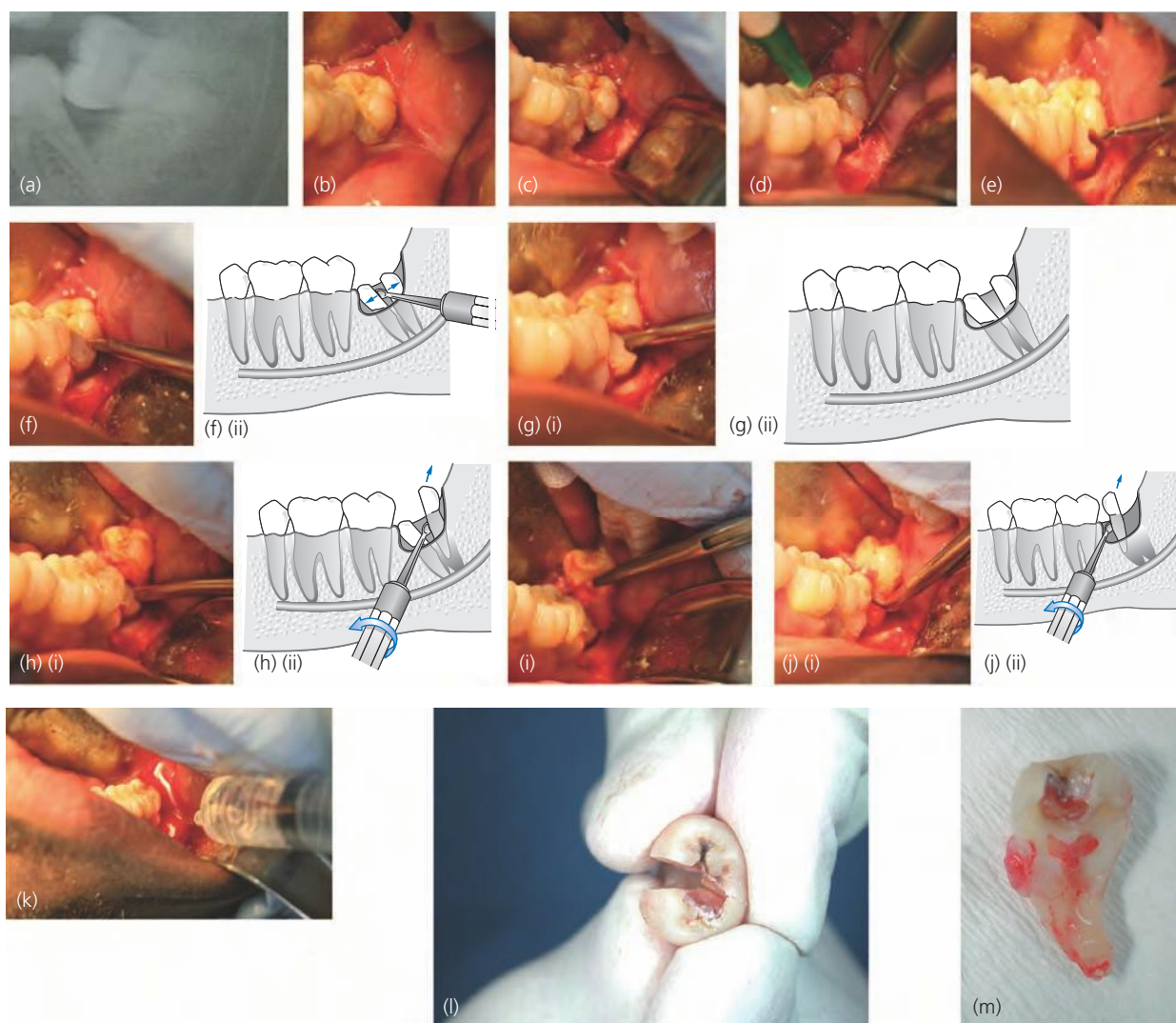


Fig. 14.29 Surgical removal of mesoangular tooth with buccal bone removal and vertical section. (a) Preoperative radiograph. (b) Clinical picture of partially erupted lower left 8. (c) Triangular buccal flap raised and retracted with Minnesota retractor. (d) Removal of buccal bone with fissure bur to form a gutter. (e) Vertical section of tooth using a fissure bur. (f) Using a straight Warwick James to "crack" the tooth into vertical sections. (g) Tooth cracked vertically. (h) The distal section of tooth elevated with No 1 Couplands. (i) The distal segment is elevated. (j) Using a contralateral Warwick James elevator mesially the mesial segment of tooth is elevated. (k) Irrigation of socket with saline. (l) Reapproximation of tooth out of the mouth to demonstrate that the tooth cut only penetrates into the dental pulp (m) not through the whole tooth in order not to penetrate the lingual aspect of the tooth.

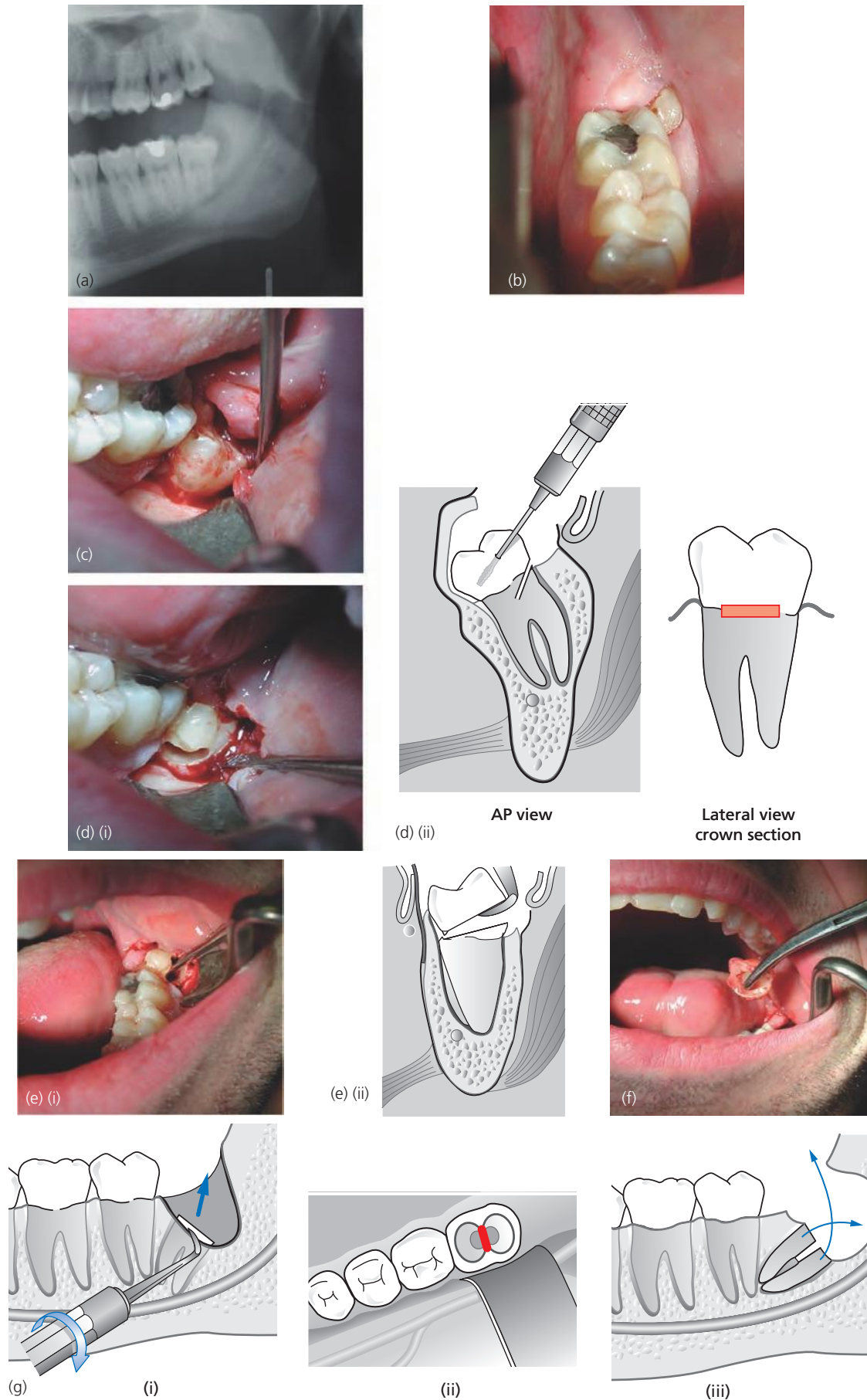


Fig. 14.30 Extraction of a distoangular 38. (a) Preoperative radiograph. (b) Clinical appearance. (c) Buccal bone removal. (d) Section of crown. (e) Elevation of crown. (f) Elevation of the crown, then elevation of the roots with curved Warwick James elevator. (g) (i) Use Warwick James elevator to elevate roots together. If roots are immobile, section with fissure bur (ii) then elevate roots separately (iii).

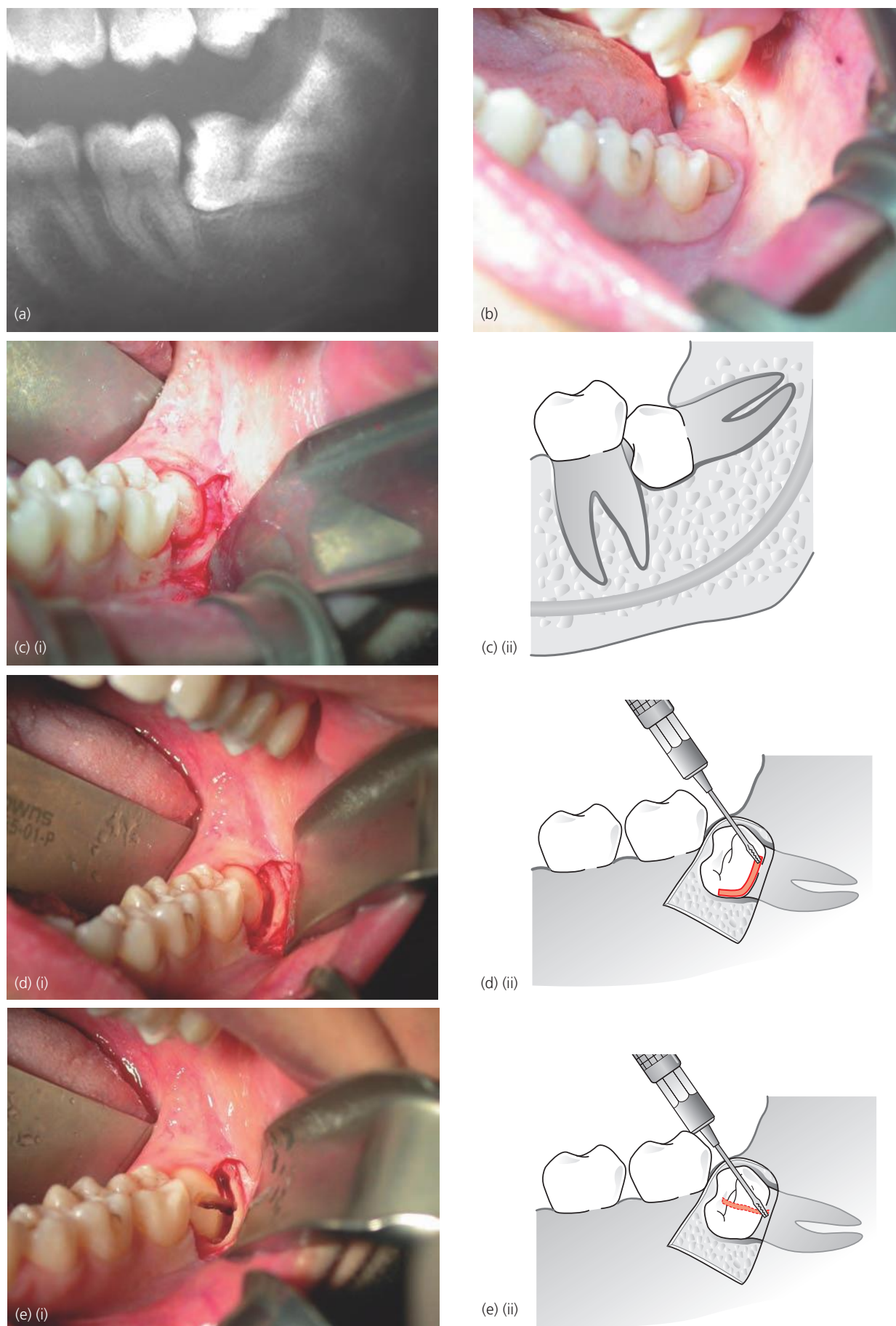


Fig. 14.31 Surgical removal of a horizontal 48. (a) Preoperative radiograph. (b) Preoperative clinical picture. (c) Flap raised using Wards periosteal elevator. (d) Removal of buccal bone with fissure bur to form a gutter. (e) Splitting of vertically sectioned tooth using Warwick James straight elevator. (*cont'd*)

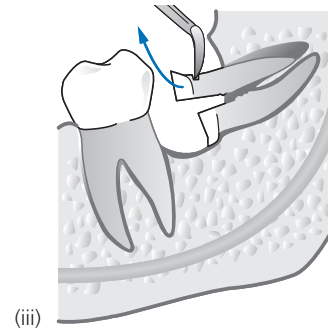
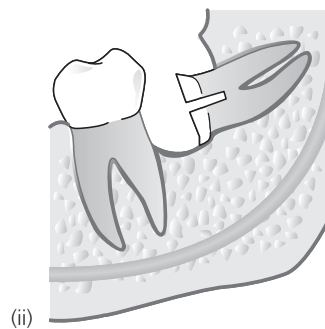
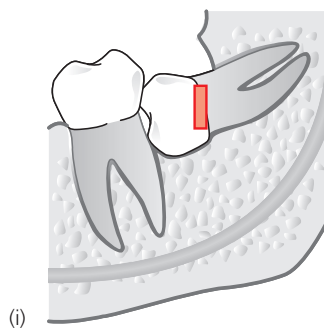
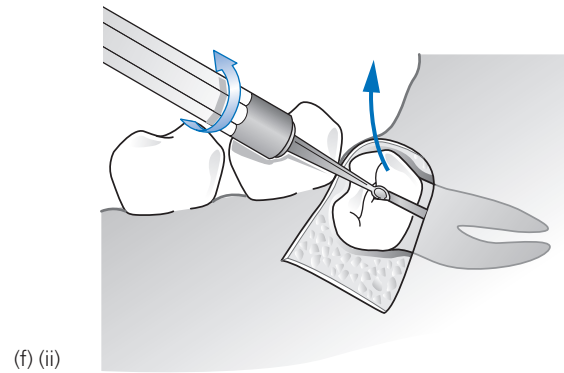


Fig. 14.31 (cont'd) (f) Elevation of distal root using No. 1 Couplands. (g) Elevation of mesial root using curved left Warwick James elevator. (h) Approximation of wound prior to suture. (i) Alternative sectioning of a horizontal tooth may require decoronation (notice the angulation of the crown–root junction cut with narrowing of section inferiorly to maximize the ability to elevate the crown in one piece (i). If the crown is too impacted then section the crown into two pieces and then elevate the crown. This leaves the roots *in situ* (ii). Using the pulp chambers as “guide” one can section the roots with a fissure bur and then elevate the roots individually. The order will depend on root curvature. Drilling a small notch into the root surface will often facilitate root elevation (iii).

Lingual flap elevation and lingual retraction

Most third molars can be removed by using a purely buccal technique preventing the necessity to encroach on the lingual tissues or to remove distal, disto-lingual or lingual bone.^{84,85} However, on occasion where the tooth is malpositioned, or for other reasons, it may be necessary to remove distal, disto-lingual, or lingual bone. If this is necessary, a subperiosteal lingual flap is raised with a suitably curved periosteal elevator that can stay in contact with the lingual plate of bone and not encroach on the lingual soft tissues.^{86,87} Once a subperiosteal lingual flap has been raised, a lingual retractor is placed. The evidence base suggests a significantly higher incidence of temporary lingual nerve involvement when a lingual retractor was used.⁴⁶ Thus this author does not advocate the routine use of lingual access for third molar surgery unless there are roots perforating the lingual plate that require removal, or the position of the tooth and associated pathology dictate its necessity.

Alternative procedures

Coronectomy

Coronectomy (also known as partial tooth removal, partial odontectomy or intentional root retention) (Figs 14.32 and 14.33) has been reported in five articles reporting series of patients. Four were case series,^{88–91} the fifth article was a randomized controlled trial.²⁶ In all cases, coronectomy was suggested as a technique of partial root removal when panoramic radiographic imaging suggested an intimate relationship between the roots of the lower third molar and the IAN and the vital tooth still needed to be removed. (Note: CBCT was not available at the time the studies were conducted.) Antibiotics are rarely recommended. Two papers mentioned that the socket should be closed primarily. Two papers stated that lingual retraction was recommended in all cases to protect the lingual nerve.^{88,89} Two papers did not use lingual retraction.^{90,91} This recommendation may reflect the different techniques applied in the studies; when complete section of the crown from the roots is undertaken then lingual protection would be advised,⁸⁹ however Renton *et al.*²⁶ recommend partial section of the crown as in the buccal technique described in this chapter which requires no lingual protection. Inadvertent root mobilization at the time of attempted coronectomy occurs in 3–38% of patients, thus failing to achieve coronectomy and the roots needing to be removed at the time of primary surgery.^{88–91} One paper noted a 38% failure rate at primary surgery, because the roots were only sectioned about halfway into the pulp chamber, before an attempt was made to split the roots from the crown.²⁶ Mobilization of the roots occurred in many cases and did result in an 8% incidence of temporary IAN involvement but no

permanent injuries. Factors that increased the likelihood of root mobilization were female patients, conical roots, and age <30 years.²⁶ Later root removal may be required in 2–6% of cases depending on the technique of coronectomy employed.^{88,89} Subsequent root migration is mentioned in all papers: 14–81% showed later migration of the roots towards the superior border of the mandible. Roots may require removal when they erupt later and become infected but often the roots have migrated away from the IAN thus minimizing potential injury to the nerve. Prevention of permanent IAN injury occurred with coronectomy technique with one exception: O’Riordan⁸⁸ reported this due to inadvertent drilling, in 1% of patients in one study.

It is generally recommended that when imaging suggested an intimate relationship between the roots of the lower **vital** third molar and the IAN and the tooth still needs to be removed, consideration should be given to coronectomy with retention of the portion of the roots associated with the IAN. This procedure may be contraindicated in patients who are immunocompromised or have had previous radiotherapy in the region.

Eruption of third molar

Many studies have been done to attempt to predict the probability of third molar eruption, most of them using dissected skulls or lateral cephalic radiographs. The lateral cephalic radiograph is an excellent aid in performing cephalometric diagnoses. Using dental pantomograms, Quirós and Palma presented a method for prediction of third molar eruption and optimal timing for extractions of third molar into second molar space. It is not possible to predict eruption of third molars in all cases; adequate space between the anterior border of the mandible and the distal of the mandibular second molar seems to be necessary to allow successful eruption to the occlusal plane. The AAOMS⁵ white paper highlights several issues:

1. The most significant variable associated with third molar impaction is inadequate hard tissue space, with the vast majority of impacted third molars having space/crown width ratios of less than 1.^{92,93}
2. It is possible to measure space for eruption to the occlusal plane using a variety of radiographic techniques.^{92,94}
3. Third molars that remain impacted after the age of 25 may still change in position.⁹⁴

Orthodontic traction

A recent suggestion is to orthodontically “extract” third molars in those patients at risk of bisphosphonate-induced osteonecrosis. This method may assist in preventing this unfortunate complication.⁹⁵

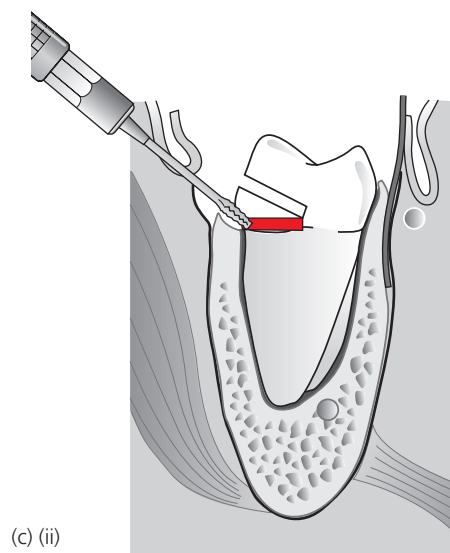
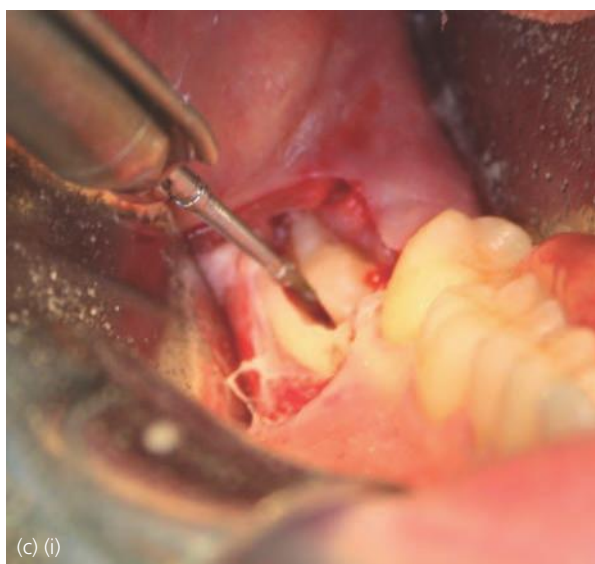
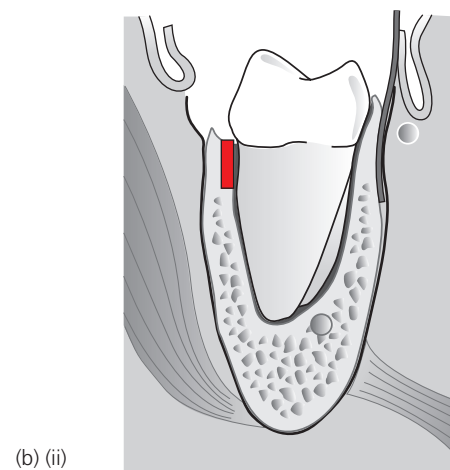
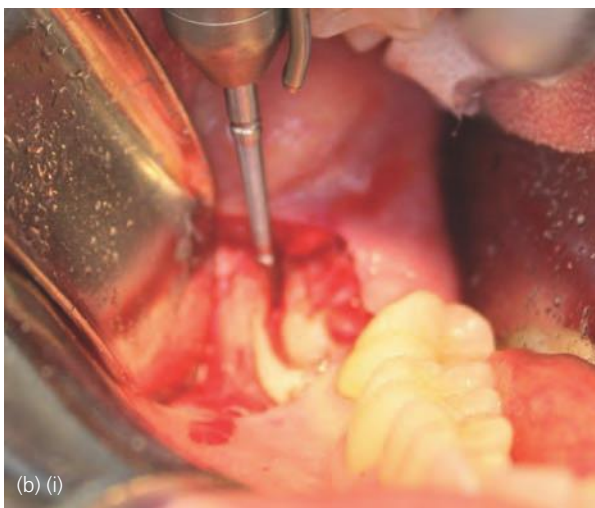


Fig. 14.32 Coronectomy. (a) Radiograph of vital vertically positioned lower right 8 with roots crossing IAN canal. (b) Buccal bone removal with fissure bur. (c) Sectioning of crown from roots level with the crown root junction subsequent to buccal bone removal with fissure bur. (*cont'd*)

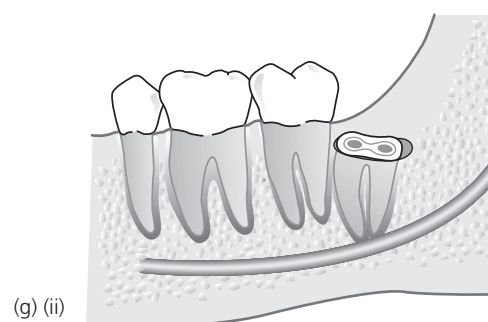
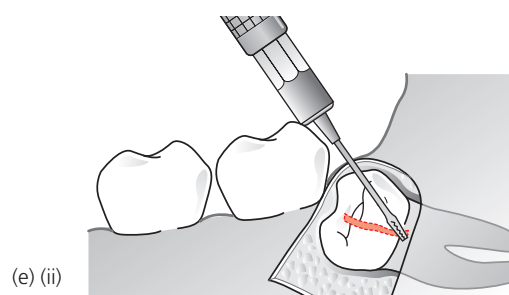
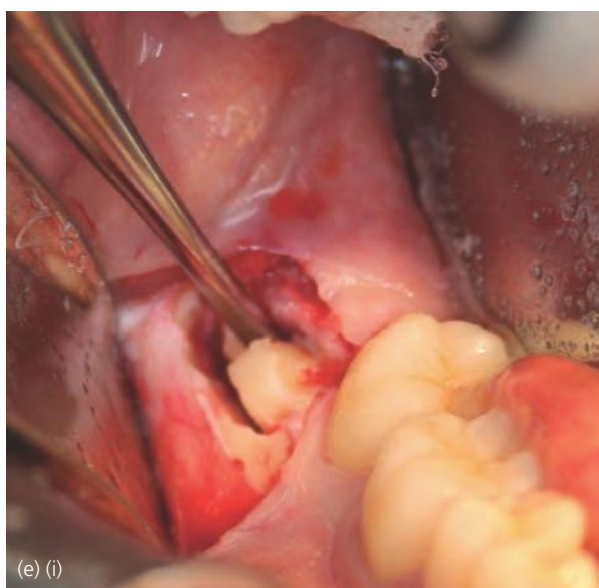


Fig. 14.32 (cont'd) (d) Elevation of crown from roots with straight Warwick James. Usually the crown will elevate whole but if it is still impacted (too wide for space) then, using a fissure bur, the crown can be vertically sectioned to allow elevation of the crown. (e) Cracking crown apart into two segments using straight Warwick James. (f) Crown fragments extraorally. (g) Coronectomized roots *in situ*. The pulp chamber and live pulp can be seen. No dressing is necessary but you must ensure no enamel is left behind and trimming with a rose head bur may be necessary. (cont'd)

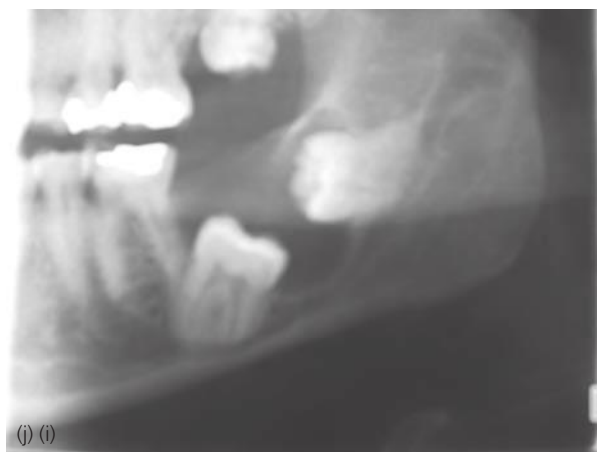
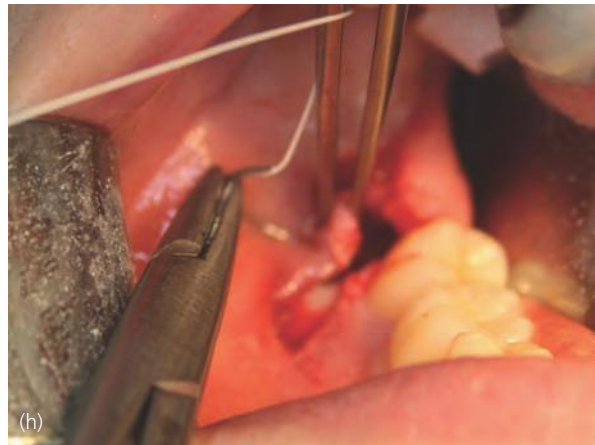


Fig. 14.32 (cont'd) (h) Closure of "socket" with single 4/0 Vicryl suture. (i) (i) Preoperative radiograph of high-risk third molar. (ii) Postoperative radiograph of coronectomized third molar. (j) (i) Preoperative radiograph of high-risk third molar and second molar with dentigerous cyst. (ii) Postoperative (6 weeks) radiograph of coronectomized third molar and second molar with enucleation with good clinical healing.

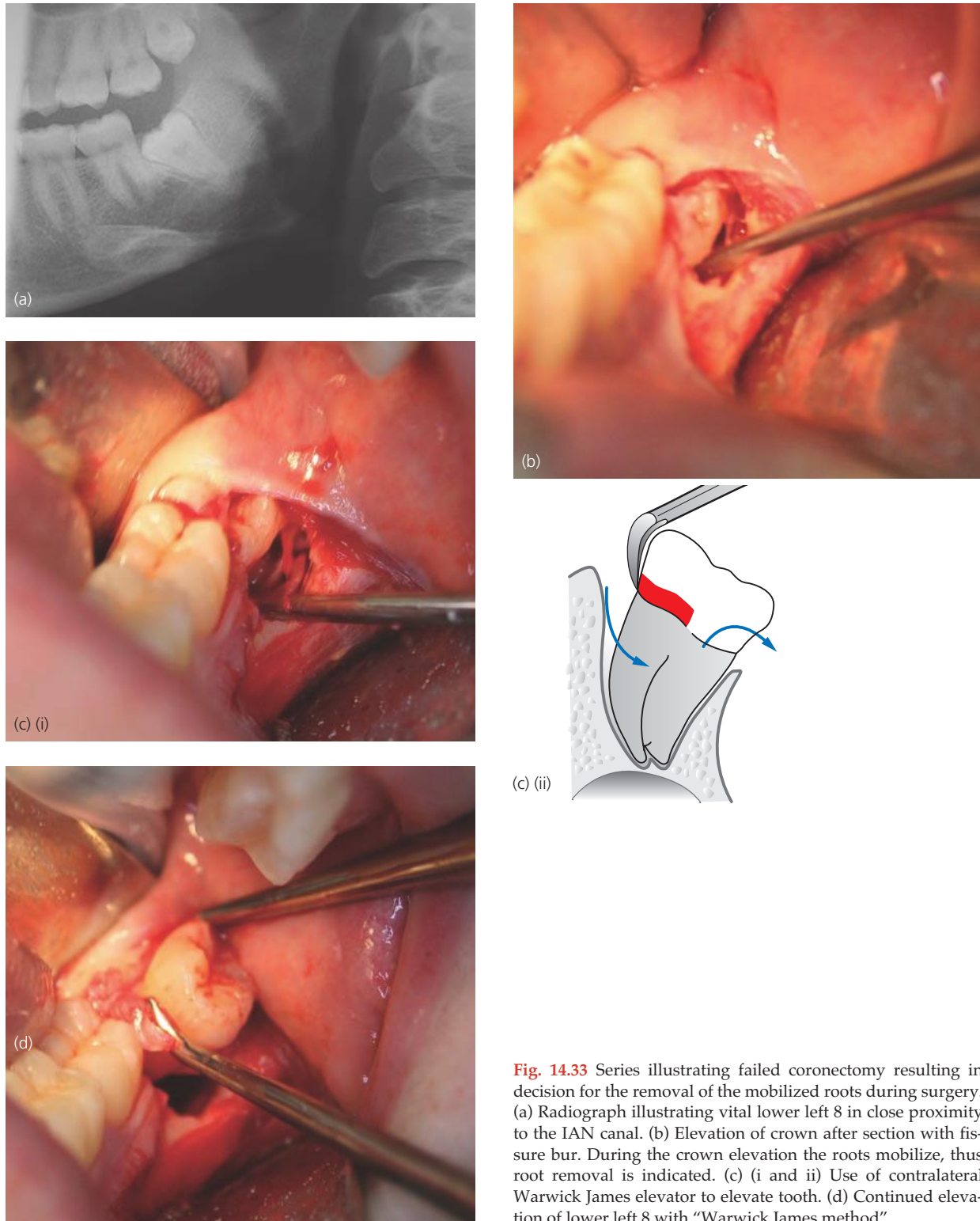


Fig. 14.33 Series illustrating failed coronectomy resulting in decision for the removal of the mobilized roots during surgery. (a) Radiograph illustrating vital lower left 8 in close proximity to the IAN canal. (b) Elevation of crown after section with fissure bur. During the crown elevation the roots mobilize, thus root removal is indicated. (c) (i and ii) Use of contralateral Warwick James elevator to elevate tooth. (d) Continued elevation of lower left 8 with "Warwick James method".

Autotransplant

Transplantation of immature third molars is a safe, useful procedure when appropriate conditions of the recipient site are present. Where the alveolus is atrophic, a splitting osteotomy should be performed only in exceptional cases and preference should be given to alternative methods such as primary bone augmentation or bone-regenerative procedures.⁹⁶

Germectomy or lateral trepanation

For the purposes of this chapter germectomy is defined as the removal of a tooth that has one third or less of root formation and also has a radiographically discernible periodontal ligament. It does appear that early third molar removal (6–17 years) may be associated with a lower incidence of morbidity and also less economic hardship from time off work for the patient. However, this is not recommended practice.^{97,98}

Restoration of adjacent tooth

Due to the routine practice of prophylactic surgery deep distocervical carious lesions of the adjacent 7s are rarely seen in the US and have become a UK phenomenon. This condition is increasingly seen in the UK where prophylactic surgery is avoided and as a result patients may present with distocervical caries of the second molar. These lesions are relatively inaccessible for the general practitioner and best access may be available during third molar surgery. There is limited evidence base for restoration of deep distocervical caries of the second molar on removal of the adjacent third molar. Excavation of caries with placement of glass ionomer cement is possible and may become routine practice in maximizing the prognosis of the second molar tooth in these cases.

Obliteration of third molar tooth bud

A recent report illustrates that in dogs the use of an intraoral 100Watt diode laser can prevent the development of third molar teeth. In the future this may have a role in selectively stopping third molar surgery.⁹⁹

Periodontal therapy

The periodontium distal to the mandibular second molar may be affected by removal of an impacted third molar.^{100,101} Some authors suggest specific strategies to minimize periodontal pocketing subsequent to third molar extraction including: scaling, root planing, and plaque control,¹⁰⁰ guided tissue regeneration,¹⁰² and platelet-rich plasma.¹⁰³

Other techniques

Fig. 14.34 shows pictures of retained root retrieval using a Mitchell trimmer to engage the (previously drilled) hole in the side of the tooth segment and elevate the root segment. Fig. 14.35 demonstrates enucleation of a cyst in association with a third molar.

Outcomes of third molar surgery

Outcomes in response to surgical or non-surgical management of third molar teeth may be successful or unsuccessful. Outcomes must be defined and quantified to enable audit to establish best practice. The success or otherwise of the procedure ideally should be viewed from the perspective of the patient.

A review appointment may be required.¹⁰⁴

- when non-resorbable sutures have been placed;
- when complications arise;
- at the patient's or surgeon's request.

Successful outcome

This is achieved when the presenting symptoms and signs of disease associated with a third molar tooth have been eliminated and the tissues have fully healed with no residual functional deficit. During normal healing it is usual for the patient to experience some discomfort, swelling and trismus over the first 3 postoperative days. Symptoms should gradually resolve over the next 2 weeks.

- Pain visual analog scale (VAS). The inflammation induced by surgical trauma results in pain of which the patient must be forewarned. This will be worst in the first 24 hours postoperatively and should be resolved within 3–4 days. If pain persists there may be dry socket or infective complications.
- Hemorrhage must be controlled at the time of surgery. Soft tissue bleeding may require hemostatic agents, bipolar diathermy, and/or sutures. Occasionally a small amount of bone wax is necessary to control bleeding from bone, but this must be used with caution. Hematoma formation outside the socket can occur and may require drainage.
- Ecchymosis. Patients should be informed that bruising is common and self-limiting and will usually resolve within 2 weeks of surgery.
- Swelling caused by surgically induced inflammation is a common sequel and usually correlates with the degree of difficulty of surgery. The patient must be warned of this risk and advised that it should resolve within 24–36 hours.

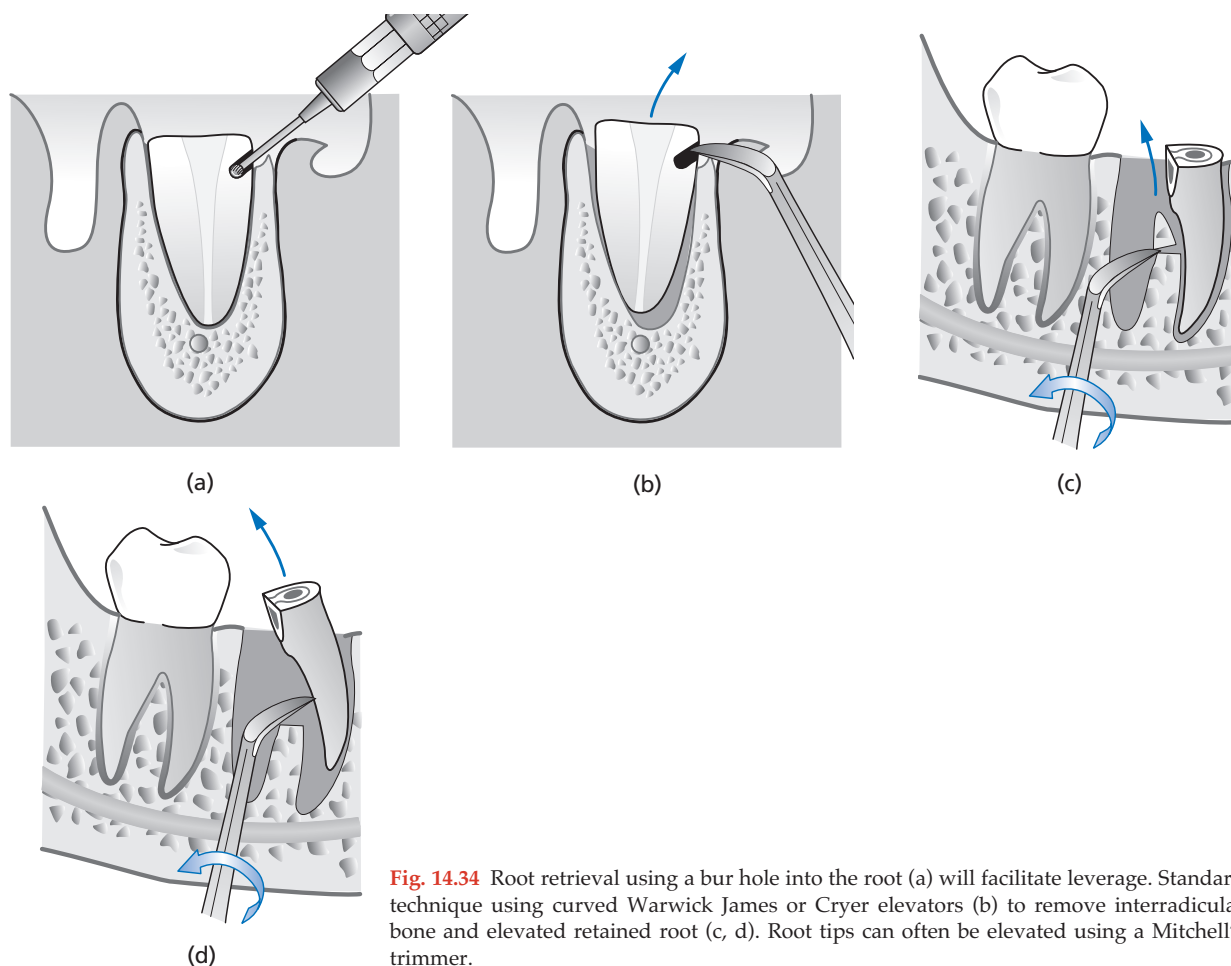


Fig. 14.34 Root retrieval using a bur hole into the root (a) will facilitate leverage. Standard technique using curved Warwick James or Cryer elevators (b) to remove interradicular bone and elevated retained root (c, d). Root tips can often be elevated using a Mitchell's trimmer.



Fig. 14.35 Enucleation of cyst in association with 38. (a) Radiograph of unicystic lesion associated with 38. (b) Cavity after enucleation of cyst. (c) 38 with associated dentigerous cyst lining.

- Trismus. Limited interincisal opening is related to the inflammation around the masseter insertion. This is interlinked with postoperative pain and swelling and should also resolve at 3–4 days postsurgically.⁵⁶
- Sensitivity. Subsequent to raising a flap adjacent to the second molar, exposure of root dentine may occur during healing of the third molar socket. This may cause local sensitivity of the second molar during this period.
- Defect. The third molar socket may take several months to completely infill and the resulting defect may cause the patient some concern. It is important to reassure the patient.
- Temporomandibular joint (TMJ) pain may arise due to prolonged opening of the patient's mouth during treatment. The joint may remain painful for several weeks and the patient should be reassured. Minimizing TMJ discomfort may be possible by routinely using a mouth prop for patients undergoing any prolonged dental treatment.

Complications associated with treatment

As noted earlier, removal of third molars is a common surgical procedure and – as with all surgical procedures – there is a risk of operative and postoperative complications. The rate of complications and their severity varies,¹⁴ but the management of common and more serious complications is described below. The following complications carry significant risk of morbidity and may require immediate referral. The patient should be informed, and a record entered in the patient's notes.

- Dry socket. Alveolar osteitis (dry socket) may occur in up to 20% of patients, particularly in those who smoke.¹⁰⁵ Other risk factors include steroid use, contraceptive pill, bone removal with a bur, age, and the "specific bacteria theory" – *Treponema denticola*.¹⁰⁶ The patient must be forewarned of this most common complication after third molar surgery and instructed to return to the surgery if persistent pain arises from the socket at 3–10 days postsurgically. Irrigation with saline (or chlorhexidine 0.2%) and/or placement of an obtundent, such as proprietary iodoform-based medication, usually immediately relieve the pain and the patient seldom returns for repeat treatment. Rarely, osteomyelitis may occur and this must be considered in patients complaining of persistent discomfort or pain. Long-term antibiotic therapy and/or further surgical exploration in a hospital environment are recommended for these patients.
- Retention of root fragment. When a retained root fragment gives rise to symptoms it should be removed. This event may become more common due to the increase in coronectomy procedures.²⁶
- Displacement of tooth or root fragment. A lower third molar or tooth fragment may be displaced into the lingual tissues, particularly if there is a pre-existing lingual plate defect or on occasion a root may perforate the lingual plate. An upper third molar may pass into the infratemporal fossa or the antrum. Appropriate instruments should be in place prior to elevation to help minimize the occurrence of displacement. Where this occurs, every effort should be made at the time of surgery to recover the displaced tooth, but referral to a specialist may be recommended. If a root is displaced into the maxillary antrum, immediate sequential radiographs may assist in identifying whether the root fragment is mobile within the antrum or entrapped under the antral mucosa. This differentiation is important in order to prevent inhalation of the fragment.
- Any tooth or fragment may be at risk of being swallowed or inhaled. In an upright patient the likely route for inhalation is the right main bronchus. The patients particularly at risk are those sedated and having maxillary surgery. All precautions must be taken to protect the airway, particularly in those with depressed cough reflexes (sedated or general anesthesia). The surgeon must be able to see the tooth/fragment at all times. If a tooth is lost, referral to A&E for radiographic assessment of the tooth position (lungs or stomach) is required. Bronchoscopy retrieval is possible for a pulmonary displaced tooth or fragment; however this is a significant surgical complication for the patient and follow-up is essential.
- Infection. Infection of the soft tissues may result in secondary hemorrhage, cellulitis or, rarely, abscess formation.¹⁰⁷ Any active infection should be controlled and any retained roots or bone sequester should be evaluated prior to surgical exploration. Where signs of systemic involvement are present (pyrexia, regional lymphadenopathy) antibiotics should always be prescribed.
- Surgical damage to adjacent structure. Patients should be told about damage to adjacent teeth at the time of surgery or, if under sedation or general anesthetic, when they are fully conscious. The consequences of this damage should be explained to the patient and recorded in the patient's notes. If repair is required, the operator should arrange appropriate management.
- Periodontal healing after third molar surgery. The periodontium distal to the mandibular second molar may be affected by removal of an impacted third molar.^{108,109} Conversely, others have reported no deleterious effects.^{110,112} Early removal of mesioangular horizontal impacted third molars is associated with better periodontal health.¹⁰⁸ The preoperative existence of an intrabony defect, age of the patient (more improvement if patient ≤25 years), and level of plaque control may help to predict adverse outcomes.¹⁰⁸ Flap design does not

appear to negate periodontal attachment loss on the adjacent second molar.¹¹² Guided tissue regeneration (GTR) may be beneficial in instances where there is evidence of significant pre-existing attachment loss. Scaling, root planing, and plaque control have the potential to reduce postoperative loss of attachment.¹⁰⁰

- Fractured mandible/tuberosity. The risk of intra-operative or postoperative fracture following third molar removal may be age related, and one study shows a mean age at fracture to be 45 years.¹¹³ Fractures should be noted at the time of surgery and repaired if necessary. If the operator is unable to do this, he/she must arrange immediate referral to a specialist. Tuberosity fractures may occur and should be treated at the time of surgery. If the operator is unable to do this, he/she must arrange an immediate referral.
- Oral antral fistula. The incidence of oroantral perforation from upper third molar removal may also increase with age past 21 years.¹¹⁴ Oro-antral communication is probably a more frequent occurrence than is realized and thus probably often heals spontaneously. Any such defect identified at the time of surgery (demonstrated with the Valsalva maneuver – blocked nose and forced air through nasal passages results in air leakage through the defect) should be repaired, usually with a buccal advancement flap. Antibiotic therapy, nasal decongestants, and antiseptic mouthwash (antral regime) are advisable and the patient should avoid nose blowing. Review is advisable at 2 weeks in order to ensure that the repair is successful. If the oro-antral fistula is persistent then an antral regime should be initiated for 2 weeks before reparative surgery. The buccal fat pad technique has been shown to be successful.
- Retained foreign body. Any broken instrument should be removed at the time of the operation. If not retrievable, radiographs should be taken if the object is radiopaque, and the patient should be informed and details recorded in the notes. If it is considered that there is a risk of infection a 5-day course of antibiotics should be prescribed. Aspiration should be excluded.
- Nerve damage. The author uses a minimal-access buccal approach for mandibular third molars as lingual flap access surgery is associated with increased temporary lingual nerve injury.¹¹⁵ Complete neural transection of the lingual or inferior alveolar nerves requires immediate nerve repair by an experienced surgeon. Where there is partial damage, gentle debridement and the maintenance of good apposition of the ends is normally undertaken. The patient should be informed of the situation. One recent study has shown that significant improvement in nerve function can be achieved by specialist surgical investigation and repair.¹¹⁶ Late recognition of nerve damage may require further surgical exploration. Persistence of

symptoms beyond 3 months indicates that a return to normal function is unlikely and that consideration should be given to nerve repair.¹¹⁶ Damage to the IAN, leading to persistent hypoesthesia/dysesthesia in its sensory distribution, is less amenable to surgical repair. The prognosis for spontaneous nerve regeneration after 6 months is poor.¹¹⁷

The spontaneous recovery rate for nerve injuries related to third molar removal is quite variable, ranging from 50–100% for both the IAN and lingual nerve.^{118,119} Several papers mention a greater spontaneous recovery rate for the IAN, but this is not well documented.

- Inferior alveolar nerve. The incidence of IAN involvement 1–7 days after surgery is around 1–5%.^{118,119} The incidence of persistent IAN involvement (still present after 6 months) varies from a high of 0.9% to a low of zero.^{25,42,118–120} A mean figure from all studies is around 0.3%.
- Lingual nerve. The incidence of lingual nerve involvement 1 day after surgery (excluding the use of lingual flap elevation) varies from 0.4–1.5%.^{25,119,121} The incidence of persistent involvement (still present at 6 months) varies from 0.5% (with the use of a lingual flap) to a low of 0%.¹²¹ Several studies indicate zero incidence of persistent paresthesia, whether lingual retraction is used or not.^{25,119,122} However, the author believes avoidance of lingual access surgery and appropriate training will avoid lingual nerve injuries.
- Long buccal nerve. Anatomical studies carried out on the long buccal nerve show that it is at risk during the initial incision for many third molar procedures. Branches of it are probably frequently cut during the incision process, but the effects are generally not noted.¹²³ A search of the literature finds no specific reports of long buccal nerve involvement, although one paper did note long buccal nerve involvement when the anatomic position was aberrant, i.e. coming off the IAN once it was already in the canal and coming out through a separate foramen on the buccal side of the mandible.¹²³ Others reported buccal nerve involvement as part of a larger study.¹²⁴
- Mylohyoid nerve. Damage to this nerve has been reported to be as high as 1.5% following lower third molar removal but this is probably due to the use of lingual retraction.¹¹⁸ The use of preoperative or perioperative steroids did not appear to influence the incidence of nerve involvement.¹²⁵

Other potentially chronic complications include:

- Temporal mandibular joint dysfunction.¹²⁶ Avoid operating on patients with TMJ pain as the prolonged opening may precipitate acute TMJ pain.

Appropriate jaw exercises, soft diet, analgesia, and oral appliances may be helpful.

- Psychological complications. Such complications are rare. It is best practice to refer the patient to their general medical practitioner as there may be other underlying contributing factors.¹²⁷

Acknowledgments

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Chapter 15

Surgical Treatment of Impacted Teeth other than Third Molars

Mehran Hossaini

The management of impacted teeth is probably the most common problem in oral surgery worldwide. The strategies and surgical management of impacted third molars are presented in separate chapters in this book. In this chapter general surgical principles for management of other impacted teeth, such as canines and premolars, are covered. Some of them have to be surgically removed. Other impacted teeth can be guided by surgical exposure to erupt spontaneously or by using orthodontic traction. Evaluation, surgical considerations, and techniques are discussed in this chapter. The use of premolars and canines as transplants to another region is covered in a separate chapter in this book.

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Introduction

Impacted teeth occur frequently and can be managed in different ways. The various options will be covered in different chapters of this book:

- surgical exposure for guided eruption (will be covered in this chapter);
- surgical removal (general principles for extraction

of impacted canines and premolars are covered in this chapter and the very common impacted third molars and all considerations are covered in Chapters 13 and 14);

- the impacted tooth can also sometimes be used as donor tooth for autotransplantation (see Chapter 17);
- an impacted tooth can also be left in place for observation.

Management of impacted teeth can be challenging. The most important part of this process is to develop an organized approach to establish a diagnosis, formulate treatment options, identify risk, assess benefits, and recognize alternatives.

Definitions

There are various definitions for impacted teeth. The American Dental Association defines an impacted tooth as “an unerupted or partially erupted tooth that is positioned against another tooth, bone, or soft tissue so that complete eruption is unlikely”.¹ The American Association of Oral and Maxillofacial Surgeons’ definition of an impacted tooth is one which is not fully erupted into the oral cavity, usually due to a lack of space, poor positioning or the presence of associated pathology.² It is possible for any tooth to follow an aborted eruptive path and become impacted.² Some have considered the impacted tooth to be pathologic and always requiring treatment. In recent years there has been much discussion and controversy regarding the need for elective treatment of asymptomatic impacted teeth. During the course of this chapter the author will use the definition which does not necessarily consider an impacted tooth to be pathologic.

Incidence

The incidence of impacted teeth varies amongst different sectors of the population. Generally, the most commonly impacted teeth in order of frequency are maxillary third molars, mandibular third molars, maxillary canines, and mandibular premolars.³ Impacted canines are seen in 1–2% of the general population, and occur in females twice as often as in males. Caucasians are affected five times more than Asians. From the anatomic standpoint impacted canines are located palatally in 85% of cases and labially in approximately 15% of cases.⁴

Theories why teeth become impacted

There is much discussion regarding the factors which may contribute to disruption of the normal eruption pattern which in turn leads to impaction of a tooth. Two main theories have been proposed as the possible explanation for the impaction of a tooth. The first is known as guidance theory which identifies the local factors and conditions as the contribution to the impaction of a tooth.⁵ These include conditions such as embryologic displacement of the tooth buds, transposition of teeth, and the presence of supernumerary teeth or odontomas. Other factors affecting the normal path of eruption are congenitally missing teeth, such as lateral incisors; it is believed this disrupts the natural guidance needed to determine the position of the tooth in the dental arch. The second theory is known as the genetic theory, which links impacted teeth to other genetically linked dental abnormalities such as the size, shape, number, and structure of the teeth.⁵ The etiology of impacted teeth has also been linked to systemic conditions, such as endocrine disorders or febrile diseases, syndromes, such as cleidocranial dysplasia,⁶ and local disease processes, such as tumors or cleft lip and palate.⁷

Table 15.1 Sequence of eruption and shedding of primary dentition.

Maxillary teeth	Eruption	Shedding
Central incisor	8–12 months	6–7 years
Lateral incisor	9–13 months	7–8 years
Canine	16–22 months	10–12 years
First molar	13–19 months	9–11 years
Second molar	25–33 months	10–12 years
Mandibular teeth	Eruption	Shedding
Central incisor	6–10 months	6–7 years
Lateral incisor	10–16 months	7–8 years
Canine	17–23 months	9–12 years
First molar	14–18 months	9–11 years
Second molar	23–31 months	10–12 years

Regardless of the etiology, a deviation from the normal sequence of eruption may indicate the existence of disruption of normal tooth eruption. It is prudent for the practitioner to consider and compare the patient's chronological age to the stage of dental growth. The normal sequences of eruption of primary and permanent dentition are demonstrated in Tables 15.1 and 15.2.

Evaluation

Although it is unnecessary to emphasize the importance of patient evaluation, it is prudent to review the different aspects of the evaluation process as it pertains to impacted teeth. The areas that must be covered in the patient evaluation include chief complaint, history of the present illnesses, medical history, clinical examination, and evaluation using appropriate imaging.

One aspect of patient evaluation which remains underemphasized is the patient's chief complaint, expectations, and the history of their present condition. Unfortunately, often patients' chief concerns or complaints are somewhat superficial or even vague. Their dislike of their smile or simple curiosity about their missing tooth may be the only information that is verbalized. A thorough clinician must take the time to explore the simple verbalization of the patients' observations and formulate their primary concerns and expectations; this fact, although basic, is quite difficult to master. Some clinicians are at times unable or unwilling to obtain this information and, worse, may superimpose their own views upon the patients' concerns. It should be recognized that patients' concerns and expectations have great impact upon

Table 15.2 Sequence of eruption of permanent dentition.

Maxillary teeth	Eruption
Central incisor	7–8 years
Lateral incisor	8–9 years
Canine	11–12 years
First premolar	10–11 years
Second premolar	10–12 years
First molar	6–7 years
Second molar	12–13 years
Third molar	17–21 years
Mandibular teeth	Eruption
Central incisor	6–7 years
Lateral incisor	7–8 years
Canine	9–10 years
First premolar	10–12 years
Second premolar	11–12 years
First molar	6–7 years
Second molar	11–13 years
Third molar	17–21 years

acceptance of, participation in, and satisfaction from their treatment plan, and ultimately success of the treatment.

The importance of the history of the present condition cannot be overemphasized. History of symptoms, trauma, premature or delayed tooth loss, a biopsy, orthodontic treatment, or siblings having similar conditions are examples of factors that will significantly affect the decision-making process. Previous biopsies or symptoms of pain or altered sensation such as anesthesia or paresthesia, are quite important when evaluating pathologic conditions associated with impacted teeth, and may guide the practitioner to perhaps consider another biopsy or surgical evaluation prior to an orthodontic consultation.⁷

A history of trauma may provide interesting clues to the nature of the condition and guide the practitioner to identify factors that can affect the prognosis of different treatment options. Tooth loss and the sequence of eruption can be valuable in the development of timing for the proposed treatment options and may guide the practitioner in the urgency of their intervention.

Medical history is a term often used broadly; it must include past medical history, review of the systems, medications, allergies, past surgical history, and social habits. Initially, this information assists the practitioner to perform a risk assessment for elective procedures, such as routine dental care and oral surgical procedures. However careful examination of this information is helpful in assessing the factors which may have contributed to the specific anomalies such as delayed eruption or malformed teeth. Histories of cleft lip and palate, syndromes, or significant systemic disease processes are important clues in formulation of treatment protocols. Dental practitioners often use various methods to collect medical history; dentists should pay particular attention to the appropriate and accurate collection of this information for evaluating patients with impacted teeth.

Clinical examination should follow a logical format. It is not expected that the dentist will completely alter their usual dental examination; however, a logical emphasis on certain findings is critical. The oral cavity is examined with an emphasis on the condition of the teeth, hard, and soft tissue. Presence of dental caries, gingivitis, or clinical evidence of periodontal disease can greatly impact the prognosis of the proposed therapy. Particular attention should be paid to the condition of the oral mucosa which may provide clues to the possible pathologic conditions associated with the impacted teeth. Presence of a mass, its consistency, and the color of the overlying mucosa are of particular concern. A bony hard mass with overlying mucosa consistent with the surrounding tissue can suggest an expansile bony lesion in this area. However, this should be differentiated from the normal bony bulge or prominence directly over an impacted tooth. This bony prominence itself is quite valuable in planning for surgical flap design. On the

other hand, a rubbery or fluctuant mass may be suggestive of an eruption cyst; the same consistency with pain and erythema may be a sign of an inflammatory process such as pericoronitis. Identification of pathologic findings such as cysts or tumors is critical, since the differential diagnosis of these findings can dictate overall patient management. Large lesions can often irreversibly affect the eruption of a tooth or its long-term prognosis. The treatment may range from a simple biopsy during the surgical exposure to complete resection of the affected area, including the impacted tooth. In all these cases an inclusive differential diagnosis must be formulated and appropriate referral should be made if there is any uncertainty regarding the appropriate management protocols.

Counting teeth, although an effortless part of the oral examination, can be easily overlooked. This simple step often helps to identify occlusal discrepancies, dental midline shift, and the extent of the dental anomaly.

The sequence of eruption, the normal standard deviation, and the patient's developmental age must be observed to differentiate between an expected delay in eruption and pathologic conditions. For example, an overall timely sequence of eruption and no evidence of primary tooth exfoliation in a specific area may indicate the likely need for therapeutic intervention. Similarly, teeth that have not erupted by the appropriate developmental age, within the accepted standard of deviation, may be considered for evaluation and treatment. In the absence of any other findings, a patient with a history of delayed eruption of primary and permanent dentition may be considered a good candidate for observation.

Dental crowding can affect the proper eruption of an impacted tooth into the dental arch and should be a part of patient evaluation, since it can affect the overall orthodontic management of the patient. For example, in a patient with limited arch space and an impacted maxillary canine, the correct sequence of management must be formulated. In these instances, a team approach is needed to manage the arch space deficiency with orthodontic treatment, and then the impacted canine can be surgically exposed to facilitate its guided eruption.

The presence of a retained primary tooth is always a puzzle. It is often unclear whether the primary tooth is retained due to the lack of contribution to its exfoliation from an unerupted permanent tooth, drifting or tilting of the adjacent permanent teeth preventing its exfoliation, pathologic conditions such as ankylosis, or a combination of these factors. Retention of the primary tooth can ultimately affect the treatment sequence and potential outcome.

Radiographic evaluation

The ultimate goal of the radiographic evaluation is to determine the location of the impacted tooth, its rela-

tion to the anatomic structure, and identification of potential pathologic findings in that area. Therefore imaging must provide diagnostic information about the impacted tooth and the surrounding structures. This presents the dental practitioner with the dilemma of what type of radiograph to obtain to provide the most diagnostic information.

Bitewing radiographs have limited value in the treatment planning of impacted teeth, but if available they can be used to assess the position of the tooth in relation to the crest of the alveolar ridge and the cemento-enamel junction of the adjacent teeth.

Periapical radiographs are helpful tools in diagnosis and treatment planning, especially if obtained correctly. A very beneficial aspect of the periapical radiograph is its role in localization of an impacted tooth; this can be achieved by repositioning of the film and the X-ray head and observing the shift in the image produced. This is known as Clark's rule, which can be remembered by the use of the mnemonic SLOB (Same Lingual Opposite Buccal) and is described as follows. The first periapical image is obtained in the standard fashion. A second image is obtained by maintaining the film in the relatively same location and then moving the X-ray head distal or mesial to its previous position. If the image of the tooth moves in the same direction as the X-ray head then the tooth is located on the lingual aspect of the alveolar ridge. If the image moves in the opposite direction of the X-ray head, then the tooth is positioned on the buccal aspect of the alveolar ridge. This is valuable information in surgical treatment planning to access an impacted tooth for removal or exposure. The biggest limitation of this technique is operator error since it is technique-sensitive. Potential problems are improper labeling of the films and inconsistency of the film placement. The personnel obtaining these radiographs should be trained and instructed properly, and preferably use film-positioning devices.^{8,9}

An *occlusal radiograph* is an appropriate imaging tool; if obtained correctly it can be used to confirm the location of the impacted teeth in relation to the adjacent dentition especially in the anterior maxilla and mandible. The disadvantage of occlusal radiographs is obtaining a high-quality and diagnostic image from the posterior region due to difficulty with film positioning and artifact from the nearby anatomic structures.

A *panoramic radiograph* is perhaps one of the most useful images available to the team involved in the patient's care; it can be used to easily identify the location of the impacted tooth and its relationship to the adjacent teeth and relevant anatomic structures. It can also be used as a tool to assess the patient's complete dentition and other pathologic findings that may be present. Some authors have developed strategies based on panoramic radiographs to identify prognostic markers for treatment of impacted canines. The axial position of impacted teeth can be readily estimated based on a panoramic radiograph; this can

impact the overall treatment plan.^{10,11} For more details regarding periapical, occlusal, and panoramic radiograph imaging see Chapter 2.

In recent years, there has been much excitement about the use of three-dimensional (3D) imaging in many areas of dentistry. These images are captured by the use of the same principles as medical grade computed tomography (CT scans) and are known as cone-beam CT; this can be a useful resource in evaluation of patients with impacted teeth. With the use of cone-beam CT the practitioner can identify the exact location and position of the impacted tooth in three dimensions. It provides accurate and useful information regarding the position of the tooth, location of the anatomic structures, and presence of pathologic findings, all of which can be valuable in treatment planning, assigning risk factors, and evaluating prognosis. Some studies have demonstrated a positive correlation between the 3D information regarding the position of an impacted tooth and its response to guided eruption.¹² However, the value and availability of this information, and its impact on patients' overall treatment should be weighed against the resources spent to obtain this information. Practitioners must identify the needs for such images and the impact that they may have on the patients' treatment. In general, cone-beam CT should not be considered a routine imaging modality, rather an adjunct to the information provided by conventional radiographs.¹³ For more details regarding CT scans and cone-beam CT imaging see Chapter 2.

Treatment planning

Indications, risks, benefits, and alternatives must be clearly identified, even though on the initial presentation the indication may appear quite clear. The factors contributing to the lack of eruption, such as lack of arch space, relation to the adjacent teeth, existence of a retained primary tooth, and existence of odontogenic and non-odontogenic pathologic findings, are the main factors that should be considered and can determine the indications for treatment. Other factors to consider include the patient's chief complaint, symptoms, age, and any findings that can identify the impacted tooth as a pathologic entity. These findings may include damage to the adjacent teeth in the form of dental caries, bone loss, recurrent infection, or pericoronitis. Also, impacted teeth can affect patients' overall dental treatment and rehabilitation. For example an impacted canine may result in inability to place an implant in the site of a missing tooth.^{3,4}

Although age in itself is not a deciding factor, it has a potential impact on the indications for treatment. For example an elderly patient with an asymptomatic impacted tooth in the absence of any pathologic findings may be a candidate for observation. On the other hand a teenager with an impacted

premolar should never be considered for an extraction unless an orthodontic evaluation has been performed and guided eruption has been ruled out.¹⁴

Once the indications have been identified, the benefits of the treatment should be communicated to the patient. Patients often assume that they must have treatment for the condition that has been identified; but often the benefits of such treatment, its impact on their overall health, and prognosis of such treatment are not well understood. It is the dentist's responsibility to clarify this information for the patient. The benefits of treating an impacted tooth include guided eruption to create occlusal harmony, improved function, and esthetics. Its removal may be beneficial to alleviate a source of pathology or damage to the adjacent teeth in the form of dental caries or periodontal defects. General practitioners, as well as specialists, must objectively recognize the benefits and prognosis of different treatment options.^{3,4}

Discussion of the risks of the proposed treatment alternatives is quite important. Patients and health care providers often view this discussion as a necessity of our current heightened medical-legal environment. In reality, however, this discussion is designed to be educational in nature and create an objective communication portal to improve the doctor-patient relationship. One must recognize the fact that, generally, litigation is not a result of occurrence of a complication, rather the fact that complications were not acknowledged, recognized, or managed appropriately. The doctor should aim to educate his or her patient to a point of recognizing the risks and complications as factors to contemplate when considering different treatment options. The doctor must not present the risks and complications of the treatment as trivial and inconsequential. Whenever possible, this information should be presented with the support of the most reliable and current evidence available. The risks of removal or exposure of an impacted tooth may include damage to the adjacent teeth, floor of the nose, maxillary sinuses, and neurovascular bundles. Guided eruption may not fulfill its objectives due to ankylosis or instability of the tooth necessitating extraction. Postoperative edema, bleeding, infection, and pain are expected risks of all surgical procedures.^{2,3}

A number of treatment options may be considered for impacted teeth other than third molars. These include observation, extraction, guided eruption, and modification of the existing dentition to allow spontaneous eruption. Observation and no treatment must always be presented as a treatment alternative. As health care professionals we consider direct involvement in our patients' care as an ethical and justified responsibility. However, the patients' right to hear and understand an objective discussion regarding observation as an option is equally justified and ethical. This right has often been enforced by medical-legal jurisdictions and the standards of care. Observation may range from no treatment to a defined

period of clinical and radiographic follow-up to monitor progress or identify pathologic conditions.^{2,3}

Extraction of the impacted tooth often appears to be a justified possibility, however in reality it is generally an option of last resort. Extraction of an impacted tooth is by definition a traumatic procedure with well recognized risks and complications. Although such risks and complications are well justified in presence of objective indications, it is prudent to avoid them if at all possible.

Guided eruption must be considered and thoroughly evaluated in all patients. This statement may further be extended to include patients of all reasonable ages, even though such treatment is often difficult to implement predictably and successfully.¹⁴ A general practitioner may consider recommending an orthodontic consultation for an older individual, if the patient is willing to consider it. Guided eruption is time consuming and often requires involvement of multiple teams in the patient's care. This should be balanced against the patient's expectations and willingness to embark on a complex treatment.

Modification of the existing dentition, realistically, is only applicable to the unerupted teeth that possess eruption potential. This is demonstrated by an unformed apex and teeth that are generally in a correct position to the arch and alveolar crest.

Orthodontic considerations and prognostic markers

In order to offer the best treatment option to the patient, there must be an attempt to identify the difficulties associated with and the prognosis of the proposed treatment. Many authors have attempted to produce treatment difficulty indices for unerupted teeth planned for guided eruption. The factors that have demonstrated greatest impact upon the success of the management of impacted maxillary canines include angulations of canine to the midline, age of the patient, vertical height, and bucco-palatal position.¹¹ These factors can also affect the duration of the treatment. An increase in the number of patient visits has been reported in patients aged 25 and older, when the cusp tip is farther from the occlusal plane, when the cusp tip is mesial to the axes of the lateral incisor, and where the tooth is less vertically inclined. In a study performed by Becker *et al.*, the treatment of impacted maxillary canines was successful in 100% of patients aged 12–16 years, compared with 69.5% in patients aged 20–47 years. In this study all the failed canines were in the patients aged 30 years and older.¹⁴ Therefore it is imperative for the clinician to promote an objective expectation for their patients in regards to the success and prognosis of their treatment. Similar prognostic markers may be stipulated for the management of all impacted teeth other than third molars.

Surgical considerations and prognostic markers

In addition to the factors affecting the successful guided eruption of impacted canines the practitioner must consider surgical implications of the treatment. Patients' tolerance for surgical procedures including management under local anesthesia versus intravenous sedation should be considered. The surgical flap and removal of bone to expose the impacted tooth may compromise the adjacent dentition and vital structures. The nature of the surrounding bone, keratinized gingiva vs non-keratinized mucosa, condition of the impacted tooth, and its relation to the roots of the adjacent teeth must be evaluated under direct vision. This information is valuable and it helps the other members of the team, e.g. orthodontist, if it is documented by the use of intraoperative photographs.

Surgical management

After thorough evaluation, treatment planning, and discussions with the patient and other team members, the surgical treatment of the impacted tooth can be planned accordingly. A critical factor in this phase of the patient's care is timing of the surgical procedures. For guided eruption of an impacted tooth, it is generally expected that the patient will undergo an initial phase of orthodontic treatment. The orthodontic treatment can range from basic bracketing and stabilization of the adjacent teeth to complete leveling and alignment of the dental arches. The primary purpose of presurgical orthodontic treatment is to create adequate arch space to facilitate the eruption of the impacted tooth. In certain cases with the proper alignment of the teeth and creation of adequate arch space, an unerupted tooth may only require surgical exposure to relieve the affect of the soft tissue on the delayed eruption. However, in many cases the surgical objective is indeed to surgically expose and bond an attachment to facilitate guided eruption of the impacted tooth.^{2,15,16}

Presence of a retained primary tooth, although not terribly critical to the management of the impacted tooth, may be an important factor to the patient, especially from the esthetic point of view. Patients are often quite satisfied with bonding of a denture tooth to their orthodontic wires. This simple and small step can significantly impact the patient's esthetic needs, acceptance, and satisfaction with their treatment.

Surgical removal

If removal of the impacted tooth is planned, a number of issues must be considered. Regardless of the radiographic position or appearance, the practitioner should plan this surgery as removal of a full bony

impacted tooth in terms of instrumentation and time allowance. A full-thickness mucoperiosteal flap should be planned. It is desirable to make an intersulcular incision extending to at least one to two teeth in each direction from the area of interest (Fig. 15.1). This will allow adequate retraction of the flap and proper visualization. A common error is to develop a small flap and extend it conservatively. This approach, although reasonable in theory, in practice often leads to tearing of the flap and/or inadequate exposure, ultimately resulting in a poor outcome. A well planned and properly retracted large flap generally heals as well, if not better, than a traumatized small flap.

Vertical release of the flap, although acceptable, must be carried out quite carefully. Improperly positioned vertical release can cause poor soft tissue outcome, in the form of recession and unesthetic gingival margins. One must attempt to position a vertical release on the line angle or in an edentulous area. A vertical release must never be placed on the midline of a tooth. Extension of the flap and location of any releases should be planned very carefully with respect to the anatomic regions. For example a vertical release should not be placed in the region served by the mental nerve, as it may transect the fairly superficial branches of this nerve resulting in sensory disturbances. Another example is the posterior mandible distal to the first molar region. An incision in this area must extend laterally from the surgical site toward the lateral oblique ridge (Fig. 15.2). This design greatly reduces the risk of damage to the lingual nerve, which in some instances can be located near the crest of the alveolar ridge distal to the second molar.

Upon dissection of the flap and exposure of the impacted tooth, adequate bony decortication must be carried out to expose the tooth. The attempt to remove these teeth whole is often not fruitful. Therefore, well planned sectioning of the impacted tooth is often needed. An important point that must be emphasized is the use of a surgical drill rather than a dental hand-



Fig. 15.1 Exposure of a buccally positioned maxillary canine using a full-thickness mucoperiosteal flap. Intersulcular incision and subperiosteal dissection has been performed to develop this flap.

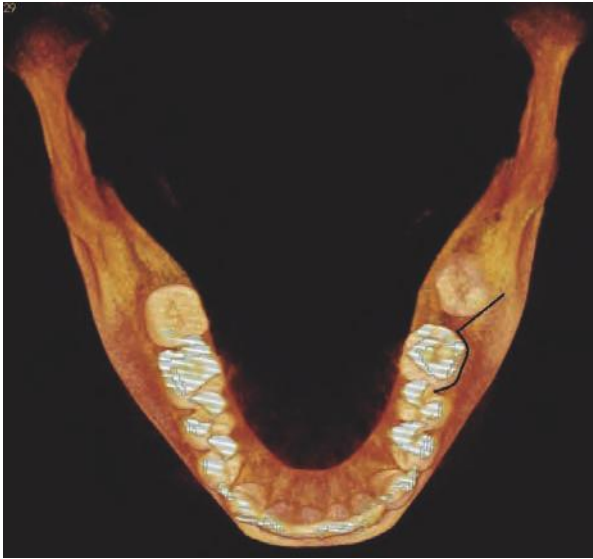


Fig. 15.2 An illustration of the incision to expose an impacted second molar in the posterior mandible. The distal portion of the incision is positioned laterally to avoid potential damage to the lingual nerve.

piece. Dental handpieces often spray an air–water mixture which can result in collection of air under the soft tissue flap, forming an air emphysema which can become potentially life threatening. Once the impacted tooth is removed whole or in sections, the surgical site can be closed primarily.

Exposure and bonding for guide eruption

Different approaches may be considered, if the impacted tooth is planned for exposure and bonding of a bracket. In this circumstance, one of the most important factors in surgical planning is buccal and palatal position of the tooth; the surgical approach is quite different for the two scenarios. If the tooth is positioned buccally, great care must be taken to maintain adequate keratinized and attached mucosa over the tooth. The presence of attached mucosa is absolutely critical to the health and prognosis of the soft tissue around the erupted tooth. An apically positioned soft tissue flap is an appropriate flap design when planning to expose and bond a buccally positioned impacted tooth. An apically positioned flap is designed as a full-thickness mucoperiosteal flap starting from the crest of the alveolar ridge with an adequate amount of attached mucosa. The flap is mobilized by creating vertical releasing incisions on each side; subperiosteal dissection is then carried out to the level of cemento–enamel junction of the erupted tooth. At this point the dissection plane is changed to the superperiosteal plane and the flap is further mobilized. Bone overlying the unerupted tooth is removed to facilitate bonding of a bracket. Upon completion of the bonding the flap is positioned apically at the cemento–enamel junction

and sutured to the periosteum.¹⁶ Some practitioners advocate placement of a periodontal dressing such as PerioPack™ over the exposed crown to limit the soft tissue overgrowth. Although it seems a reasonable idea, there has been no evidence supporting its routine use and positive impact upon the overall patient care. It is generally accepted as anecdotal and personal preference by practitioners.

If the tooth is positioned palatally, the development of a soft tissue flap is quite straightforward; however it is quite cumbersome due to the patient's position and tenacity of the palatal tissue. The best patient position is in a supine or slight Trendelenburg position which requires considerable attention for a patient who is under moderate to deep sedation. Once the flap is reflected, hemostasis to achieve a field dry enough for bonding is the biggest challenge. Although frank bleeding is not common, the slow oozing of the tissue can be a nuisance and hinder visualization of the tooth and bonding of the bracket. The oozing is best controlled by infiltration of local anesthesia containing a vasoconstrictor throughout the planned flap or packing with gauze or cotton pellets soaked in local anesthesia or an epinephrine solution. The bone overlying the impacted tooth is at times quite thin and may be removed with a hand instrument such as a periosteal elevator. Otherwise a round bur on a surgical handpiece can be quite effective. Care must be taken to not remove bone in a manner that might compromise the crown of the impacted tooth or the adjacent teeth. The overlying bone can be removed conservatively, and often it is not necessary to remove all the bone overlying the crown of the impacted tooth, but at times this does provide better visualization and access for bonding. If possible, a path for eruption should be created by conservative decortication of the palatal bone extending from the crown of the impacted tooth toward its planned location.

Once the crown of the tooth is exposed the next step is bonding of the bracket. For this purpose the site should be kept dry and free of blood. Again packing of the site with a gauze or cotton pellet soaked in epinephrine solution or infiltration of local anesthetic containing vasoconstrictor may be helpful to maintain the bonding site dry. A bracket or button attached to a chain or ligature wire is bonded to the crown of the impacted tooth (Figs 15.3, 15.4). Ligation of the impacted tooth with ligature wire or other methods is no longer an acceptable technique and may readily result in complete failure of the treatment. Auto- or light-cure bonding agents are used following the sequence recommended by the manufacturer. There are many bonding agents and orthodontic brackets commercially available specifically for bonding and bracketing of impacted teeth. Although it is not always possible to achieve, the ideal location of the bonded bracket is the most incisal portion of the crown. This position provides the most favorable application of orthodontic forces and best mechanical

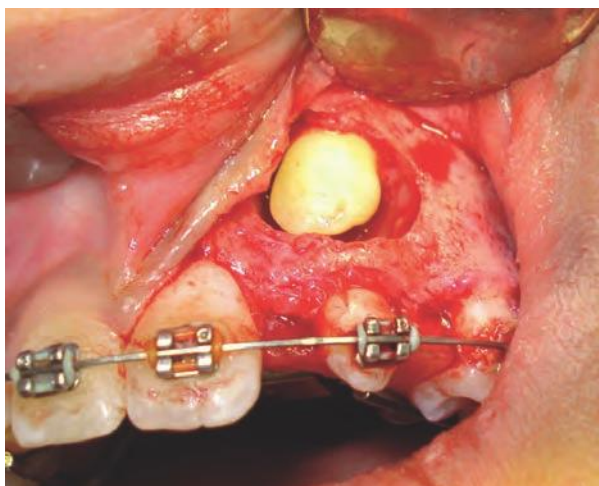


Fig. 15.3 Exposed crown of an unerupted maxillary canine. Bony defect around the crown of the impacted tooth is due to the dental follicle.

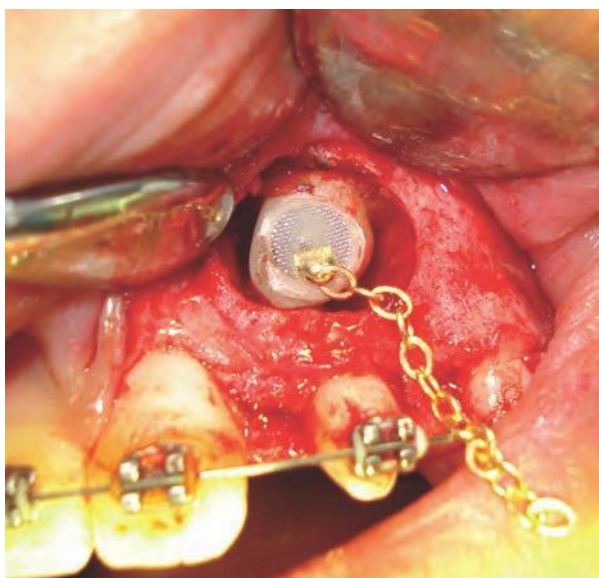


Fig. 15.4 A bracket and gold chain have been attached to the buccal surface of the impacted tooth. The excess bonding material should be cleansed from the tooth to reduce tissue irritation.

advantage needed to move the impacted tooth into the desired position.

Once the tooth is successfully bonded, soft tissue closure of the flap can be performed. The flap can be closed primarily or alternatively the overlying mucosa can be excised and the wound packed with wound dressing such as PerioPack or Iodoform gauze. Soon after recovery patients may continue orthodontic treatment.

Antibiotic use is often unnecessary postoperatively. Patients may experience moderate to severe pain for the first and second day which can be relieved with prescribed analgesics. Orthodontic manipulation is initiated after the initial healing of the soft tissue and when the patient is comfortable, which can usually be expected 7–14 days after the surgery.

Postoperative management and complications

The postoperative events and complications associated with exposure of impacted teeth are generally similar to those from other dentoalveolar surgical procedures.² Patients can experience postoperative pain, edema, bleeding, wound dehiscence, and infection. Effective pain management is quite appropriate after this type of surgical procedure. Additionally, the use of soft diet and oral wound care may improve patient comfort and reduce wound dehiscence. Limited data are available regarding the incidence of postoperative infection after exposure of impacted canines and premolar teeth. However, it may be safe to extrapolate the data from the third molar studies which report the incidence of postoperative infection to be approximately 0–12%.^{17–19}

The complication most specifically associated with exposure of impacted teeth is the failure of the initial bond or debonding of the bracket from the exposed tooth. This has been reported to range from 1–5% and has been attributed to failing to obtain a dry field during the bonding process.¹⁹ Postoperative debonding is a serious problem; at times it can be quite difficult to remedy for the surgeon, and traumatic for the patient as they may have to undergo a secondary surgical procedure. Therefore it is quite critical to not only ensure successful bonding of the bracket initially, but also to test this bond critically prior to closure of the wound. This can be simply achieved by applying a gentle yet persistent traction on the chain attached to the bonded bracket after complete curing of the bonding agent. In the event of bond failure or the need for rebonding the cured material should be cleaned from the tooth and bracket thoroughly. When there is a failure of the initial bond, the material may be scraped off and the tooth may be etched and bonded again.^{20,21} Cleaning partially cured bonding agent from a debonded bracket is awkward and time consuming, although it can be achieved by the use of a sand blaster. The use of a new bracket may be more appropriate and less time consuming. Reattaching a bracket after a postoperative debonding is somewhat more cumbersome since the tooth may have to be re-exposed. This can be performed successfully by the use of some of the more specialized etching and bonding agents.^{20,21} However, simply removing the cured bonding agent from the surface of the tooth using a diamond bur or gentle strokes of a carbide bur, and rebonding a new bracket to the tooth in a dry field is equally rewarding.

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Chapter 16

Nerve Involvement in Oral and Maxillofacial Surgery

M. Anthony Pogrel

This chapter describes the prevention, diagnosis, evaluation, and management of injuries to the inferior alveolar nerve, lingual nerve, long buccal nerve, mylohyoid nerve, and facial nerve as a result of oral and maxillofacial procedures. Nerve damage from inferior alveolar nerve blocks, dental implants, root canal therapy, and dental alveolar surgical procedures are described, as well as the semi-objective evaluation techniques that can be used in connection with these injuries. Work on objective evaluation techniques is also described. The management of these injuries, including microneurosurgical procedures, is discussed.

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Trigeminal nerve

Injury to the terminal sensory branches of the trigeminal nerve (the lingual nerve, the inferior alveolar nerve, and the long buccal nerve) is relatively frequent and can occur as a result of many different forms of dental treatment.¹

Inferior alveolar nerve block

It is known that both temporary, and occasionally permanent, nerve damage can occur as the result of an inferior alveolar nerve block.² The nerve predominantly affected appears to be the lingual nerve, which is affected approximately twice as often as the inferior alveolar nerve. The incidence is unknown with esti-

mates of permanent nerve damage resulting from inferior alveolar blocks varying from 1 in 20 000 inferior alveolar nerve blocks to 1 in 850 000 inferior alveolar nerve blocks. Transient damage from inferior alveolar nerve block (those recovering even if they take up to 9 months to do it) probably occurs five or six times as frequently. The reason for the predominance of lingual nerve injuries is unknown; it could possibly be related to the relative fascicular pattern of the nerve in the area of the lingula, since in this area the lingual nerve may be unifascicular in up to one third of cases, which may make it more liable to be damaged.³ The exact cause of injury with an inferior alveolar nerve block is unknown, but suggestions have included direct trauma from the needle in some way, hematoma to the nerve, or a neurotoxic effect from the local anesthetic itself. The fact that these patients appear to have a high proportion of dysesthesia (over 30% of such patients suffer from dysesthesia compared with only 8–10% of patients having nerve involvement as a result of third molar extraction) and a somewhat different pattern of recovery suggests that a mechanism other than simple trauma may take place. All local anesthetics appear to have the ability to cause this problem, though there may be some variation between the local anesthetics.⁴

Treatment is symptomatic; most cases recover within an 8–10 week-period, and a smaller number recover over a 9-month period. About 10% of cases prove to be permanent, with occasional disabling dysesthesia.

Inferior alveolar nerve damage from root canal treatment

Root canal therapy carried out on lower molar teeth has the ability to damage the inferior alveolar nerve either from direct trauma from overinstrumentation, from a hydrostatic pressure phenomenon, or neurotoxicity from extruded root canal sealant (Fig. 16.1).⁵ All root canal sealants appear to be neurotoxic, the only differences appear to be in the time they need to be in contact with the nerve to cause damage. Those

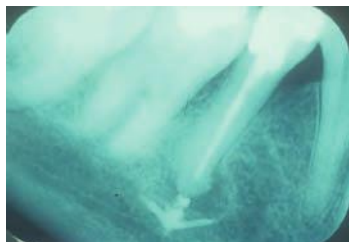


Fig. 16.1 Extruded root canal sealant in the inferior alveolar canal.

containing paraformaldehyde, or an analog, may cause damage almost immediately, whilst less toxic sealants, such as eugenol or calcium hydroxide (because of its high pH), may not cause damage until they have been in contact with the nerve for 24–72 hours. The incidence of this complication is unknown, but a paper describing 61 cases suggests that early surgery (preferably carried out the same day as the injury to remove any sealant from around the nerve) may be successful.⁵ Otherwise, these injuries tend to be permanent, with a relatively high incidence of dysesthesia, and respond relatively poorly to surgery carried out at a later date.

Nerve damage from dental implants

The insertion of osseointegrated implants into the mandible has the potential to damage the inferior alveolar nerve and mental nerves due to overextension (Fig. 16.2).^{1,6,7} In this case, the injury may be related to drilling prior to implant insertion, particularly since most drills are 0.5–1.5 mm longer than the implant that will be fitted (Fig. 16.3). Accurate measurement of the amount of alveolar bone available to place implants is necessary and plain radiographs are



Fig. 16.2 Implants impinging on the inferior alveolar nerve.

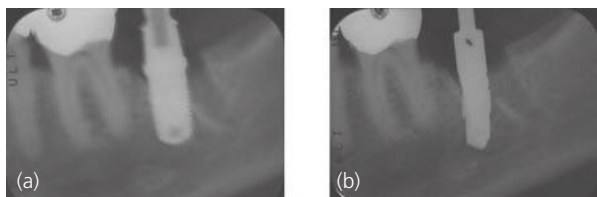


Fig. 16.3 (a) Radiograph of implant appearing not to impinge on the inferior alveolar nerve. (b) Radiograph showing that the drill used to prepare the implant site was longer and did impinge on the nerve.

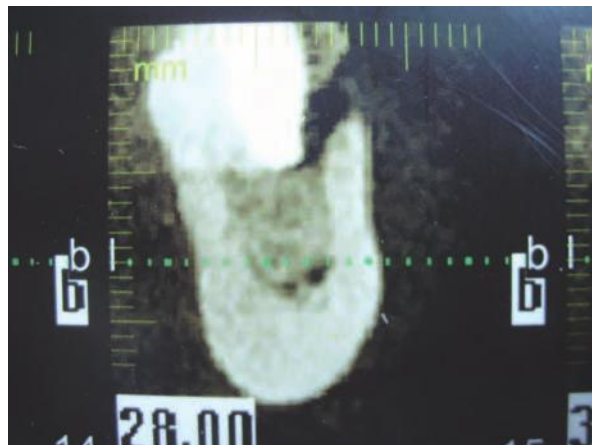


Fig. 16.4 A coronal cone-beam CT scan showing where the inferior alveolar nerve is placed and allowing planning for implant placement to be performed in three dimensions. This implant is too close to the nerve.

often supplemented by cone-beam computed tomography (CT) scans to gain an accurate impression of the amount of bone available (Fig. 16.4). If there is anesthesia or paresthesia of the inferior alveolar nerve following implant insertion once the local anesthetic has dissipated, consideration should be given to obtaining a cone-beam CT scan to accurately relate the position of the implant to the nerve, and early consideration should be given to shortening or removing the implants. This can be successful in the early stages when the involvement is due to pressure alone from the implants or hydrostatic pressure. However, many cases do not recover, presumably since the damage is more profound or was caused by the twist drill. The authors have explored a number of these cases and have found that the injuries were

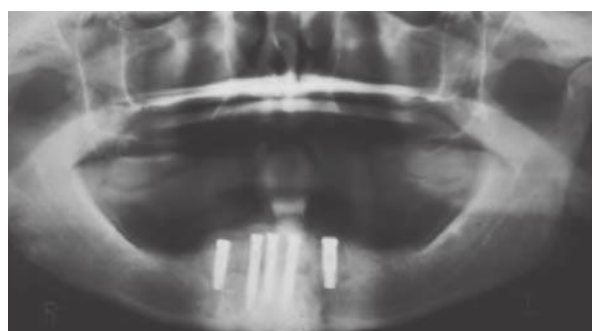


Fig. 16.5 An implant placed anterior to the mental foramen which has damaged the mental nerve as it loops back to the foramen.



Fig. 16.6 Twist drill for implant site preparation with a stop on it prevent overdrilling in the posterior mandible.

often so profound that they were either impossible to repair or they could only be repaired with a graft, which does not give good results. Therefore, at present, it is probably only advisable to explore cases where there appears to be a realistic chance of improving the condition. Cases can occur from implants inserted anterior to the mental foramen if consideration is not given to forward looping of the inferior alveolar nerve before it exits the mental foramen. Although implants are normally placed sufficiently anterior to the mental foramen to avoid this loop of inferior alveolar nerve, on occasions it can be visualized from the radiographs⁸ and additional space needs to be allowed (Fig. 16.5). Most implant systems come supplied with a stop of some kind to avoid overdrilling, which may inadvertently involve the inferior alveolar nerve (Fig. 16.6). These stops should be used when available to limit drill penetration to the predetermined length only.

Periodontal surgery

Periodontal surgery in the form of the distal wedge procedure (Fig. 16.7) and extensive surgery or deep root planing on the lingual side in the lower molar region can cause damage to the lingual nerve,¹ particularly when the nerve lies in an aberrantly superior position as is known to occur in between 15 and 20% of cases.⁹ In these cases the nerve can lie at the level of the crest of the lingual alveolar bone or even slightly above it. Studies have shown that in the majority of cases the lingual nerve lies around 8mm below the crest of the alveolar ridge and some 2–3mm lingual to the lingual plate and would not be at risk from normal periodontal procedures. If periodontal surgery is to be performed in these areas, the surgeon must have knowledge of the possible aberrant positions of the lingual nerve and take this into account when performing the surgery. If the nerve is damaged with a sharp instrument such as a scalpel, early surgical nerve repair may give satisfactory results.



Fig. 16.7 Diagram to show how a distal wedge-type procedure can damage the lingual nerve if it lies in an aberrantly high position.

Nerve damage from dentoalveolar surgery

Dentoalveolar surgery carried out on the posterior mandible can cause damage to the lingual nerve, the inferior alveolar nerve, the long buccal nerve, and even the mylohyoid nerve. The commonest dentoalveolar procedure carried out in this area is third molar removal, but other surgical procedures in this area have the potential to cause damage.¹

The incidence of inferior alveolar nerve damage from the removal of third molars varies in the literature from 0.5–5%.¹⁰ The cause is directly related to the anatomical relationship between the inferior alveolar nerve and the roots of the third molar. In many cases, this can be determined from panoramic type radiography, and criteria have been developed to determine more exactly from a panoramic radiograph the relationship of the inferior alveolar nerve to the tooth.¹¹ This is shown in Fig. 16.8. When the image of the inferior alveolar canal is superimposed over the tooth with no loss of lamina dura of the canal and no narrowing or change of direction, the relationship is probably one of superimposition with a low risk of nerve involvement as shown in Fig. 16.8a. If, however, the nerve loses its lamina dura it may in fact be grooving the tooth, and the risk of inferior alveolar nerve involvement on removal of the tooth may be higher. This is shown in Fig. 16.8b. Fig. 16.8c shows loss of the lamina dura, narrowing, and possible deviation of the path of the inferior alveolar canal and denotes an intimate relationship between the tooth and the nerve with a high risk of nerve involvement that may be 50% or higher. A pair of periapical films, taken at different angulations, may reveal relationships between the roots and nerves, by applying the parallax principle (“same lingual, opposite buccal”). Cone-beam CT technology can now accurately show the relationship of the inferior alveolar nerve to the third molar in three dimensions^{12,13} and this has

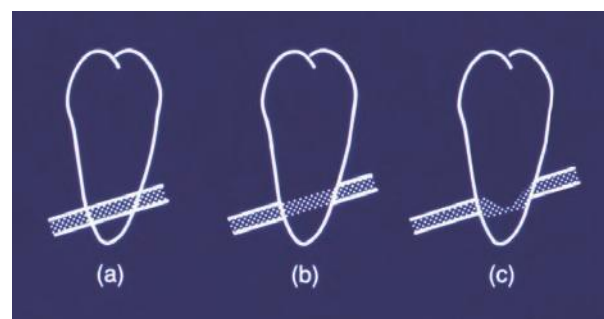


Fig. 16.8 Diagram to show the relationship of the inferior alveolar nerve to the roots of a mandibular third molar as seen on a panorex radiograph. (a) Superimposition only with no loss of cortical outline, narrowing or deviation. (b) Loss of cortical outline denotes grooving of the roots by the nerve. (c) Loss of cortical outline, narrowing, and deviation denote an intimate relationship between the nerve and roots of the third molar. (From Pogrel MA. Complications of third molar surgery. *Oral Maxillofac Surg Clin N Am* 1990; 2: 441–51. With permission. Copyright © 1990 Elsevier.)

helped to quantify the issue of the risk involved in the removal of such teeth (Fig. 16.9). Where suitable imaging shows an intimate relationship between the roots of the third molar and the inferior alveolar nerve, a decision must be made whether to proceed with the removal or not, or possibly consider intermediate strategies varying from orthodontic forced eruption of the teeth away from the inferior alveolar nerve prior to extraction¹⁴ or possible coronectomy or partial tooth removal (Fig. 16.10) whereby the portion of the root intimately related to the inferior alveolar nerve is left behind.^{15,16} For this technique to be successful the patient needs to be placed on antibiotics prior to the removal so that antibiotics are in the pulp chamber of the tooth when it is removed, the retained roots need to be at least 3mm below the level of the surrounding alveolar bone so that bone can heal over them, and primary closure of the socket (coupled with periosteal release, if necessary) must be carried out. Lingual retraction is necessary in these cases to avoid damage to the lingual nerve when the crown is sectioned. Studies show that this is a safe and predictable technique with a high success rate in avoiding injury to the inferior alveolar nerve, although some of the roots may later move and some may even require subsequent removal.^{15,16}

Lingual nerve

Lingual nerve injuries in relation to dentoalveolar surgery are less frequent than inferior alveolar nerve injuries. Frequencies have been quoted as occurring in 0.2–2% of all lower third molar removals, although they do appear to be more troubling to patients.¹⁷ In most cases the lingual nerve is protected beneath the lingual plate of bone, as it lies approximately 6–8 mm inferior to the lingual crest and some 2 mm medial; studies have shown, however, that in between 15 and 20% of cases the lingual nerve may lie at or above the level of the lingual plate and is therefore at risk during third molar removal.¹⁷ It is felt that it may be at risk:¹⁷

- during the initial incision if it is made too far lingually and the patient has an aberrant lingual nerve;
- during flap retraction if a lingual flap is raised with a misdirected or sharp instrument and the lingual nerve is in an aberrant position;
- the lingual split technique, if used to remove lingual bone prior to removal of third molars, can damage the lingual nerve either by the lingual flap retraction or by sharp edges of the bone itself;
- removal or fracture of lingual or distal bone during removal of the tooth can damage the lingual nerve;
- tooth sectioning, if the drill is placed too deeply and penetrates the lingual plate of bone;

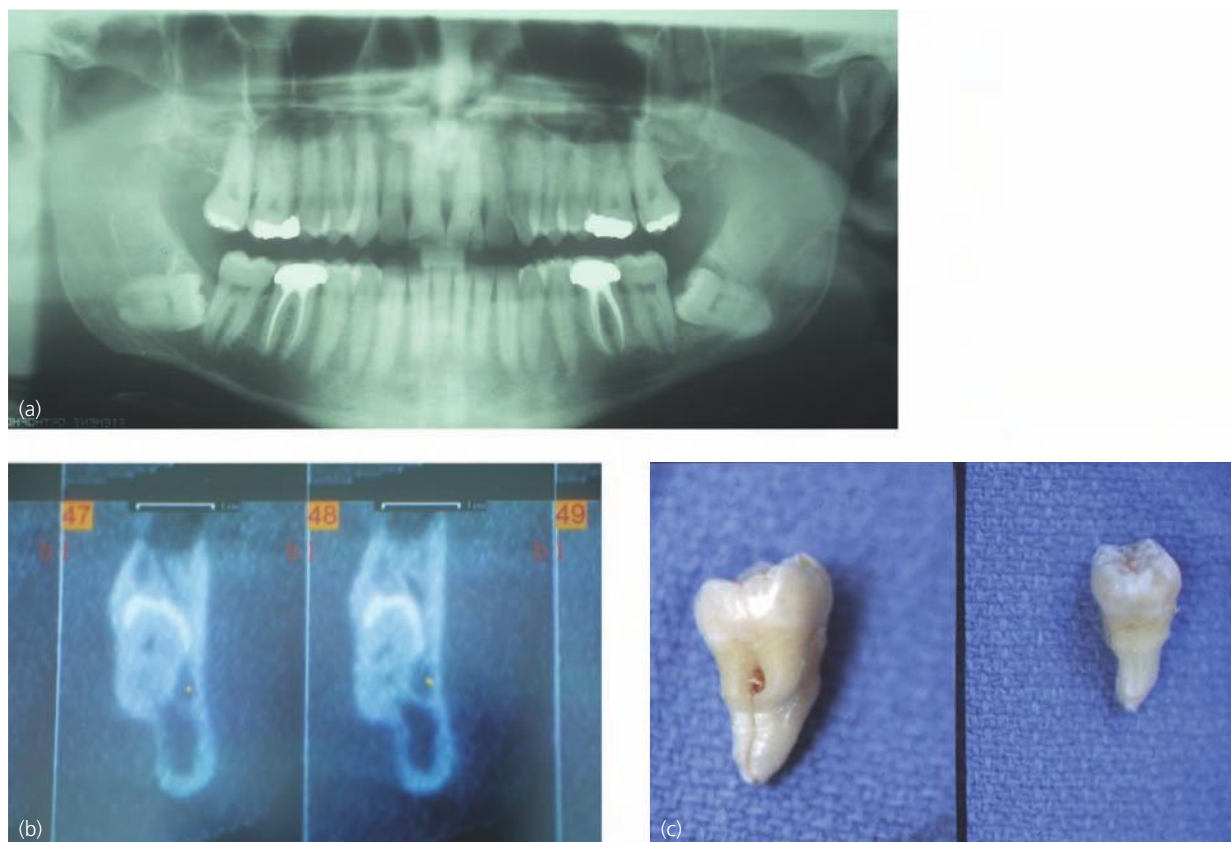


Fig. 16.9 (a) Example of a case where the shadow of the mandibular canal loses its cortical outline, narrows, and is deviated, denoting an intimate relationship between the nerve and the tooth root, which was confirmed on coronal CT (b) and on subsequent tooth removal where the tooth has been grooved by the nerve (c).

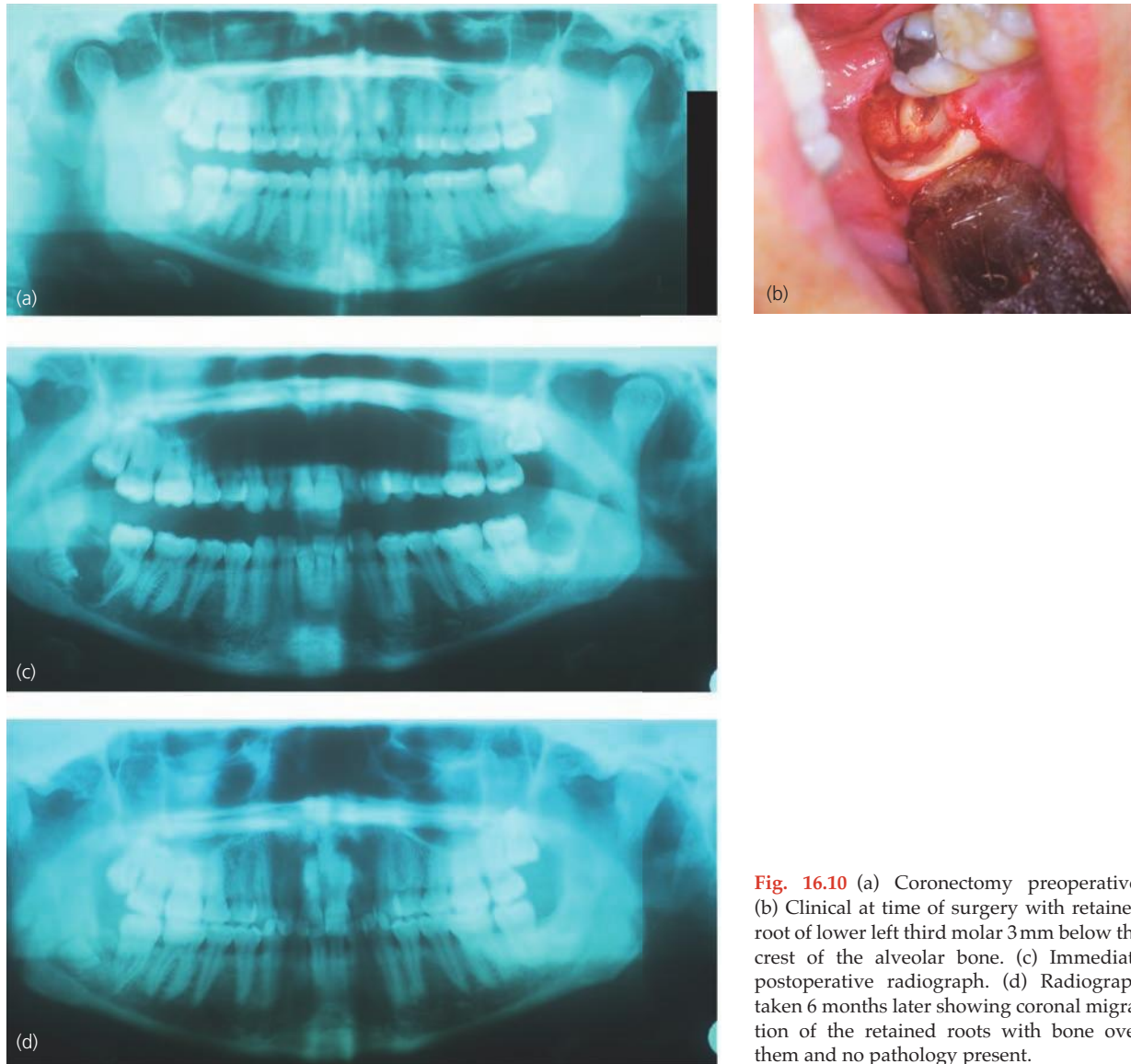


Fig. 16.10 (a) Coronectomy preoperative. (b) Clinical at time of surgery with retained root of lower left third molar 3mm below the crest of the alveolar bone. (c) Immediate postoperative radiograph. (d) Radiograph taken 6 months later showing coronal migration of the retained roots with bone over them and no pathology present.

- overaggressive removal of retained dental follicle on the lingual side may damage an aberrantly placed nerve;
- deep suturing may damage an aberrantly placed lingual nerve on the lingual side of the incision;
- in some cases the lingual plate of bone may be absent congenitally or due to infection or other pathologies, and the lingual nerve may be in direct contact with the tooth and therefore at considerable risk when the tooth is removed.

Fig. 16.11 denotes recommended incisions for the removal of a lower left third molar, which should not endanger the lingual nerve. Fig. 16.12 demonstrates the problem of retained dental follicle adjacent to an aberrantly placed lingual nerve, whilst Fig. 16.13 demonstrates that sutures placed too deeply can damage the lingual nerve; sutures should be placed very superficially. Lingual flap elevation and the placement of a subperiosteal lingual retractor is an acceptable technique in order to protect the lingual

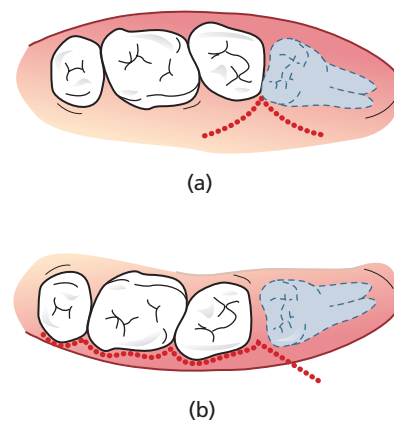


Fig. 16.11 Recommended incisions for removal of a lower third molar. (a) Triangular flap with buccal release. (b) Gingival margin envelope flap. Note that both incisions are buccally placed.

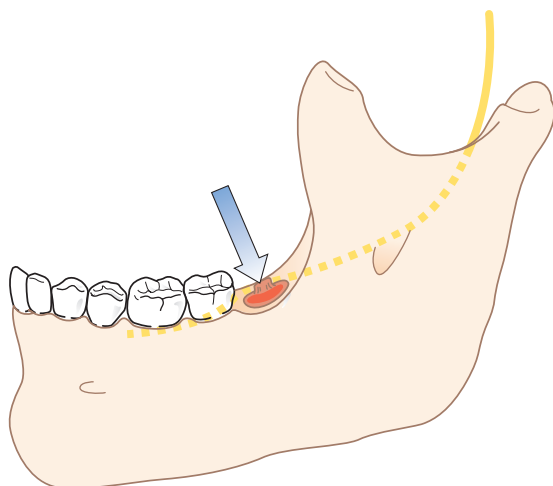


Fig. 16.12 Diagram to show how an aberrantly positioned lingual nerve can be attached to remnants of the dental follicle, which should be removed with caution.

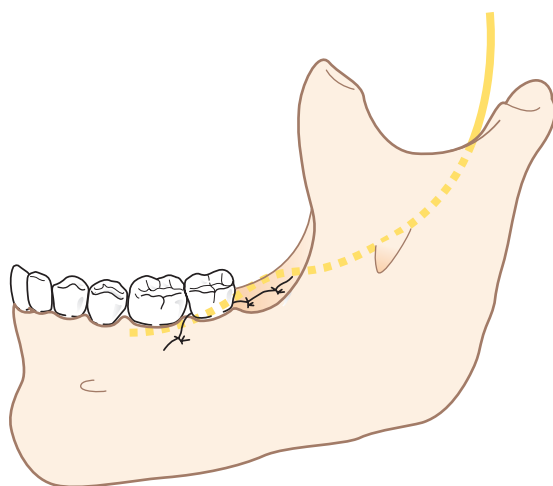


Fig. 16.13 Diagram to illustrate how deeply placed sutures following third molar removal can endanger an aberrantly placed lingual nerve. Sutures should be superficial only, or the sockets should be packed without suturing.

nerve during third molar removal if it is anticipated that bone will need to be removed in the distal or disto-lingual areas or if the crown must be totally sectioned. It is accepted that there is a small risk of transient nerve involvement following the use of lingual retraction, but this virtually always recovers spontaneously in a few weeks.¹⁷

Long buccal nerve

Considering the anatomy of the long buccal nerve (Fig. 16.14), it might be anticipated that this nerve would be involved in many cases during third molar removal.¹⁸ However, it is unusual for patients to be aware of damage to this nerve, partly because they are unaware of the altered sensation on the inner aspect of the cheek, and often because there is an overlapping nerve supply from surrounding nerves. Nevertheless, damage has been documented on a number of occasions and can occasionally be trouble-

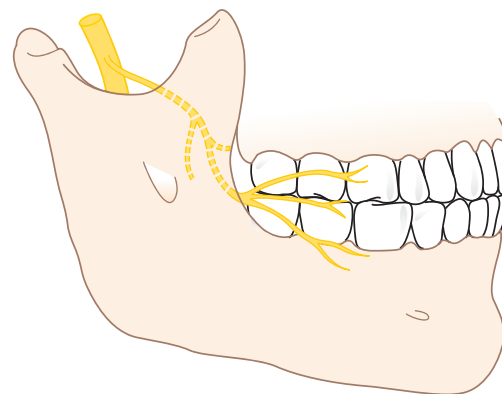


Fig. 16.14 Diagram to illustrate the usual position of the long buccal nerve which is at risk with any extended incision for third molar removal.

some to patients.¹⁹ In practice, it is virtually impossible to find the long buccal nerve surgically, and equally impossible to repair it.

Mylohyoid nerve

Involvement of the mylohyoid nerve has been reported in up to 1.2% of third molar removals but is normally associated with lingual retraction, where the retractor has been placed too deeply.²⁰ There is generally a localized area of paresthesia beneath the point of the chin on the affected side. It has been suggested that the mylohyoid nerve may provide part of the nerve supply to the tip of the tongue, possibly explaining different manifestations of nerve damage on the tip of the tongue versus other parts of the tongue.²¹ Involvement of the mylohyoid nerve is usually temporary and of little clinical significance.

Evaluation of trigeminal nerve damage

Most evaluation techniques for nerve involvement are semi-objective at best and do rely on the presence of a cooperative patient.²² The different testing techniques are used to evaluate the different types of nerve filaments that might be involved. In all cases the normal side is tested first and the abnormal side is compared to it. Semmes-Weinstein plastic filaments (often called Von Frey's hairs after the original such system which used different thicknesses of horse hair) (Fig. 16.15) or its equivalent are used to test sensation quantitatively, and since the numbers applied to the filaments represent the reciprocal logarithmic value of the weight in grams that it takes to bend the filament, this results in a straight line graph of the force required to detect the sensation. Two-point discrimination is used to qualitatively detect sensation in the larger nerve filaments, whilst temperature sense and direction sense can also be evaluated. Temperature sensation can be tested with Minnesota

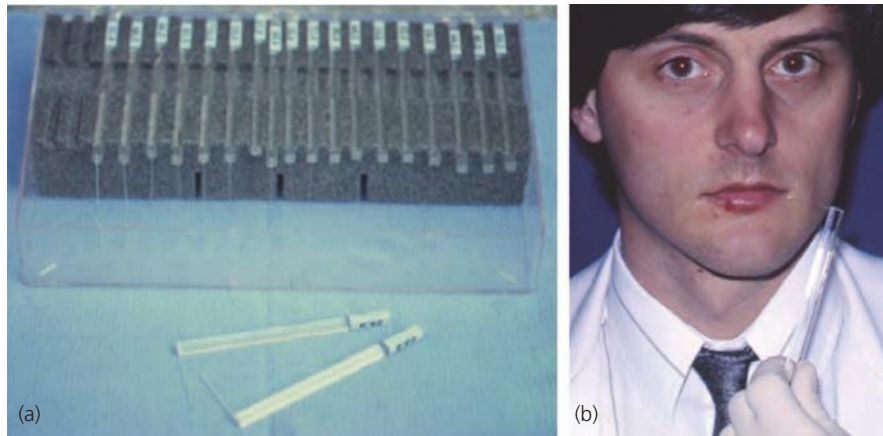


Fig. 16.15 (a) Semmes-Weinstein filaments (Von Frey's hairs) for quantitatively testing sensation. (b) Use on a patient to map out the affected area. Filaments should be pushed into the tissue and not stroked along it as this can test direction sense and give erroneous results.

Thermal Conductivity Discs or with ice and warm water, and direction sense can be tested with a cotton wisp or the Semmes-Weinstein filaments. By a combination of these means, one can map out the area involved and the degree of involvement within that area. Taste is transmitted via the chorda tympani, which is technically the pretrematic branch of the facial nerve, but it does "hitchhike" within the same overall epineurial sheath as the lingual nerve when it joins it shortly after the third division of the trigeminal nerve exits the foramen ovale and divides into the lingual and inferior alveolar branches. Taste is normally tested with fairly crude dropper bottles containing the four basic tastes (salt, sweet, bitter, and sour), which are tested on the different parts of the tongue without allowing the tongue to come in contact with other taste buds in the mouth (Fig. 16.16).

More objective sensory testing facilities have been evaluated including somatosensory evoked potentials (SEP),^{23,24} magnetic resonance imaging (MRI),²⁵ and magnetic source imaging (MSI).²⁶ SEP have been extremely difficult to reproduce and to standardize and are not used clinically. MRI has not been shown to be able consistently to identify a nerve the size of the lingual nerve. It may, however, be able to identify a moderate to large neuroma forming on a damaged nerve, since the neuroma may be two to three times the diameter of the original nerve, and may appear brighter than the surrounding nerve on MRI scanning. A magnetic resonance technique to enhance nerve visualization, which has been termed magnetic resonance neurography, may cause some enhancement, but still cannot consistently identify the lingual nerve. MSI, particularly superimposed on MRI scans, can give an absolute indication of a totally severed nerve, but it is unclear whether it can detect different degrees of nerve injury. Also, MSI is experimental and only available in a small number of centers. For taste, electrogustometry is available and can give a more objective measure of altered taste.²⁷ Mapping of the area affected and recording photographically can



Fig. 16.16 A typical taste testing kit but with dropper bottles of the primary tastes.

be of help in documenting cases of nerve injury and determining whether recovery is occurring.

Facial nerve

The facial nerve, or VII cranial nerve, is the motor nerve which innervates the muscles of facial expression. It is at risk in a variety of oral and maxillofacial surgery procedures, mostly from procedures performed from an extraoral approach. The only time it is at risk from intraoral procedures is from an inferior alveolar nerve block, the local anesthetic injection technique commonly used to anesthetize the hemimandible, when the needle is incorrectly positioned and the local anesthetic is deposited posterior to the mandible in the parotid tissue where it can permeate to affect the branches of the facial nerve (Fig. 16.17). In this case, there will be a facial nerve weakness which should be transitory and last as long as the local anesthetic effects last (Fig. 16.18). However, long-lasting, if not permanent, cases of facial nerve involvement have been described from this particular procedure, for which no active treatment is available.^{28,29}

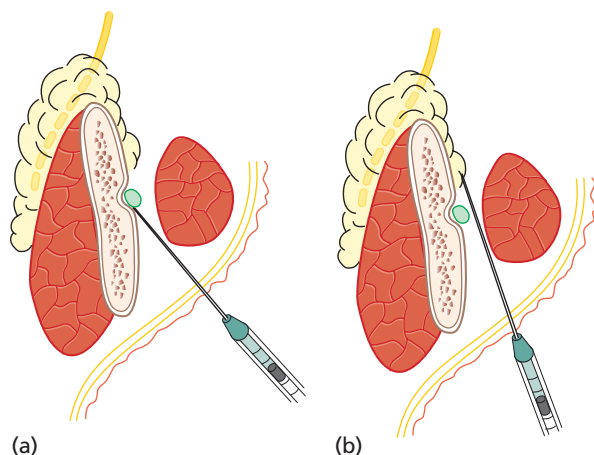


Fig. 16.17 Diagram to show how an inferior alveolar nerve block can affect the facial nerve if the local anesthetic is given too far posterior and the needle does not contact bone: (a) normal, (b) injection into parotid gland where facial nerve can be affected.



Fig. 16.18 Transient facial nerve weakness from an inferior alveolar nerve block.

On the face, the main trunk of the facial nerve emerges from the stylomastoid foramen and travels forward and laterally into the parotid gland, where it normally divides into an upper and lower trunk, which further subdivide into the five terminal branches, but there are many variations on this. The zygomaticotemporal, frontal, and buccal branches arise from the upper trunk, while the mandibular and cervical branches emerge from the lower trunk. As they exit the parotid gland anteriorly, they lie in relation to the investing fascia of the neck, deep to the

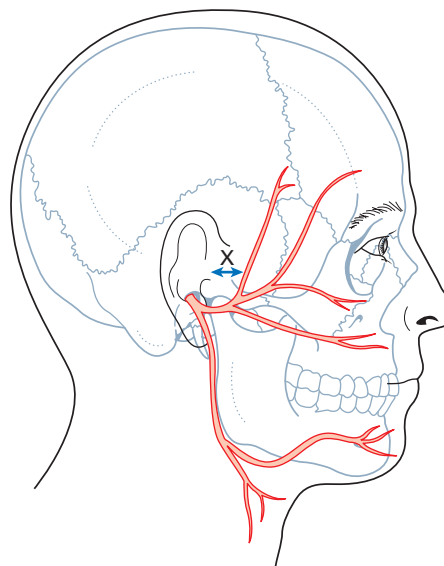


Fig. 16.19 Relationship of the branches of the facial nerve to the underlying bony structures. X = 0.8–3.5 cm.

platysma muscle. In this position they are at risk in many of the incisions and procedures carried out on the face. All of the branches are particularly at risk during parotid surgery, and this is described elsewhere in this textbook. Similarly, the upper branches (particularly the zygomaticotemporal branch) are at risk during temporomandibular joint surgery, particularly if the dissection is carried forward on the arch of the zygoma in the region of the articular eminence. The anatomy of the facial nerve is variable in this area, and has been described as being anywhere from 0.8–3.5 cm anterior (mean 2.0 cm) to the anterior cavity of the external auditory meatus (Fig. 16.19).³⁰

In the retromandibular approach to the mandible for exploration and fixation of condyle neck fractures, the dissection is normally carried out bluntly between the two main trunks of the facial nerve,³¹ and although damage in this area is possible, it is actually quite unusual.

In the submandibular approach to the submandibular salivary gland and the mandible, both the mandibular and cervical branches of the facial nerve are at risk. The mandibular branch supplies the orbicularis oris, causing a weakness of the corner of the mouth, while the cervical branch supplies the platysma. An involvement of this nerve branch can also cause weakness of the corner of the mouth, though it is usually less severe than with mandibular branch involvement and is often transitory. The studies by Ziarah and Atkinson have shown the positions of these two nerves and where incisions need to be placed in order to avoid them.^{32,33} In 53% of cases, the mandibular branch of the facial nerve does travel below the lower border of the mandible, but never more than 1.2 cm below the lower border, and it has always crossed above the lower border of the mandible by the time the second bicuspid area is reached. Therefore, an incision placed 2 cm, or one finger's breadth, below the angle of the mandible should avoid the mandibu-

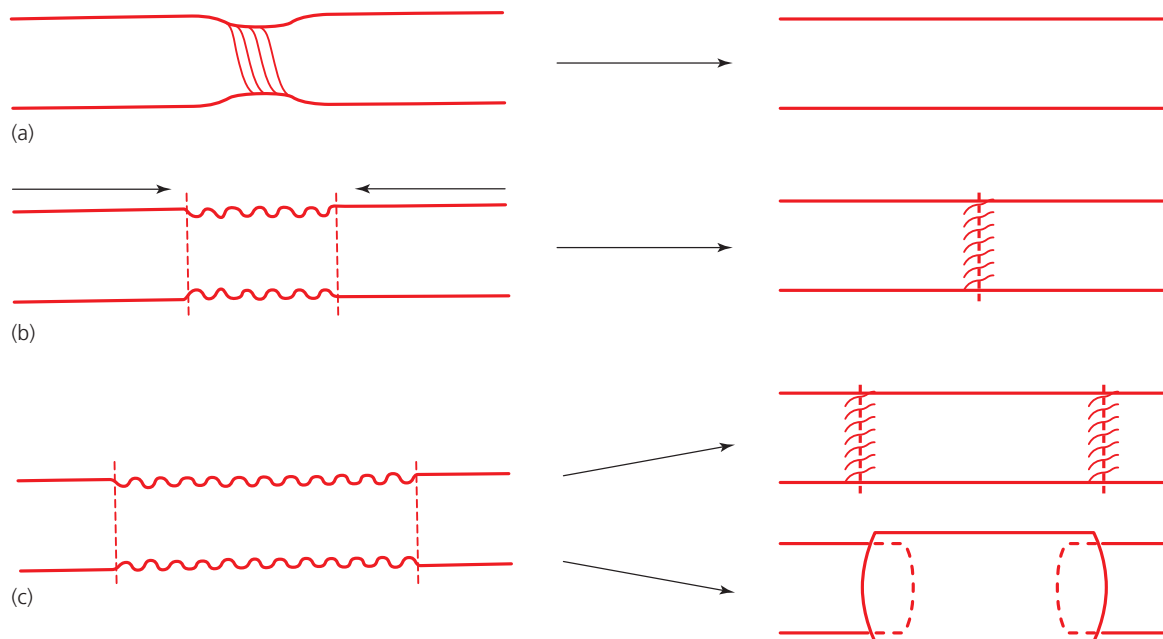


Fig. 16.20 Diagram to show the different types of microsurgical procedures possible. (a) Decompression for a localized compression from fibrosis, scarring or a bone fragment. (b) Direct anastomosis for a localized transection, or other injury. (c) A graft to repair a more extensive injury. (From Pogrel MA, Kaban LB. Injuries to the inferior alveolar and lingual nerves. *Cal Dent J* 1993; 21: 50–4. With permission. Copyright © 1997 California Dental Association.)

lar branch of the facial nerve at all times. The cervical branch, however, runs progressively inferiorly as it progresses anteriorly, and in order to avoid contact with this nerve the incisions need to be placed 4 cm below the lower border of the mandible in the mandibular notch area, although 3 cm below the mandible at the angle is sufficient to avoid involvement of this branch. Both of these approaches are feasible for sub-mandibular access surgery.

If these peripheral branches of the facial nerve are damaged during surgery, it is normally not possible to identify or repair them and spontaneous recovery is often incomplete. Surgical repair and reconstruction of the main trunk and the upper and lower branches of the facial nerve may be possible on some occasions, but the results of surgery are generally less than satisfactory.

Microneurosurgery

Microneurosurgery has been attempted to repair injuries to the lingual and inferior alveolar nerve, but

it is normally felt to be impractical for the long buccal, mylohyoid, and chorda tympani nerves. Most authorities feel that it is not possible to restore taste surgically, since there is normally end-organ degeneration after some 3 weeks and the taste buds will not recover. Nevertheless, there are reports of microneurosurgery, carried out up to 6 months after injury, which has restored some taste.^{34,35} Conceptually, the type of nerve injuries that might be treated surgically would include compression injuries where decompression might result in a cure, and partial or complete severing of the nerve (Fig. 16.20). In these latter cases excision of the affected area of nerve is normally required, freshening up of the ends, and direct epineurial repair when this is possible (Fig. 16.21). Undue stretching or devascularization of the nerve should be avoided, and in cases where direct apposition is not possible, a nerve graft or conduit must be used. Donor sites for nerve grafting can come from the great auricular nerve in the neck or the sural nerve behind the lateral malleolus of the ankle (Fig. 16.22); the medial antebrachial nerve of the forearm has also been described as a donor. Conduits have also been

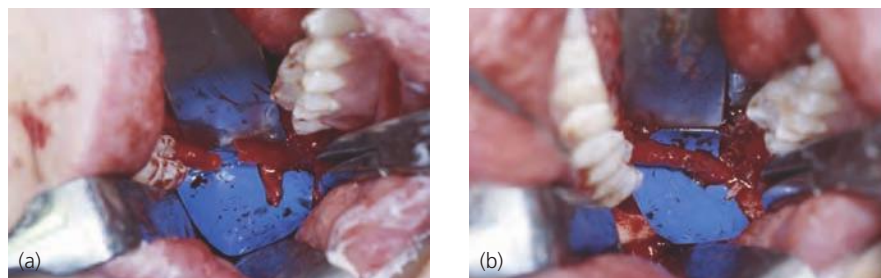


Fig. 16.21 Direct epineurial repair for a recent (within 10 days) transection of the lingual nerve. (a) After exposure showing the two nerve ends. (b) After direct anastomosis with five 8/0 nylon sutures.



Fig. 16.22 Sural nerve graft taken from behind lateral malleolus of the right ankle.

described rather than using a nerve graft, and vein grafts are an autogenous conduit which has met with some success (Fig. 16.23),³⁶ whilst alloplastic conduits such as Dacron have not been shown to be successful.³⁷

Most authorities do feel that if surgery is to be successful, it should probably be carried out within a few months of the injury. The longer one waits the more chance there is of degeneration of the distal segment of the nerve and neuroma formation on the proximal segment, which would need excision and would complicate any repair procedure. Many authorities would feel that surgery was not indicated if the patient has protective reflexes, which occur at about 30% of normal feeling.³⁸ If protective reflexes are present, then it is felt that the patient can protect themselves from further damage. Reports of results of microneurosurgery on the terminal branches of the third division of the trigeminal nerve are few, but do suggest that if surgery is carried out early on patients who do not have protective reflexes between 4 and 7 months after the injury occurring, then there can be some improvement in over 50% of cases, although virtually no cases return to normal.^{39–42} Other reports have suggested that nerve repairs carried out as late as 47 months after the injury can result in some success.⁴³ Repairs are normally performed under magnifying loupes or an operating microscope at magnifications of between 4 and 20 times and the use of 8/0 or 9/0 atraumatic nylon sutures. Four to six epineural sutures are normally placed.

The author's current protocol is to recommend immediate microneurosurgical repair (within 3–7 days) for a witnessed total transection, particularly of the lingual nerve, where the ends tend to retract and spontaneous recovery is rare, and also those cases with symptomatic endodontic paste within the inferior alveolar canal. Surgical exploration is also offered at 8–10 weeks postinjury, in those patients who are either totally anesthetic, or have severe dysesthesia, even in the absence of a witnessed transection. For other patients who have less than 30% of normal sensation (i.e. they lack protective reflexes) or have dysesthesia at 4 months, exploratory surgery may be

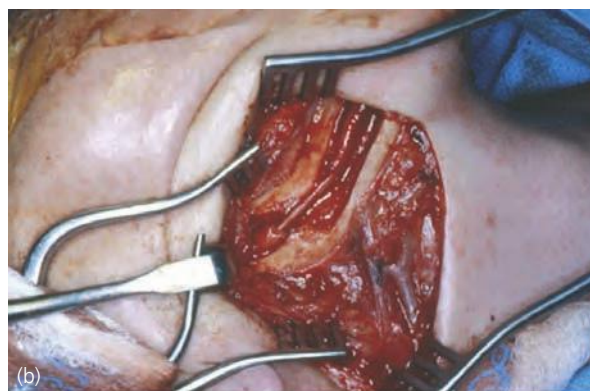


Fig. 16.23 Use of vein graft to reconstruct nerve. (a) A neuroma on the right inferior alveolar nerve – extra oral approach. (b) A facial vein graft used to reconstruct the inferior alveolar nerve.

offered at that time, but the results of surgery are poor.

Surgery appears to have little to offer for nerves injured by local anesthetic injections or by implant-related procedures; in the latter case the area of damage is often extensive and may require a graft.

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Chapter 17

Autotransplantation of Teeth

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This chapter describes autotransplantation of teeth which is a treatment alternative for replacing congenitally missing teeth and teeth lost due to trauma or disease by moving a tooth or a root to a more suitable position within the same individual. This chapter will give an overview of important principles for case selection, surgical technique, and tissue healing which are prerequisites for a successful result. Different situations when autotransplantation can be an advantageous alternative are presented.

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Introduction

Transplantation of teeth has been performed for many centuries.^{1–3} Earlier in history teeth were transplanted from one man to another, so called allotransplantation. This always resulted in a limited survival of the transplant and high failure rate due to immunogenic reactions destroying the periodontal ligament (PDL), resulting in root resorption and loss of the transplant.^{4,5} There is also a risk for transmission of diseases between individuals.

However there are numerous clinical studies during the past decades showing that teeth can be transplanted with good prognosis within the same

individual, so called autotransplantation.^{3,6–67} Experimental research carried out in recent decades has given us knowledge to better understand wound healing following autotransplantation.^{68–79} Autotransplantation of a tooth can be defined as a tooth extracted from one location and placed in a different location within the same individual. Teeth can be transplanted from one place to another and a tooth or a root can also be transplanted to a more suitable position within the same socket.

Although replacement of lost or missing teeth in adults is nowadays more often carried out by implant treatment, there is a number of situations where autotransplantation seems to be a better alternative, especially in growing children and adolescents where implants cannot be used due to the interference with growth of the alveolar process.^{6,7,80,81} A transplanted tooth with PDL will follow and contribute to the development of the alveolar process and in many situations is therefore the first alternative for replacement in growing individuals. There are also situations in adult patients where autotransplantation is a possible alternative.^{3,6,22,30,31,33,35–37,44,59,61}

Donor teeth

Transplants can be taken by using teeth in crowded regions or by strategic extraction when equalizing the number of teeth between quadrants. Moreover, many premolars are extracted as part of the orthodontic treatment and can be used as transplants. Third molars can also be used (Fig. 17.1). The transplanted tooth must have a suitable root length and shape.^{3,6,54,57,58,61,68}

Teeth with roots under development are easier to extract and have better prognosis. Developing teeth can revascularize while fully developed teeth do not revascularize and have to be endodontically treated.^{3,6,7,76–78} It is also important to take into consideration the development and growth status of the individual and an interdisciplinary approach is important when planning.

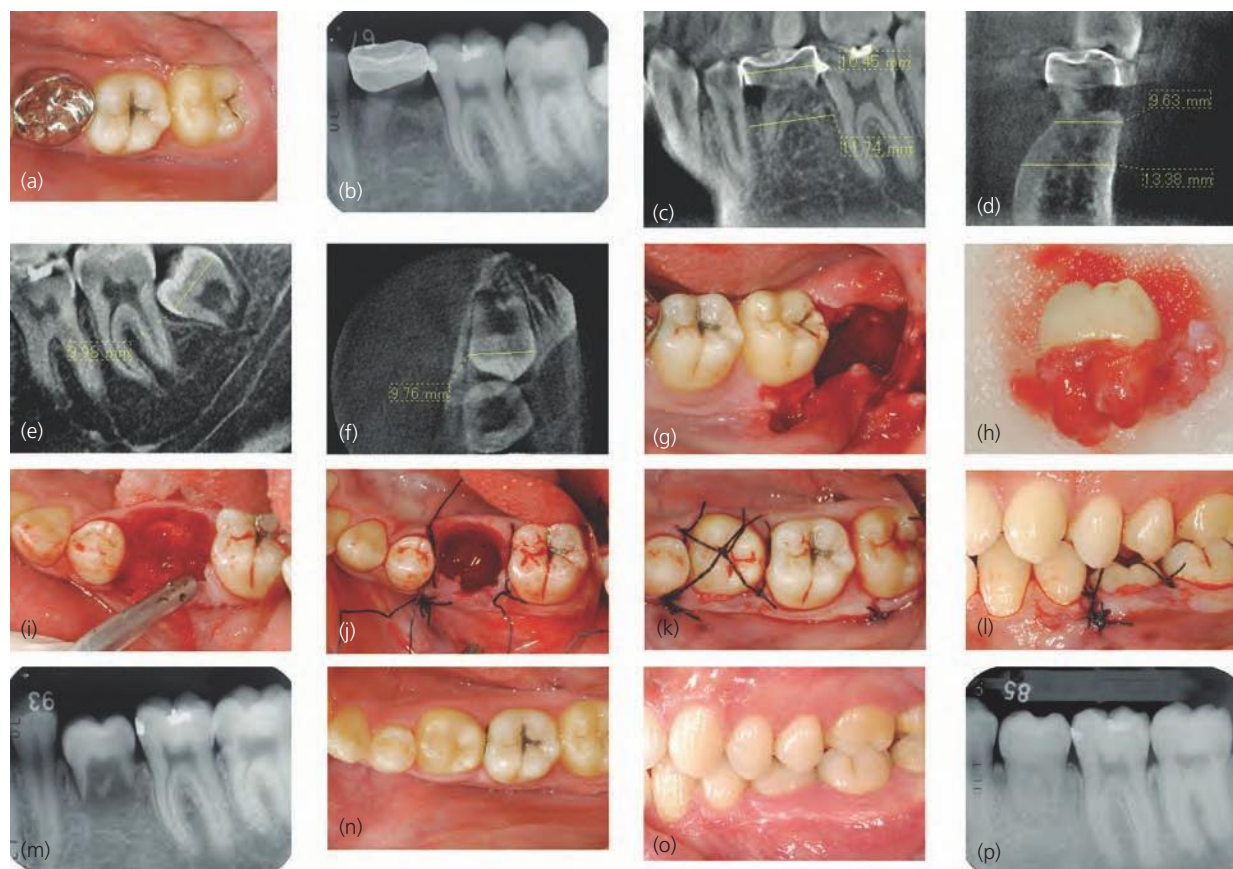


Fig. 17.1 Transplantation of immature impacted third molar to the site of congenitally missing second premolar. (a) The patient is a 20-year-old male with a congenitally missing premolar 35 and a remaining deciduous molar 75. The root has been resorbed. (b) Radiograph showing resorption of 75. (c, d) Cone-beam CT images of the recipient site. The measurement of the recipient space has shown it is wide enough to accommodate the donor mesio-distally and bucco-lingually. (e, f) Cone-beam CT images of the donor tooth. The measurement has revealed the tooth is small enough to be placed into the recipient site. The lower left impacted third molar 38 is considered to be a good donor tooth for transplantation. (g, h) Extraction of the donor tooth 38. Note the Hertwig's epithelial sheath is preserved at each apex. (i) Extraction of the tooth from the recipient site. (j) After careful try-in of the donor into the recipient site, suturing of the flap is performed without placing it in the socket. A knot is tied on the buccal aspect of the interproximal area and about 3 cm of one of the sutures is left. Another interrupted suture is tied across the vertical releasing incision on the mesial aspect. Again, 3 cm of one end is left. Next a suture is placed through the interproximal area of the mesial aspect but in this procedure only one knot is tied loosely so that it allows space for the donor tooth to be placed into the socket. (k) Just after suture splinting. The loosely tied suture is tied tightly after placing the donor tooth. The tooth is tied into place by using a crisscross method. The remaining distal buccal end is tied with the mesio-lingual end and a knot is made on the center of the occlusal surface of the donor tooth. A horizontal mattress suture is tied on the disto-lingual aspect, and the remaining suture is tied to the remaining suture from the first crisscross knot. The remaining suture is then tied to the long remaining end of the suture from the vertical releasing portion. This allows the donor tooth to be seated in the correct position. Any excess suture is cut. (l) Occlusal adjustments are made on the antagonist so that articulating paper can freely slide through the contact. In this case, the opposing tooth was slightly extruded, so with the patient's permission, some adjustments were made to the opposing tooth in contact. (m) Radiograph immediately after transplantation. Note the apices are wide open. (n, o, p) Follow-up after 1 year, 8 months. The transplanted tooth has erupted and is in contact with the antagonist. Pulp healing and root development are seen.

Indications

Congenitally missing teeth

Tooth aplasia is a suitable indication for autotransplantation (Fig. 17.1). Donor teeth can be other erupted or impacted teeth or strategically extracted, e.g. taken from a crowded area or when a tooth does not have an antagonist. Another indication can also be to equalize teeth between quadrants. If the space is too small preoperative orthodontic widening may be necessary.^{3,82} An advantage with this type of transplantation is that a natural biological condition for bone and soft tissue development of the alveolar

process in the young growing patient is achieved and normal eruption is promoted.^{7,8}

Unrestorable teeth

Autotransplantation may be indicated in situations with deep caries or where restoration or crown therapy is not feasible or when endodontic treatment has failed (Fig. 17.2).

Crown-root and root fractures

Intra-alveolar transplantation,⁸³⁻⁸⁶ also called surgical extrusion,⁸⁷⁻⁸⁸ can be used to move a fractured

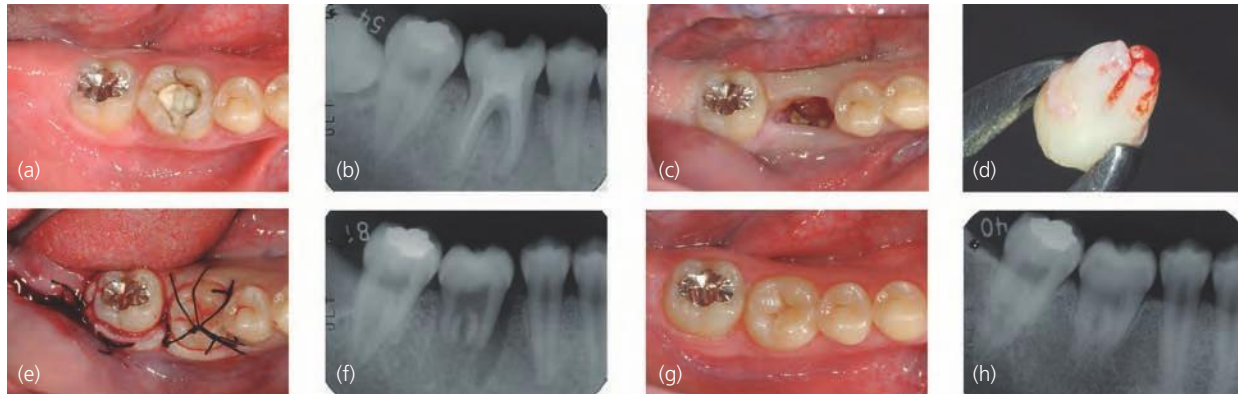


Fig. 17.2 Transplantation immature third molar 48 to replace an unrestorable first molar 46. (a) Occlusal view of the non-restorable 46. (b) Radiograph of 46. It was considered transplantation of an immature third molar would give a better prognosis than preserving 46 in this young patient. (c) 2 weeks after the extraction of 46. Transplantation was performed on the same day. (d) Extracted 48 as a donor. Note Hertwig's epithelial sheaths are preserved apically. (e, f) Immediately after transplantation. (g, h) 4 years' follow-up. The transplant shows a positive electric pulp test and normal healing is seen clinically and radiographically.

root to a more coronal position in the alveolar socket so crown therapy will be enabled. The root can be extracted either directly with forceps or via an open flap approach if the root is submerged. If the remaining root is long enough to carry a crown the root can be transplanted to a more suitable position in the socket. In some cases it may be suitable to rotate the root to 180°, to achieve a larger area of the root within the alveolar bone (Fig. 17.3). By doing this, the root is secured in its new position and does not slide back. Endodontic treatment should be performed within the first weeks after transplantation and crown therapy can usually be finished within 3 months after the transplantation. This method is a first choice of treatment in growing individuals where implant treatment is contraindicated, but can also be performed in adult patients as an alternative to implant treatment (Fig. 17.3).

Lost teeth in young growing patients

Incisors are sometimes lost because of tooth avulsion or cannot be restored after complex trauma. In adults such lost teeth are often replaced by implants; however when the patient is young and still growing implant treatment is contraindicated because implants will not follow the growth and development of the alveolar process. In such a case it can be difficult to achieve a good esthetic final result because the tissues do not develop normally in an esthetically sensitive area. Tooth transplant will enable continuing growth and development of the bone and soft tissues of the alveolar process during the time an individual is still growing and is therefore often a first choice when replacing teeth in young individuals who are still growing (Figs 17.4 and 17.5).

Lost teeth in adults

Teeth lost in individuals who have finished growing are usually replaced by implants. However autotransplantation can also be performed in adult

patients after tooth loss. However since these teeth have fully developed they will not revascularize and must therefore have root canal treatment.

Autotransplants vs implants

Since the introduction of implants many surgeons today prefer implants to replace lost teeth in *adult patients* who have finished growing. However, autotransplantation of teeth can also be performed in adult patients. Third molars may still be undergoing root development in young adults; these can be excellent transplants and revascularization can occur. Teeth with fully formed roots can be transplanted provided root canal treatment is performed. Implant treatment cannot be afforded by all patients and autotransplantation may be an alternative.

In *young growing patients* implant treatment is contraindicated in the anterior maxillary region because it stops the vertical growth of the alveolar process and it can be difficult to achieve a good esthetic final result if an implant is placed before growth is completed. To support the development of normal tissue in the trauma area and to secure growth and development of the alveolar process in an individual who is still growing, transplantation of the tooth is an excellent form of treatment (Figs 17.4, 17.5; see also Fig. 17.13).

Principles of healing after autotransplantation

Two tissues are of main importance for an autotransplantation to be successful, the pulp and the periodontal ligament. When a tooth is transplanted the vascular supply is immediately lost. Revascularization is therefore of utmost importance if the pulp is going to survive. Studies of transplanted teeth have shown that young teeth with ongoing root development and open apex can revascularize (Fig 17.6).^{72,74,76–78} Successful healing is related to the stage of root



Fig. 17.3 Crown-root fracture and surgical extrusion. (a, b, c) A 32-year-old female has sustained dental trauma in the anterior area. Three incisors have been injured. Both central incisors were subjected to lateral luxation with crown fracture and the right lateral incisor suffered crown-root fracture. In this presentation, the treatment is focused on the lateral incisor. (d) Removal of the coronal fragment of 12. (e) The extracted crown root fragment 12 before transplantation. (f) The transplanted 12 immediately after surgical extrusion. The tooth was rotated 180°, so that the palatal aspect is now facing buccally, and transplanted to an extruded position. A suture has been placed to hold the tooth in the correct position. (g) Radiograph immediately after surgical extrusion and rotation of root fragment through 180°. The endodontic treatment was performed before surgery and the canal was filled temporarily with radiopaque calcium hydroxide. (h) Cone-beam CT image immediately after surgery. (i) 5 weeks postoperative appearance just before the restoration of the crown with composite. (j) 4 years' follow-up. The crowns of the three traumatized incisors 12, 11, and 21 have been restored with composite. (k) 4 years' radiographic follow-up. Normal healing is seen. (l) Cone-beam CT image of tooth 12 at 4 years' follow-up. Normal PDL space along the root surface is observed and the thin buccal bone plate is preserved.

development (Fig. 17.7).³ In order to optimize conditions for revascularization, transplantation should ideally be carried out before the root reaches full root length and when the root apex is still open (Figs 17.7, 17.8). However, the transplantation cannot be performed at too early a stage of root development since there is a risk of arrested root development after transplantation (Fig. 17.9). Teeth with fully developed roots and a closed apex cannot be expected to revascularize but can still be transplanted provided root canal treatment is performed.

Pulp

Immature roots (open apex)

In the first weeks after transplantation before the pulp has regained its vascularization, the tissue is very

prone to infection. If the pulp is infected the transplanted tooth can be destroyed by inflammatory root resorption. Experimental studies have shown that systemic antibiotics can reduce this risk and for this reason systemic antibiotic administration is routine during the first week after transplantation.⁷ Routes of infection to the non-revascularized pulp must be avoided, so grinding on the transplant should be avoided since this may open up communication routes for bacteria through the dentinal tubules to the pulp. If these principles are followed infection of the non-vascularized transplant is very rare, and after a few weeks revascularization has taken place. Because of the temporary interruption of vascular supply, later calcification or obliteration of the pulp is seen (Figs 17.4–17.6).^{3,6,7} This is a normal finding in a tissue that has had an interrupted vascular supply and this condition requires no treatment. The condition should



Fig. 17.4 Autotransplantation of mandibular premolar to the central incisor area with a closed technique. (a) Transplanted premolar sutured into position. The postoperative immediate radiograph shows incomplete root development. (b). Follow-up nine years after transplantation. The crown has been built up by composite. Radiograph shows that full root development has been achieved and the usual obliteration is seen indicating the pulp has survived. (From Andreasen, Andersson, and Tsukiboshi.⁷ Reproduced with permission from Wiley-Blackwell.)



Fig. 17.5 (a, b) Autotransplanted premolars. (c) The transplants were later restored with porcelain laminate veneers. (d) Radiograph after restoration. (From Andreasen, Andersson, and Tsukiboshi.⁷ Reproduced with permission from Wiley-Blackwell.)

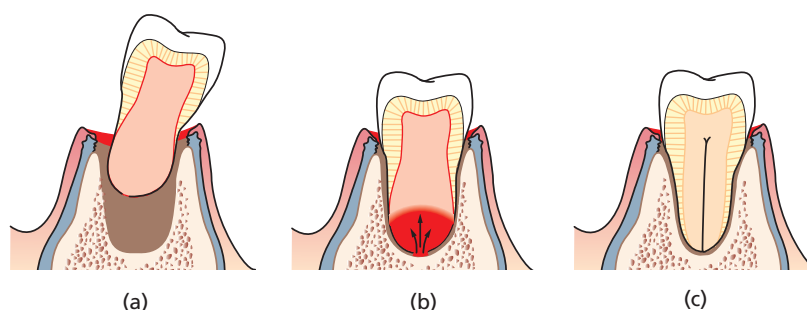


Fig. 17.6 Schematic illustration of pulp healing and root development after transplantation of an immature (open apex) tooth. (a) Before transplantation: an immature tooth, with Hertwig's epithelial root sheath attached to the apex, is transplanted into the recipient site. (b) Several days after transplantation: capillary vessels proliferate coronally from the apical foramen and pulp cells proliferate along with them and the pulp canal is filled with vital tissue during the first weeks after transplantation. (c) Several months later: rapid calcification of the root space occurs resulting in a partial obliteration. At the same time Hertwig's epithelial root sheath may allow for some root development to take place.

rather be seen as a sign that the pulp has survived the transplantation trauma, since it had a capacity to form hard tissue after it had been transplanted.

Mature roots with closed apices

Transplanted teeth with fully formed roots and closed apices will not revascularize and must always have root canal treatment to avoid later infection and inflammatory root resorption.

Periodontal ligament

It is very important for the prognosis of the transplanted tooth to minimize damage to the PDL. It can be damaged in different phases of the transplantation

procedure: when the transplant is mobilized from its original position, when it is stored outside the mouth, and when it is placed in its new position. Small areas of damage to the root surface can heal by proliferation of cells from the side of the defect, however if a larger area of cell damage has occurred during the transplantation procedure, the remaining PDL cannot proliferate and bridge over the defect with new PDL.⁷⁰ Instead there is risk for bone overgrowth from the alveolar socket so the tooth becomes fused with the alveolar bone.⁶⁹ This will result in ankylosis and replacement resorption and the transplant will be replaced by bone and finally lost. To avoid damage and root resorption careful surgical technique is of utmost importance. A higher risk for replacement resorption is seen in transplantation of teeth with

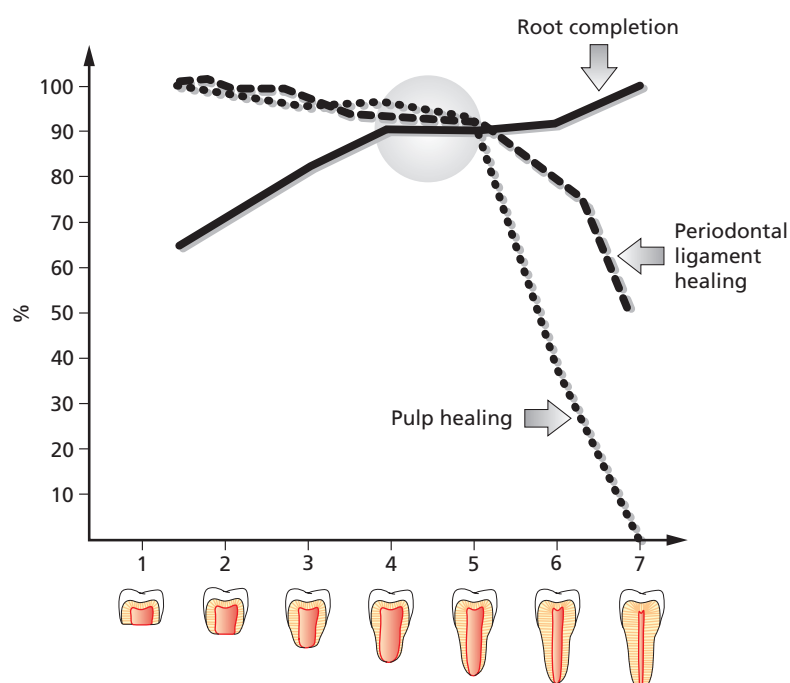


Fig. 17.7 Relation between root length and pulp healing. Stages of root development of donor teeth, modified after Andreasen.³ The solid line represents expected root completion after transplantation. The dotted line represents the probability of revascularization and pulp healing. The dashed line represents periodontal ligament healing. After closure of apex, the chance for revascularization and healing of the dental pulp decreases. Conversely, as there is less root development, the healing of dental pulp increases. For there to be an ideal point where there are both maximized root completion (final root length), periodontal and pulp healing, the autotransplant should be performed when the donor tooth is at stage 4 to 5 of tooth development (i.e. when the root is 70–80% developed).

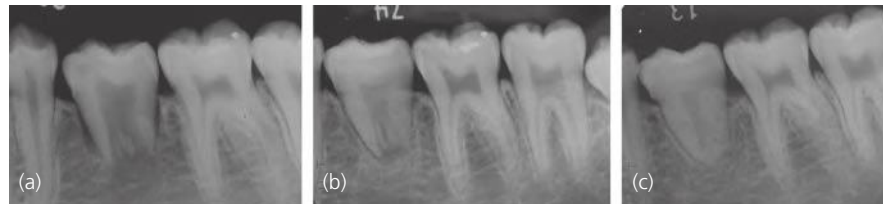


Fig. 17.8 Transplanted molar with optimal root length. The patient is a 17-year-old female. (a) Immediately after transplantation. (b) 8 months later. The root of the transplant is developing. (c) 6 years later. The root development is completed to full root length.



Fig. 17.9 Total arrest of root development after transplantation carried out too early before root development was optimal. The patient is a 16-year-old male. (a) Immediately after transplantation. The upper right third molar is transplanted. (b) 4 months later: Pulp healing is observed but little or no root development is seen. (c) 1 year later: The root development of the donor is almost totally arrested.

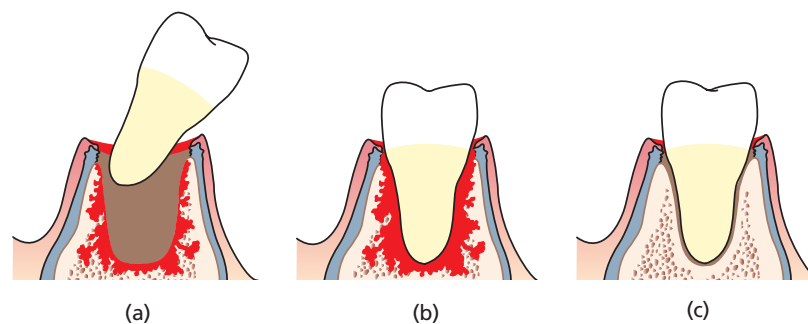


Fig. 17.10 Principle of healing after placement of transplant in a socket which is wider and deeper than the transplant. (a) Before transplantation. The donor tooth 48 with intact PDL is placed in the surgically and newly formed socket 46. Healing of the PDL is dependent on the viable PDL of the donor tooth. (b) Just after transplantation. The PDL which is present above the crestal bone level allows for reattachment to the gingival connective tissue. A blood clot forms between the bony socket wall and the rest of the root. (c) After completion of healing. The blood clot is replaced by granulation tissue and immature bone in 1–3 weeks and mature bone is in place after about 1–6 months, depending on the width of the space and the age of the patient.



Fig. 17.11 Case illustrating bone healing capacity of PDL after transplantation. (a) Before transplantation. Tooth 36 is going to be replaced by 38, which is not in occlusion and lacks an antagonist. (b) Just after transplantation. Note the wide space between the donor and the socket walls. (c) 2 years postoperatively. New bone has been formed and a normal PDL space is seen around the transplanted tooth.

fully developed roots, probably because these teeth are more difficult to manage without damaging the PDL.³ If there is a chance of choosing a transplant with a root under development the prognosis is better. Short-term fixation and some mobility during the healing period will promote healing with periodontal ligament.^{91,92} For this reason transplants should not be rigidly splinted and can be held in place by sutures or a flexible splint instead of rigid splints. The PDL of the transplant also has an osteogenic regenerative capacity and bone will be formed around the transplant provided the PDL is not severely damaged

even if the transplanted tooth is placed without bone contact (Figs 17.10–17.13).^{3,6,7}

Principles of surgery

Possible transplants can be strategically extracted teeth, e.g. premolars from areas with crowded teeth or areas where the number of teeth in a quadrant is going to be reduced and equalized to an adjacent quadrant. The treatment should therefore ideally be planned together with an orthodontist who can



Fig. 17.12 Case illustrating bone healing capacity of the PDL after transplantation. The patient is a 15-year-old female. (a) Just after transplantation. The tilted and ectopically erupted upper right second premolar has been transplanted on the aplasia site 45. (b) 3 months postoperatively and just after the root canal treatment. The necrotic pulp was removed and calcium hydroxide preparation was placed for apexification. Note that new bone has started to form around the transplant. (c) 1 year, 5 months postoperatively. Normal healing with complete bone regeneration.

advise on a suitable transplant and perform preoperative and postoperative orthodontic treatment when required. It is important to have a thorough preoperative radiographic examination with panoramic radiographs and periapical dental films of both the donor and recipient areas to estimate root anatomy, root development, and space conditions. Cone-beam computed tomography (CT) is a helpful preoperative tool (Figs 17.1 and 17.13). Other important structures such as the mandibular canal, mental foramen, and maxillary sinus must also be taken into consideration. The available space in the recipient area is measured with cone-beam CT but a sliding caliper and study casts are also helpful if the donor tooth is erupted. A simple conical root anatomy of the transplant is ideal. A position in the jaw enabling a non-complicated removal is especially valuable for the prognosis. Transplants in complicated positions must be avoided since removal will often result in damage to the PDL. Estimation of the degree of root development is the key factor in order to time the transplantation. A root development of 70–80% seems to be optimal (Fig. 17.7).

Surgical techniques

A prerequisite for a successful result of transplantation is that the capacity of the tissues for healing and regeneration is preserved. For this reason there are special requirements regarding the surgical techniques. It is important that the surgeon has a full understanding of the highly sensitive tissues involved. At the moment of extraction of the transplant, the pulpal tissue in the transplant is losing its vascular supply and must later be revascularized. In order to protect the pulp from infection, systemic antibiotics are given 1 hour before surgery so there is a high concentration of antibiotics in the pulp tissue. The surgical trauma must be minimized so that the transplant and the tissues of the recipient region are not damaged; this means that the recipient area must first be prepared before moving of the transplant. After careful raising of the flap the alveolus is prepared under intense cooling with saline. For this reason measurement of the donor tooth should be performed preoperatively on radiographs and clinically if the

transplanted tooth is erupted. In many cases the alveolar bone is completely removed which does not seem to have any negative effect on the PDL as long as the PDL of the transplant is intact.^{3,6,7} An intact PDL has an ability to form new periodontal tissue including new bone (Figs 17.10–17.13). Soft tissue in the recipient region should be protected during the preparation. The next moment is exposure and loosening of the transplant. An unerupted tooth germ can usually be lifted out with its intact follicle and root sheath. In ideal situations the transplant can be moved directly over to the recipient site. However, adjustments are often needed and the extra-alveolar time should be kept as short as possible. The transplant can be stored in physiologic saline if preparation of the recipient site is required. A sliding caliper or probe can be used for measurement. Plastic or metal dummies or even earlier extracted autoclaved premolars can be used as try-in dummies. Changing the direction of the tooth by rotating it 90° can help to find a better fit. Grinding on the transplant should be avoided. Grinding of the enamel of the adjacent teeth may be necessary but the space should ideally have been prepared before surgery by orthodontic treatment. The graft should ideally be placed somewhat below the occlusal plane and placed passively into its new position without any pressure from the adjacent tissue. The flap is replaced, covering the transplant without pulling too hard on the tissue, and the transplant is secured with a suture over the occlusal surface or with a flexible splint (Fig. 17.1).

Postoperative care

The patient should have systemic antibiotics and should be recommended a soft or liquid diet and rinsing with chlorhexidine during the first week. After 1 week the sutures can be removed and after 4 weeks the transplant will have achieved almost normal mobility. During the first year, eruption and continuing root development with obliteration of the root canal are signs of successful transplantation with revascularization and pulp survival. Teeth with fully formed roots should be root canal treated and this should be initiated 2 weeks after transplantation.

Long-term prognosis

There are some studies reporting tooth survival after tooth transplantation in almost 100% of cases. However, evaluating with more strict criteria, i.e. not only survival but healing without any signs of progressive root resorption during an observation time

of several years, achieves a more relevant success rate. Using such evaluation criteria the success for a transplanted molar is 72–90%, for premolars is 89–98%, and for canines is 25–81%.³ The higher success figures are for studies with teeth with developing roots with open apices and revascularization. Transplanting teeth with fully developed roots has a

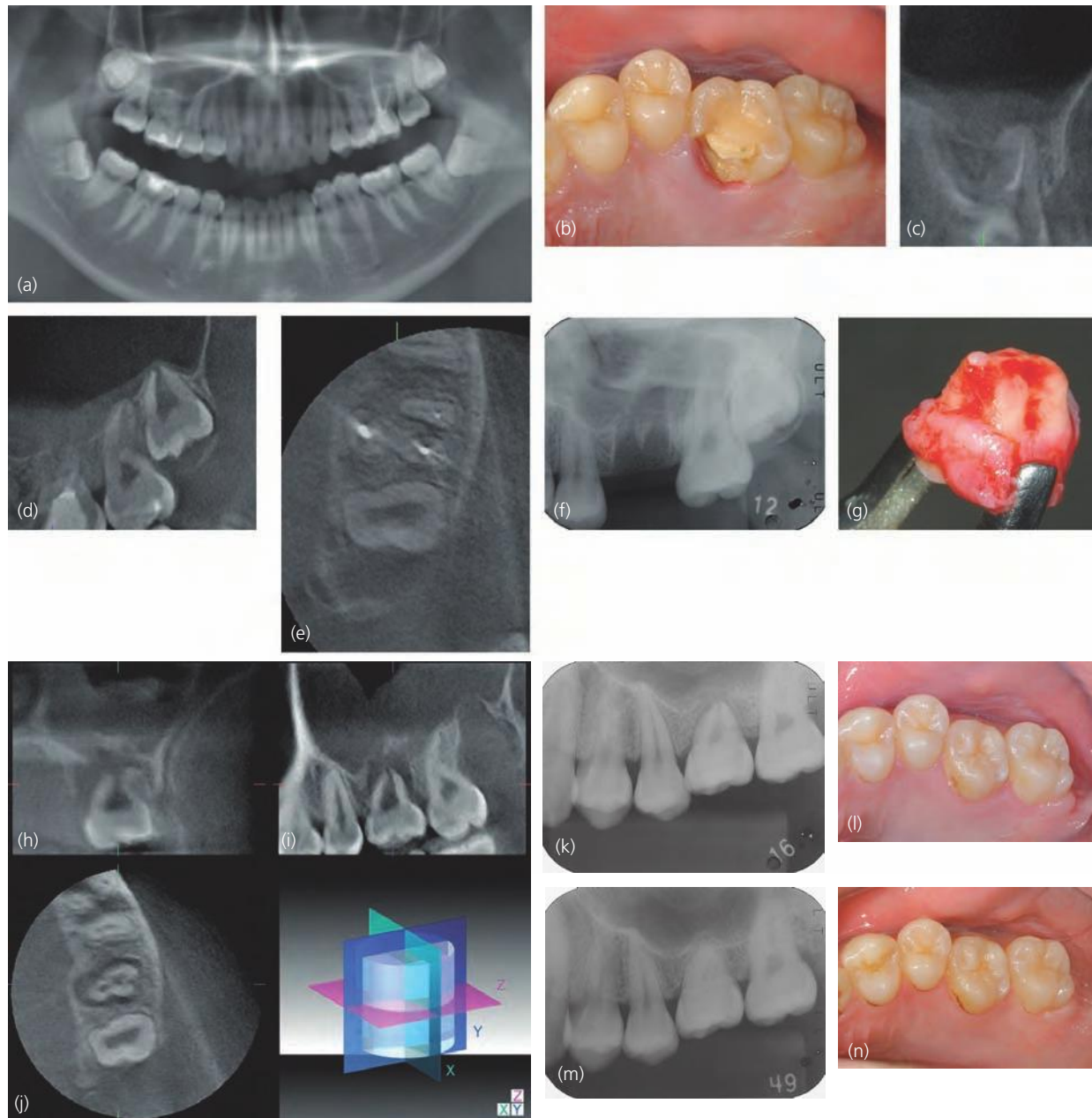


Fig. 17.13 Transplantation with sinus elevation. (a) Panoramic views of 21-year-old male at the first examination. He had an infection in an earlier endodontically treated tooth 26. Tooth 28 is a potential donor tooth. (b) Clinical picture showing deep palatal crown–root cusp fracture. (c) Cone-beam CT facial image around the recipient site. There is apical periodontitis which has caused the breakdown of the sinus floor around the mesio-buccal root. In addition, the sinus floor is located inferior to the root apex. The vertical bone height is 5 mm. (d) Cone-beam CT sagittal image of the donor tooth verifying its suitability as a donor tooth with conical root shape and open apex (stage 5, Fig. 17.7) with a diameter wider than 1 mm which means there is a chance for pulp revascularization after transplantation. (e) Cone-beam CT coronal image of the recipient. The second canal of the bucco-mesial root of 26 has been untreated and caused the apical inflammation. (f) Radiograph taken 2 weeks after extraction of 26 and just before transplantation. (g) The extracted donor tooth 28, an open apex with Hertwig's epithelial sheath is observed. (h) Radiograph immediately after transplantation. Note the sinus has been elevated to enlarge the vertical space to accommodate the donor. (i) Clinical view immediately after transplantation. (j) Cone-beam CT images 3 days after the surgery. There is a wide bony space around the transplant. (k, l) 5 months' follow-up. Bone has formed and the transplant showed positive sensibility. (m, n) 3 year, 5 months' follow-up. PDL healing, normal pulp and bone healing. (*cont'd*)

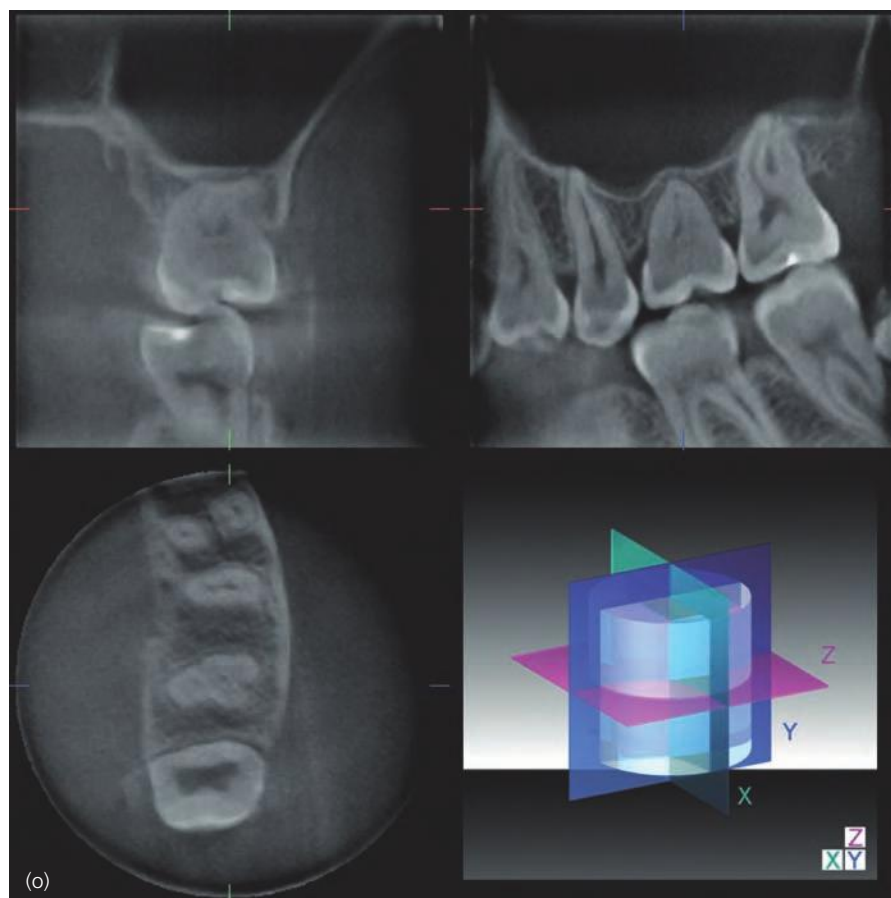


Fig. 17.13 (cont'd) (o) Cone-beam CT images at 3 years, 5 months' follow-up showing successful bone regeneration and healing.

lower success rate due to a higher risk for damage of the PDL with resulting root resorption. There have been studies trying to improve the prognosis in auto-transplanted teeth with fully developed roots by using a two-stage technique.^{35,42,43,77} For intra-alveolar transplantation (surgical extrusion) a success rate of 88–90% has been reported.^{83–89} Numerous clinical and experimental studies of autotransplantation and replantation have resulted in today's understanding of the healing process and how to avoid or reduce the risk of complications after autotransplantation.^{3,5,6,7,53–58,60,63–65,67–71,74–80,90–94}

Summary

Transplantation of teeth is a well documented treatment method that should always be taken into consideration as a first treatment alternative in young growing patients with congenitally missing teeth and in situations of tooth loss after trauma in growing patients. A transplanted tooth can contribute to normal development of the alveolar process when the patient is still growing. In adult patients there are also certain situations where autotransplantation can be a preferable alternative to other treatments.

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Chapter 18

Endodontic Surgery

Peter Carrotte and Colin Murray

Periradicular surgical procedures are carried out to treat diseases and conditions of the tooth root that are not amenable to orthograde root canal treatment. The majority of these surgical procedures will involve resection of the root apex (apicectomy) and retrograde obturation of the root canal to address persistent disease that has not resolved following an acceptable root canal treatment. Other surgical procedures on the tooth root, including the repair of resorption defects and root perforations, will also be considered in this chapter. Great emphasis has been placed on describing modern microsurgical techniques and instrumentation, and a comprehensive literature review is included to support this move away from a more conventional surgical approach. These microsurgical techniques appear to result in improved prognosis, with better and more esthetic healing of the alveolar and gingival tissues. This is of considerable importance in today's climate of esthetic dentistry and patient expectations.

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Introduction

Periradicular surgery procedures

Periradicular surgery (PRS) is a generic term for treatment that encompasses three main categories of surgical endodontic procedures:

1. Periapical curettage of persistent periradicular disease, including removal of the root apex (root-end resection) and retrograde filling of the root canal.
2. Surgical repair of root surface irregularities such as external root resorption defects or iatrogenic perforations.
3. Root resection of posterior teeth to remove diseased roots and retention of roots suitable for further coronal restoration.

The majority of PRS procedures are related to the first of the above categories. This is a procedure often referred to as an apicectomy (or apicoectomy) and will be discussed first in this chapter. The latter two surgical endodontic groupings will be discussed towards the end of the chapter. However, the primary objective of all these surgical therapies is to repair any underlying defect thereby facilitating regeneration of local dentoalveolar tissues.

Box 1 Abbreviations used in this chapter

CAD/CAM	Computer-aided design/computer-aided manufacturing
CBCT	Cone-beam computed tomography
EDTA	Ethylene diamene tetra-acetic acid
GTR	Guided tissue regeneration
MTA	Mineral trioxide aggregate
NSAIDs	Non-steroidal anti-inflammatory drugs
NSRCT	Non-surgical root canal treatment
PRD	Periradicular disease
PRS	Periradicular surgery
SEM	Scanning electron microscopy
SOM	Surgical operating microscope

Historical perspective of surgical endodontics

The first published description of a periradicular surgical procedure found by the authors was in 1890. Using examples of his own clinical cases as illustration, Dr Rhein¹ began by suggesting that *“although amputation of a portion or the whole of the root for the restoration of health of teeth which did not yield to milder treatment had long been advocated by the few, the textbooks were singularly quiet about this important procedure”*. He proceeded to describe the technique known today as “through-and-through” apicectomy as follows. *“Usually the operation presented no difficulty and, except in the region of the antrum or mental foramen, no dangerous anatomical points were involved. The instruments required were a sharp spear-shaped drill to open through the process and root, and a new fissure-bur, which, following the drill and worked laterally, severs the root. As a rule, no anaesthetic was required...”* The method of treatment which the writer advised was *“To fill the root or roots, excise the diseased portion, following with a vigorous use of the bur in the surrounding pathological tissue. If the operation were performed under aseptic conditions and the parts kept so until the wound was entirely healed, an immediate and radical cure resulted.”* The first published description of an apicectomy coupled with a retrograde cavity and restoration of the root apex found by the authors was in 1916. Using excellent clinical photographs, Carl Lucas² described a procedure which bears remarkable similarities to the conventional surgical techniques advocated until quite recently, before today’s oral surgeons recognized the importance of, and adopted, microsurgical techniques described in this chapter. Lucas even appears to have used the “Ochsenbein-Luebke flap” which would not be described in the literature for another 10 years.³

Initial clinical assessment

Prior to embarking upon any PRS procedure, the operator must undertake a comprehensive medical and dental assessment combined with a thorough extra- and intraoral examination. The overall condition of the mouth, including both the soft tissues and dentition, needs to be assessed. In particular, the presence of local infection, swelling and sinus tracts should be recorded. The caries status of teeth should be evaluated and the presence of and quality of the coronal restoration on the tooth requiring surgery should be carefully considered. If the tooth is restored with an indirect restoration, the patient should be asked if it has ever become decemented or debonded. The periodontal status of the tooth, in addition to the rest of the dentition, requires careful evaluation with appropriate periodontal probes for assessing the presence of isolated deep pockets and analysis of tooth mobility. The occlusal function of the tooth should be noted and it should be ascertained if the

tooth is a functioning unit. Radiographic assessment is covered later in this chapter.

Why periapical surgery?

An apicectomy is indicated when conventional non-surgical root canal treatment (NSRCT), with an orthograde root canal filling, has failed or is impractical and the tooth is associated with clinical symptoms or signs of continuing periradicular disease (PRD). It must be emphasized at the outset that a surgical approach is not a substitute for providing satisfactory orthograde root canal treatment. If orthograde treatment has failed, the reason for failure should be diagnosed and, whenever possible, NSRCT retreatment attempted. Therefore, surgical root canal procedures should not be considered as the primary treatment option to treat teeth with associated apical pathology.⁴

The radiographs in Figs 18.1 and 18.2 depict two such cases. An inadequate root canal treatment that has failed is illustrated in Fig. 18.1. A periapical radiolucency persists, which was associated clinically with a draining sinus. The reason for failure is inadequate preparation and disinfection of the root canal system. Before even considering a surgical approach, such a case should be endodontically treated by orthograde retreatment to the accepted standard. A root canal treatment that has failed because an endodontic instrument has separated in the root canal preventing radicular access for disinfection and obturation is presented in Fig. 18.2. If it is not possible to remove the separated instrument and undertake orthograde retreatment, a surgical procedure may be necessary. Surgery has been demonstrated to be more commonly performed if there are persisting symptoms and/or progression of the periradicular lesion and in teeth restored with post and core crowns.⁵ Before proceed-



Fig. 18.1 A radiograph showing an inadequate root canal treatment that has failed. A periapical radiolucency persists, which was associated clinically with a draining sinus. The reason for failure is inadequate preparation and disinfection of the root canal system. Before even considering a surgical approach, such a case should be endodontically treated by orthograde retreatment to the accepted standard.

ing, it may therefore be helpful to briefly review current guidelines for achieving satisfactory orthograde root canal treatment.

Root canal infections

Kakehashi and co-workers⁶ demonstrated more than 40 years ago that pulpal and subsequent periradicular disease is caused by microbial contamination. This commonly occurs via a carious lesion. As resultant necrosis of the pulpal tissue proceeds, initially inflammatory products and later pathogens and their by-products exit the confines of the root canal system through the apical foramen. Unless there is a dysregulated acute inflammatory reaction resulting in an apical abscess, chronic periapical periodontitis develops. This frequently results in the formation of a PRD lesion, most commonly an apical granuloma (Fig. 18.3). This chronic inflammatory tissue response comprises granulation tissue, containing fibroblasts, epithelial cells, lymphocytes, neutrophils, plasma cells, mast cells, and macrophages, and directly contributes to destruction of the surrounding dentoalveolar bone.

An effective immune response leading to resolution of PRD is not possible as the source of infection, contained within the root canal space, is inaccessible to mediators of host immunity. Therefore, the pathological apical lesion will only resolve when the origin of infection is eradicated, the root canal system effectively sealed, and a satisfactory coronal restoration placed. An effective three-dimensional seal is prerequisite to successful outcome. As it is never possible to completely sterilize the root canal system, an apical seal will prevent egress of any remaining toxins into the surrounding periradicular tissues. Furthermore, an effective coronal seal is essential to prevent re-entry of microorganisms from the oral cavity into the root canal.

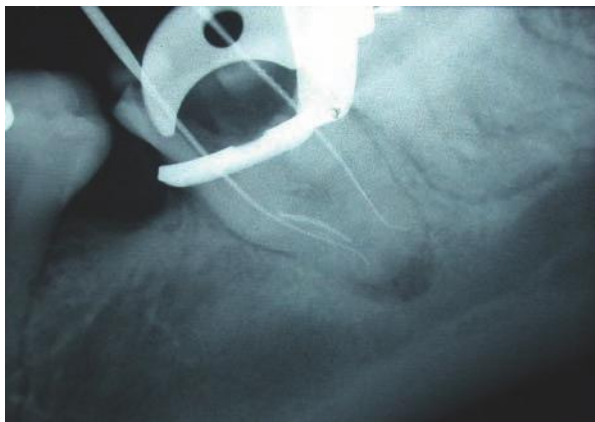


Fig. 18.2 A radiograph showing an attempt at a root canal treatment that has been referred for specialist treatment because an endodontic instrument has separated in the root canal preventing radicular access for disinfection and obturation. If it is not possible to remove the separated instrument and undertake orthograde retreatment, a surgical procedure may be necessary.

Orthograde root canal treatment

NSRCT is performed using endodontic files and other instruments to shape the canal sufficiently in order to permit the ingress of antibacterial medicaments to the entire root canal system. The antiseptic irrigant of choice is a solution of sodium hypochlorite, the concentration of which is between 1% and 5% depending upon the operator's preference. In conjunction with this, a solution to disperse the smear layer formed on the root canal wall during instrumentation should be used; 17% EDTA (ethylene diamene tetra-acetic acid), which will disrupt the bacterial biofilm and smear layer, is preferred for this purpose but citric acid is also suitable. Other antibacterial solutions have been advocated for disinfection of the root canal system including 0.2–2% chlorhexidine gluconate and iodine–potassium–iodide. However, these lack the tissue dissolution properties of sodium hypochlorite and are not recommended as the sole irrigant for *de novo* treatment.

If the root canal space is inadequately prepared and shaped, the disinfecting irrigant solutions will not reach the apical extremity of the canal and infection will likely persist. Fig. 18.4 shows an example of failed root canal treatment due to inadequate shaping of the apical part of the root canals. If PRS was undertaken in such a situation, the reservoir of infected material remaining within the root canal system would significantly reduce the prognosis.

Non-surgical root canal treatment outcomes

Many clinical studies report high success rates for NSRCT of greater than 90%.^{7,8} Conversely, others have reported the success of NSRCT to be as low as 45%.⁹ Epidemiologic studies consistently report significantly lower rates of NSRCT success than



Fig. 18.3 An apical granuloma attached to the root apex. The chronic inflammatory response comprises granulation tissue containing fibroblasts, epithelial cells, lymphocytes, neutrophils, plasma cells, mast cells, and macrophages and directly contributes towards destruction of the surrounding dentoalveolar bone.



Fig. 18.4 A radiograph showing a failed root canal treatment due to inadequate shaping of the apical part of the root canals. If PRS was undertaken in such a situation, the reservoir of infected material remaining within the root canal system would significantly reduce the prognosis.

endodontic treatment undertaken within controlled clinical settings.¹⁰ Indeed, the majority of investigations reporting high success rates in the treatment of PRD are limited to procedures undertaken upon selected cases treated by endodontic specialists or supervised trainees.¹¹ Nevertheless, Cheung¹² ascertained that success rates of root canal treatment in patients receiving treatment within training institutions can be as low as 50%.

Studies analysing cross-sections of the general population identify that the technical quality of NSRCT is performed to a satisfactory standard in only 30–42% of cases.¹³ It appears that root filled teeth are 5–12 times more likely to have radiographic evidence of PRD than teeth without root fillings.^{13,14} Furthermore, teeth with inadequate root fillings demonstrate a significant correlation with the presence of PRD.¹⁵ Part of the reason for treatment failure with root canal fillings may be attributed to the lack of effective intracanal medicaments that eliminate a broad spectrum of microorganisms during cleansing of the root canal. A recent meta-analysis of published root canal treatment literature calculated the success rate for root canal treatment to be 82.8% for vital teeth and 78.9% for non-vital teeth. This provides a cumulative success rate of 82% for teeth without evidence of PRD and 71.5% for teeth with a periradicular lesion.¹⁶

Periradicular disease lesions and outcomes

Associations have been made between failed root canal treatment and missing adjacent teeth, greater plaque accumulation, the degree of marginal bone support, a history of trauma to the treated tooth, the lack of postoperative coronal restoration, and older patients.^{17,18,19} Importantly, current evidence clearly identifies that the most significant factor influencing the successful outcome of NSRCT is the presence and magnitude of a PRD lesion prior to commencing treatment.^{16,20,21}

In addition to localized tissue destruction, it is suggested that chronic persistence of PRD may have an effect upon the host's systemic inflammatory mediators.²² Indeed, a reduction in peripheral blood levels of acute-phase proteins compared to controls has been demonstrated after completion of endodontic treatment.²³ However, it is only gradually being recognized and accepted that the host's immune response is an important factor in the outcome of endodontic treatment, this having previously been largely ignored.²⁰

Microflora of the PRD lesion

There is controversy as to whether the PRD lesion *per se* is sterile or whether microorganisms invade and inhabit the inflamed tissue.²³ Early studies provided evidence that PRD lesions contained significant numbers of bacteria.^{24,25} Nonetheless, others^{26,27} were not able to corroborate these findings. Recent investigations^{28,29} have reinforced the conjecture that PRD tissue contains bacteria. Moreover, when using conventional culture techniques, these bacteria are established as being viable.^{30,31} Indeed, it has been demonstrated that as many as 90% of PRD lesions contain cultivable bacteria.³² Nevertheless, microbes are frequently found in only very small numbers within the lesion.³³ Furthermore, only a small percentage of PRD tissue has been shown to contain bacteria within the body of the lesion.²³ It therefore remains controversial as to whether the majority of these microorganisms are contaminants arising during surgical removal of lesions. The possible contribution of these extraradicular microbes towards the perpetuation of PRD therefore remains to be elucidated.

Apicectomy procedures

Historically, the operation of "apicectomy" has frequently been performed without due regard to these aforementioned endodontic concerns thereby resulting in literature reports of a poor prognosis from such surgical procedures. In 1970, Harty *et al.*³⁴ published a report of 1016 periradicular surgery cases claiming a success rate of 90%. However, almost 30% of their

cases did not return for recall. Indeed, two other papers published during that time, using amalgam retrograde fillings, established prognoses following apicectomy procedures of only 59–70%. In 1992, Frank *et al.*³⁵ observed that in cases where amalgam had been used as the retrograde filling material the success rate was less than 56%. The concluding sentence in their paper reads, “It is our hope that this report will be an impetus for further investigations on materials and methods to replace apically placed amalgam fillings to give improved long-term results.”

It is therefore essential to note that recent reports of outcomes from PRS procedures using modern techniques and materials outlined in this chapter have been much more favorable. When using magnification and ultrasonic root-end preparation, Taschiere *et al.*³⁶ recorded successful outcomes in over 92% of their PRS cases. Tsesis *et al.*³⁷ presented a comprehensive review of the literature of prognosis of traditional and modern techniques. Employing current surgical techniques described in this chapter, they observed complete healing in 91% of cases. This is in comparison to successful outcome in only 44% of cases where traditional surgical methods were used for PRS. Before embarking upon PRS, the clinician must therefore be fully conversant with current orthograde endodontic teaching and practice. Thereby, cases that are more suited to NSRCT retreatment will be directed accordingly towards such treatment modalities rather than PRS approaches.

Therefore, in teeth with failing root canal treatment and persisting periradicular lesions, NSRCT retreatment is generally regarded as the treatment of choice as this has a higher success rate than surgical procedures.³⁸ Nevertheless, in certain clinical situations root canal retreatment may not be feasible. Teeth restored with large post-core crowns may suffer root fracture on attempting post removal and some teeth may have inaccessible canals. According to the Royal College of Surgeons of England guidelines, periradicular surgery may therefore be the preferred treatment option in these cases.³⁹ Indications and contraindications for undertaking PRS are outlined in Table 18.1.⁴⁰ It is evident that many patients referred by general

dentists for PRS actually require NSRCT retreatment.⁴¹ It is therefore important that the surgeon has a good liaison with endodontic colleagues for the appropriate management of such cases.

The surgical procedure aims to remove the necrotic and infected dental root apex, curettage and remove the periradicular lesion, and seal off the apical aspect of the root canal. Indeed, the apical seal of the root canal with a retrograde root filling has been demonstrated to be a major factor towards the successful outcome of PRS.^{34,42} There have been several significant advances in endodontic surgical procedures over the past decade. These include the development of improved root-end filling materials such as mineral trioxide aggregate (MTA),^{43,44} the introduction of magnification,⁴⁵ and the use of ultrasonics in root-end preparation.^{36,46} We discuss each of these in turn in this chapter.

Local anatomic considerations

General principles in the handling, management, and healing of the soft tissues of the oral cavity, both alveolar mucosa and attached gingiva, are discussed elsewhere in this textbook. However, before embarking upon PRS some fundamental observations on the hard tissues, both bone and teeth, are of particular relevance to PRS treatments.

Mandible

When carrying out PRS in the mandible, consideration must be given to the path of the inferior dental nerve bundle in the mandibular canal, which lies in close proximity to the root apices. Surgeons must also be aware of the position of the mental nerve exiting through the mental foramen, which lies between the apices of the lower premolars. If a vertical relieving incision is required when gaining access to posterior mandibular teeth, it should be made over the mandibular canine, having carefully palpated the mental foramen. If a more distal incision is prescribed for PRS to the molar teeth, then care must be taken to avoid the facial artery as it crosses the border of the

Table 18.1 Indications and contraindications for surgical endodontics. (Adapted from ESE guidelines 2006.⁴⁰)

Indications for surgical endodontics	Contraindications for surgical endodontics
Radiological findings of periradicular periodontitis and/or symptoms associated with a canal where an obstruction cannot be removed or will result in damage	Local anatomic factors, such as an inaccessible root end
Extruded material with clinical or radiological findings of apical periodontitis and/or symptoms over a prolonged period	Tooth with inadequate periodontal support
Persisting or emerging disease following root canal treatment where retreatment is inappropriate	Uncooperative patient
Perforation of the root or pulp chamber floor which cannot be treated from within the pulp cavity	Patients with a severely compromised medical history
Biopsy of persisting periradicular lesion is required	Tooth is unrestorable
If patient considerations preclude prolonged non-surgical root canal retreatment	Inexperienced operator with no training

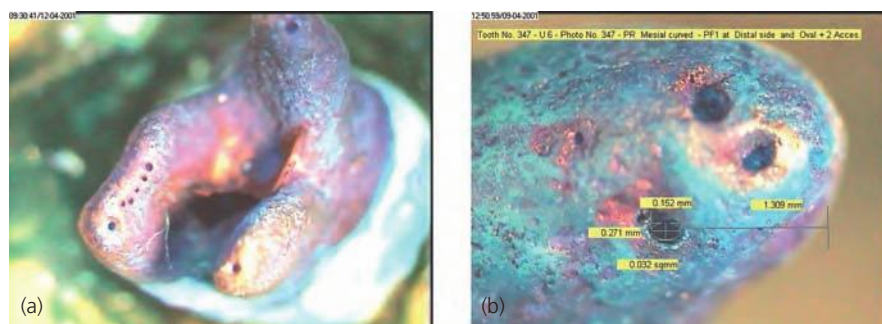


Fig. 18.5 The root apex frequently possesses multiple portals of exit. (SEM illustrations courtesy of Professor Benjamin Briseño.)

mandible adjacent to the first molar. The further posterior the tooth, the denser the overlying bone will be and the neurovascular bundle will be closer to the root apices. Access to second mandibular molars may be so difficult as to preclude surgery. A degree of difficulty may also be experienced when attempting to access lower incisor roots. A shallow sulcus, especially when coupled with a prominent mandibular protuberance, together with the lingual inclination of the roots may make access to the root apices extremely difficult. As 40% of lower incisors have two root canals, these difficulties may preclude adequate resection and retrograde filling of the root apex which may ultimately lead to treatment failure.

Maxilla

The principle anatomic consideration when performing PRS in the maxilla relates to the maxillary sinus. As the floor of the sinus may be only 1 or 2 mm above the root apices of the posterior dentition, the risk of perforation when disease is present is relatively high. Should preoperative assessment suggest that exposure of the sinus during surgery is a possibility then incisions should be designed to ensure that the operation site can be completely closed with a mucoperiosteal flap. Considerable care must be taken to prevent debris from entering the sinus during the procedure and, if adhered to, postoperative sinus-related problems may be avoided.⁴⁷

The other anatomic consideration in the maxilla relates to the position of the palatal root apices. It may be impossible to access these from a buccal approach, thereby necessitating a palatal flap. However, the greater palatine artery may restrict the size of flap raised and access can be difficult. Furthermore, replacement of palatal flaps can be challenging due to the concave shape of the palate and the potential for blood pooling beneath the flap. A surgical stent for postoperative use may be prepared in advance should such problems be anticipated.

Root and root canal morphology

A good knowledge of dental anatomy is essential to ensure that apical root resection is effective. The shape of individual roots varies as does the number

of root canals. Indeed, it is now accepted that root canal systems have much greater complexity at the level of root resection than previously recognized.⁴⁸ Furthermore, the root apex frequently possesses multiple portals of exit (Fig. 18.5). West⁴⁹ demonstrated that 100% of failed root canal specimens had unfilled or underfilled portals of exit at the apical area. Therefore, at least 3 mm of the root apex should be removed to ensure the best possible chance of successful retrograde obturation of all portals of exit. The angle of resection should be as close to horizontal as possible (see “Root-end resection” on page 303). The serious flaw in the traditional method of root resection at an angle of 45° can be seen in Fig. 18.6.

Preoperative imaging

Maxillofacial imaging is covered elsewhere in the textbook. However, certain aspects are of fundamental importance to undertaking PRS procedures. Conventional radiology has been the principle technique for assessing periradicular anatomy prior to performing endodontic surgery. This facilitates the assessment of the number, length and shape of the roots, the presence of a sclerosed canal, and type of

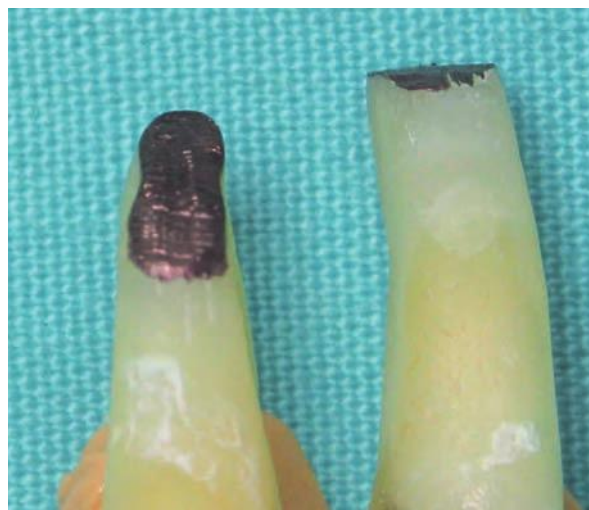


Fig. 18.6 The apex of the tooth on the left has been sectioned at 45° to the long axis. The serious flaw in the traditional method of root resection at an angle of 45° can be seen by the increased amount of infected root canal exposed compared to resection at 90° (right).



Fig. 18.7 A surgical operating microscope (SOM) in use during routine root canal treatment.

restoration present. However, these two-dimensional (2D) images of three-dimensional (3D) structures have their limitations. For example, conventional periapical radiographs do not facilitate accurate predictions in the preoperative assessment for the potential perforation of the maxillary sinus.⁵⁰

As a consequence, the use of 3D cone-beam computed tomography (CBCT) imaging is now becoming

established in preoperative assessment for PRS. The detail provided by 3D CBCT imaging in diagnosing the precise size and location of resorption defects⁵¹ or periradicular pathology^{52,53} is greatly beneficial over conventional radiography. Such enhanced pre-operative information allows improved precision in performing PRS procedures, thereby preventing complications arising through trauma to adjacent anatomic structures.⁵⁴ There is also emerging evidence that CAD/CAM guidance using CBCT imaging is capable of delivering more accuracy and consistency when undertaking endodontic surgical techniques.⁵⁵

Equipment and materials

Magnification

The modern approach to PRS is through magnification, illumination, and microsurgery. As referred to earlier, the dental literature is now reporting greatly improved prognosis when these techniques are employed.⁵⁶ Surgeons who have developed their skills in this field would not return to traditional PRS procedures. Although loupes are helpful, the improved illumination and magnification make the use of the surgical operating microscope (SOM, Fig. 18.7) essential for PRS.⁵⁷ A typical microsurgical instrument set-up is demonstrated in Fig. 18.8. Individual microsurgical instruments in comparison to their normal counterparts are presented in Figs 18.9–18.11. In addition to the SOM, endoscopy is being employed as a supplementary aid to further enhance magnification during PRS.⁵⁸

Along with greatly improved vision and illumination, microsurgery has many other benefits. The operator's posture is fixed comfortably in relation to



Fig. 18.8 A typical microsurgical instrument set-up for undertaking periradicular surgery.

the microscopic eyepiece. A correct upright posture is maintained, thereby reducing operator fatigue and strain. Far more detailed examination of the root apex is possible, allowing anatomic features such as isthmuses, accessory canals, fracture cracks, and crazing



Fig. 18.9 Comparison of microsurgeal mirrors to facilitate visualization of the root canal anatomy with a conventional dental mirror (left).



Fig. 18.10 Comparison of microsurgeal blades with conventional surgical blade.

to be identified. Angled ultrasonic instruments can be used to prepare the retrograde cavity with the aid of micromirrors. This allows visualization of the whole root apex in order to check that all infected debris and old root canal obturation materials have been removed. If failure to shape, clean, and fill the root canal system is acknowledged as the main reason for failure of *de novo* orthograde root canal treatment, exactly the same concerns apply to the surgical approach.

Root-end filling materials

As well as developments in microsurgery techniques, significant advances have been made in root-end filling materials. First described by Torabinejad *et al.* in 1995,⁴³ MTA (Fig. 18.12) is now recognized to have significantly better properties than any other material for this purpose. Unlike all of the other traditionally used root-end filling materials (amalgam, zinc oxide-eugenol cements, glass ionomer cements) the set of the freshly placed material is not affected by the presence of moisture, including blood. It is less cytotoxic and more tissue compatible than previously used materials. Reports from animal studies demonstrate less inflammation and greater cementum deposition over set MTA compared with other materials within days of surgery. Furthermore, scanning electron microscopy (SEM) images reveal cementoblasts adhering to the material. The only reported disadvantage of MTA is the slight difficulty in handling and placement. This has been overcome to a certain extent with the introduction of a white MTA material which in some respects has superseded the original gray powder. When properly mixed, it has a creamy consistency and is less difficult to place when using appropriate instruments.

Local anesthesia, medication, and hemostasis

The choice of local anesthetic agent for periradicular surgery is related to three distinct surgical requirements: anesthesia during the surgical procedure, hemostasis, and postoperative pain control. Clinical trials have not been able to elucidate any significant clinical differences between the various anesthetic agents currently available for dental use. However,



Fig 18.11 Microsurgical suture forceps and 5/0 suture in comparison to standard suture forceps and 4/0 suture (left). Microsurgical suturing with papillae preservation (right).



Fig. 18.12 Mineral trioxide aggregate (MTA) is available in three presentations: ProRoot original gray powder, the more recent ProRoot white powder (both Dentsply UK), and the white MTA Angelus (QED, Peterborough).

there is widely reported anecdotal evidence that the most profound anesthesia with rapid onset is achieved when using a local infiltration of articaine with 1:100 000 adrenaline (epinephrine). However, it has been suggested that this solution may not be suitable for an inferior dental nerve block due to the potential increased risk of paresthesia and nerve damage. It is recommended that for lower teeth an inferior dental nerve block is administered using lidocaine or other proprietary agent, with an additional buccal local infiltration of articaine. Hemostasis is greatly facilitated with an additional infiltration of lidocaine containing 1:50 000 adrenaline.

Postoperative pain control may be enhanced by the administration of 0.5% bupivacaine immediately following the surgical procedure. Although not as affective as other agents at achieving surgical anesthesia, this long-acting agent acts by reducing central sensitization following PRS. Clinical trials suggest that bupivacaine may reduce postoperative pain for up to 3 days. Non-steroidal anti-inflammatory drugs (NSAIDs) are also recommended postoperatively for pain control unless there is a medical contraindication to their use. There is no indication for the use of prophylactic antibiotics when undertaking PRS procedures other than for treating patients with a specific systemic medical condition.⁵⁹

Surgical procedures

Flap design and soft tissue management – introduction

Before a decision is made on the design of the surgical flap in any patient, careful assessment of the periodontal tissues and restoration margins is essential. Inflamed, swollen gingivae with associated bleeding on probing indicate the presence of plaque-related microbial disease. This should be addressed before treatment or postoperative outcomes may be compromised. Cause-related periodontal therapy may be supplemented with a 0.2% chlorhexidine gluconate mouthrinse twice daily (after brushing) for 1 week before and 1 week following the surgical procedure. This will significantly reduce the accumulation of plaque and enhance tissue healing.

The natural retraction of the gingival tissues following surgery with a full-thickness sulcular flap may expose previously placed subgingival restoration margins.⁶⁰ The patient should therefore be informed of this possibility in advance of the surgical procedure. If this is an unacceptable esthetic risk then an alternative flap design will be required and may involve papillae preservation. Traditionally, three mucosal flap designs have been described: semilunar, rectangular, and submarginal. Only the latter two will be considered within this chapter. The semilunar flap, where a curved incision is made over the root apices in the alveolar tissues, can no longer be advocated in endodontic surgery for a number of reasons. The semilunar incision frequently lies over the bony defect following the surgical procedure; the incision severs an excessively large number of blood vessels which run vertically through the tissues and the natural elastic and muscle fibers tend to stretch the wound margins making approximation difficult, with delayed healing and impaired esthetics.

The rectangular flap

The rectangular flap (Fig. 18.13) comprises two vertical relieving incisions with a horizontal marginal incision. Conversely, a triangular flap has only one relieving incision. However, this creates difficulty in accessing the apical tissues without applying excessive pressure to the flap which may result in tearing of the tissues. Therefore the additional relieving incision may be favored to create a rectangular flap. Although easily repositioned, the disadvantage of this flap design is the potential for gingival recession and exposure of restoration margins. It is important that the relieving incisions are placed on to sound bone.

The submarginal flap

Also referred to as the Ochsenbein-Luebke flap, the vertical relieving incisions of the rectangular flap are commenced approximately 2 mm from the gingival sulcus and the horizontal incision is made in the attached gingivae, in a scalloped pattern to follow the natural tissue contours (Fig. 18.14). This can only be



Fig. 18.13 Outline of rectangular flap.



Fig. 18.14 Outline of submarginal flap.

used where there is a good width of attached gingiva to preserve the blood supply and integrity of the remaining tissue. Any failure here and resultant necrosis of the residual attached gingiva will have catastrophic esthetic outcomes.

Microsurgical approaches

The SOM has facilitated the use of microsurgical scalpel blades and other microsurgical techniques. The potential for micro-suturing has permitted procedures designed to preserve the integrity of the interdental papilla with resultant improved postsurgical esthetics.⁶⁰ This procedure of papilla preservation is difficult and requires a good level of understanding and surgical skill (Fig. 18.11). An initial shallow incision is made at right angles at the base of the papilla through the epithelium only. The line is then retraced with the blade at an angle vertically towards the bone, leaving the papilla intact with a split-thickness incision at its base. Normal full-thickness vertical relieving incisions are then made. Micro-surgical sutures will result in an excellent esthetic result if the procedures are followed precisely and the tissues kept moist during the surgery, especially if hemostatic agents are used. However, if the incisions are made at an incorrect angle or the significance of the procedure is not fully understood, loss of the papilla may result in a worse appearance than with a conventional approach. There is early clinical evidence⁶⁰ that such papilla-based flaps achieve the best postoperative esthetic result.



Fig. 18.15 Very fine surgical elevators designed for delicately raising mucosal flaps without causing any damage to the soft tissues (left). Microsurgical instruments for curetting the underlying pathological defect (right).

Flap reflection

Whichever flap design is selected, care should be taken to retract the entire mucoperiosteal tissue without damage. Very fine surgical elevators designed for delicately raising mucosal flaps without causing any damage to the soft tissues are shown in Fig. 18.15. Starting at a vertical relieving incision, the periosteum and mucosal tissue should be elevated as one piece, bearing in mind that the surface of the underlying cortical plate may be uneven. The flap should be elevated towards the attached gingivae, with microsurgical instruments being used for gentle elevation of the gingival and papillary tissues. The flap should extend well above the anticipated position of the root apex to facilitate unhindered access to the surgical site without stretching or traumatizing the retracted tissues. Tissue retraction should follow normal surgical principles with no undue pressure, tearing or other damage to the tissues. To avoid impeding direct vision with the SOM, the vertical relieving incision(s) are made longer than in conventional apical surgery. Hard and soft tissues should be kept moist by regular irrigation with sterile normal saline throughout the operation.

Hard tissue management

Access to the root apex is frequently facilitated through a perforation of the cortical plate created by a sinus draining from the periradicular lesion. Once the flap has been raised sufficiently, the defect is identified and used as a landmark for bone removal. In those cases where such a perforation has not occurred, location can be more difficult. Clues may be found in that root tissue is harder than bone, cannot be marked with a probe, is more yellow and does not bleed. Occasionally, a methylene blue dye may be used to outline the root surface as it will stain the periodontal ligament. Careful assessment of the root length from preoperative radiographs prior to bone removal will help in locating the root apex.

Extreme care must be taken when removing bone to identify the root apex, and when extending an exposed crypt so as not to generate heat which may rapidly cause irreversible damage to the bone. Firstly, adequate and efficient coolant with sterile normal saline must be applied throughout the cutting proce-

ture. Secondly, a round bur is preferred. A fissure bur may penetrate bone which will isolate the tip from the coolant. The resultant rapid heat generation will cause, at the very least, inflammation and delayed healing. A similar adverse incident may occur if a diamond bur is used, as the grit will trap bone particles and increase frictional heat generation. Furthermore, a blunt bur will generate more heat as greater pressure is applied to cut through bone. A new surgical bur is therefore recommended for each procedure. With the improved vision afforded by the SOM, the use of micromirrors and fine ultrasonic tips, bone removal can be kept to a minimum.

A thorough and detailed knowledge of the local anatomy is essential. PRS on maxillary anterior teeth may be relatively straightforward but on mandibular anterior teeth may be far more difficult due to the shallow labial sulcus and lingual inclination of the roots. Maxillary posterior teeth present additional problems in their proximity to the floor of the sinus, although the buccal roots usually lie very close to the cortical plate and frequently perforate this. Access to palatal roots will necessitate a significantly larger osteotomy. In addition, the mandibular posterior teeth are generally covered with a thick cortical plate making access very difficult. Reports are published of failure following PRS within these anatomic regions due to necrosis of the bone. This may be attributed to the increased heat generated during the removal of significant quantities of bone.

Periradicular curettage

Periapical periodontitis is the body's natural immune response to the inflammatory mediators and infective agents within and around the root apex. Thus, no matter how thoroughly the granulation tissue that forms the PRD lesion is removed from the surgical site, unless the actual source of infection within the root canal is addressed by root resection and retrograde restoration, the surgery will likely fail. The presence of many different foreign body materials within PRD lesions has been reported by Nair.⁶² These include, of particular interest, remnants of

paper points caused by inadequate measurement during canal preparation procedures, and vegetable material entering the exposed root canal when a tooth is "left open for drainage". This procedure of "open drainage" is no longer recommended.

Every effort should be made to dissect out the granulation tissue cleanly and without damage so that it may be placed immediately in a container of formalin and sent for a pathological report. This may be accomplished using two separate instruments. A sharp curette or spoon-shaped excavator is used to carefully dissect the PRD lesion from the bony wall of the crypt (Fig. 18.15). However, this instrument is not as effective on the root surface where a sharp periodontal scaling instrument is preferred. The surgical micromirror and SOM will greatly assist when working behind the root to ensure that all remnants are released before attempting to transfer the lesion to the histological receptacle. A microscopic examination should be made to identify any remaining granulation tags. However, caution should be taken when these are attached to anatomic features such as neurovascular bundles or sinus linings.

Occasionally, the patient may complain of pain during this procedure and supplementary anesthesia may be required. Should hemostasis be inadequate, the crypt may be packed with cotton pellets impregnated with epinephrine or ferric sulfate and pressure applied for a few minutes. Both these agents provide satisfactory surgical hemostasis without any adverse effects (Fig. 18.16).⁶² Careful examination with the SOM is essential to ensure that all traces of these materials, in particular fibers from cotton pellets, have been removed before the wound is closed.

Root-end resection

The main reason for a surgical approach to an endodontic problem is to remove extraradicular bacteria and other contaminants causing clinical symptoms. However, the anatomy of the root apex is complicated, with multiple portals of exit and accessory canals. Indeed, the presence of apical ramifications may be a major contributing factor towards the occurrence of



Fig. 18.16 Hemostasis may be aided with the placement of hemostatic agents within the surgical area, for example Surgicel.

refractory PRD.⁶³ Microbial biofilms form on the root apex and these penetrate the dentinal tubules. Thus, however effective the apical curettage has been, there is a potential for recontamination, maintained inflammation, and ultimately surgical failure unless the root apex is removed.

It is generally accepted that an apical resection of 3mm will remove the majority, if not all, of these potential problem areas. This resection will additionally permit thorough inspection of the root canal, preparation of a cavity to encompass the canal shape and placement of a retrograde seal. As illustrated in Figs 18.6 and 18.17, the angle of resection should be as close to horizontal as possible (i.e. at right angles to the long axis of the tooth). This exposes a minimal number of dentine tubules, thereby reducing apical leakage and providing the best potential for healing.⁶⁴



Fig. 18.17 Large apical lesion with root apices resected at 90°. Preparation of root canal with ultrasonic tips and use of micro-mirror prior to placement of retrograde root canal filling.

The smoothest possible cut should be made, ideally using a straight fissure bur in a slow-speed handpiece. Cross-cut fissure and diamond burs tend to create a rough surface that may harbor debris, particularly remnants of the root filling material, thereby impairing healing. Ideally, the cut surface should be smoothed with a tungsten carbide or micro-fine diamond, keeping the bur moving across the root surface and not cutting backwards against the rotation of the bur. Of course, if a high-speed motor is used it must be of a surgical design with appropriate air venting. Conventional air rotor drills must be avoided in periradicular surgical procedures so as to reduce the possibility of producing pyemia or emphysema.

Root-end preparation

The preparation of the retrograde cavity has two primary objectives. Firstly, it must thoroughly remove all traces of the previous failed and contaminated root canal filling material. Secondly, it must shape the apical aspect of the root canal system in order to permit placement of a hermetic seal. This will help to prevent the exit of further microbial contaminants. Once again, good visual inspection of the root apex using the SOM, good illumination, and the use of surgical micromirrors is essential. Traditionally a micro-handpiece with a round bur was used to undertake root-end preparation. However, everyone who has performed this procedure will testify to the difficulty of preparing a cavity of at least 3mm depth that encompasses the whole of the root canal including any isthmuses. The currently preferred method involves the use of specially designed ultrasonic tips in a piezoelectric ultrasonic handpiece (Figs 18.18 and 18.19).

Ultrasonic preparation of the root end produces cleaner, well centered, and more conservative root-end cavities than conventional rotary instrumenta-

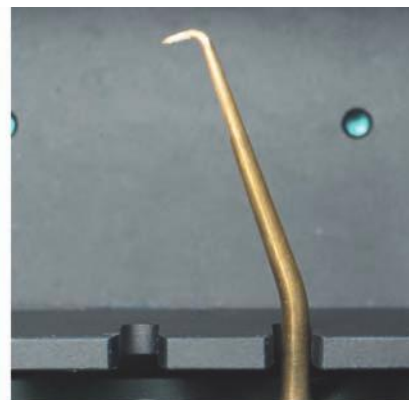


Fig. 18.18 Ultrasonic unit and close-up of ultrasonic tip used for root-end preparation.

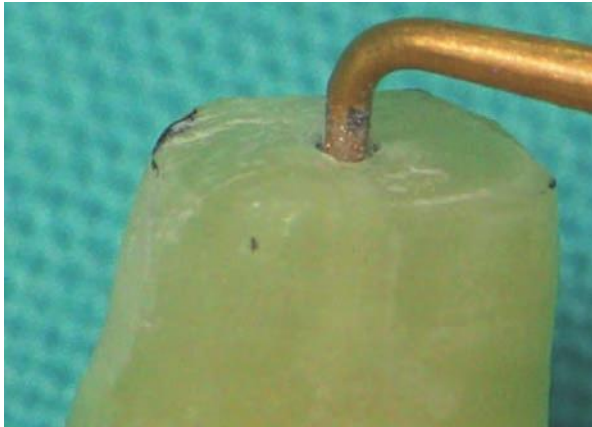


Fig. 18.19 Ultrasonic cavity preparation of 3–5 mm of the root canal for placement of the retrograde root canal filling.

tion.⁶⁵ Various inclinations may be used to access the root apex and explore the root canal. Diamond coated tips appear to be the most effective and are more efficient in the removal of old gutta-percha. However, stainless steel tips may leave a cleaner surface with less smear layer. In the hands of an inexperienced operator, diamond tips may be more aggressive and remove excessive material. Therefore, a very gentle touch is essential using light brush strokes to plane and shape the canal. Sufficient coolant spray must be used to remove debris and prevent overheating without obscuring vision. Overheating has been a cause of concern in contributing towards micro-cracks at the root apex. Careful technique will prevent this, particularly in avoiding excessively high power settings.^{66,67} Nevertheless, even at lower power settings cracks and chipping may occur.⁶⁸

The instruments are used slowly until the desired cavity has been created, at least 3 mm deep, with clean walls uncontaminated by the old root filling material. The remaining gutta-percha at the base of the preparation should be carefully packed with a microsurgical plugger. Where two canals are present in one root it is advisable to create an isthmus between them which is filled to prevent any potential leakage. The apical preparation can then be carefully inspected with the SOM and micromirrors. The use of ultrasonics for root-end preparation has been demonstrated to improve surgical outcomes in comparison to traditional techniques.⁶⁹

Restoration of the retrograde cavity

Before restoring the root-end preparation, there must be adequate hemostasis within the surgical site. This can be achieved by placing dry impregnated cotton wool balls into the bony crypt or a hemostatic material, for example Surgicel (Johnson & Johnson, UK, Fig. 18.16). These agents also help to act as a barrier to prevent the accumulation of excess retrograde filling material around the root apex tissues. The root-end preparation can then be dried with either paper points or the use of a micro-irrigating syringe.

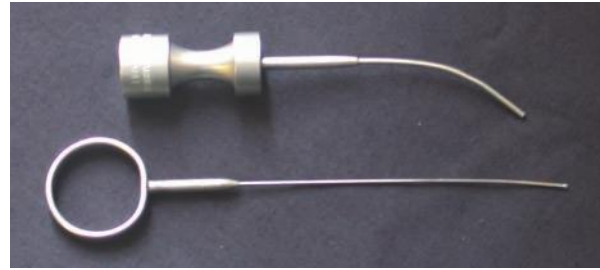


Fig. 18.20 Instrument for placement of MTA.

The restorative material selected for the root-end filling must be radiopaque, easy to manipulate, and exhibit two principal characteristics. Firstly, it must provide an effective permanent seal to prevent leakage from the root canal system of any microorganisms or their by-products. As such, it must be stable and insoluble. Secondly, the material should be biocompatible, ideally promoting regeneration of the surrounding cementum and bone directly on to its surface. Although many materials have been advocated for this purpose in the past, only two are now recommended as retrograde filling materials. These are MTA or reinforced zinc oxide–eugenol cements.

Mineral trioxide aggregate

As indicated earlier, MTA is now widely accepted as the preferred restorative material for retrograde root canal fillings. MTA was derived from Portland cement from which various oxides were removed to improve its biocompatibility and handling characteristics. The first MTA product introduced to the clinics was a gray material that was mixed to a gritty or sandy consistency that was rather difficult to manipulate. The latest version, white MTA (ProRoot MTA, Dentsply, UK), can be mixed to a smooth, creamy paste that is easier to place. It can be carried to the cavity in a specially designed carrier gun (Fig. 18.20a); it can be rolled into a stiff cylinder using the Lee Block (Fig. 18.20a); or carefully manipulated on a flat plastic type dental instrument directly into the cavity. Once in place, it is gently condensed. The MTA retrograde filling is subsequently finished by gentle wiping of the apical preparation with a moistened cotton wool pellet.

MTA is hydrophilic and requires moisture to set. When used within the root canal via an orthograde approach the dry conditions require a moist cotton pledget to be placed for up to 4 hours' setting time. This cotton pledget is removed at a subsequent appointment. In a retrograde cavity, absolute dryness is neither possible nor desirable, although care must be taken not to wash the material from the cavity during closure of the surgical site. It is preferable to place 3–4 mm of MTA at the root-end to provide a satisfactory seal.^{70,71} An alternative presentation, MTA Angelus, has a much faster setting time. MTA has been widely studied and is reported to be more biocompatible than any other retrograde restorative

material, inducing cementogenesis and bone deposition with little if any resultant inflammation.^{72,73}

Reinforced zinc oxide–eugenol cement

There are primarily two reinforced zinc oxide–eugenol cement materials routinely used for root-end filling (Fig. 18.21). Intermediate Restorative Material (IRM[®], Dentsply, UK) is a zinc oxide–eugenol cement with approximately 20% polymethacrylate in the powder. The second material, SuperEBA[™] (HJ Bosworth, Illinois, USA), is an alumina-fortified material in which the liquid comprises 32% eugenol and 68% ethoxy benzoic acid (EBA). Both materials are mixed to a stiff paste and packed firmly into the prepared cavity. The operating field should be dry and clean. Some clinicians have recommended cleaning the root-end cavity with citric acid or EDTA prior to restoration but as yet there is little published evidence in favor or against this procedure.

These reinforced cements have superior properties in comparison to conventional zinc oxide–eugenol cements or amalgam. They exhibit lower solubility, less leakage, and greater biocompatibility.⁷⁴ The eugenol in these materials has a localized effect upon the surrounding tissues that may cause inflammation and slightly delayed healing. However, clinical studies have produced very satisfactory results that are comparable with outcomes when MTA is used as the root-end filling material.⁷⁵ Although reinforced zinc oxide–eugenol materials appear to offer satisfactory clinical success, unlike MTA they do not stimulate any dental hard tissue or cementum regeneration.^{76,77}

Other materials

There have been several publications investigating the potential for dental adhesives such as glass ionomer cements, compomers, and composite resins, to seal resected root apices. Although there are reports that they can be effectively used in periradicular surgery,⁷⁸ there remain continuing concerns over moisture contamination preventing an adequate seal.⁷⁹ Trope *et al.*,⁸⁰ reporting research using a range of

restorative materials for retrograde obturation, expressed deep concern that even when a dry operating field was possible some moisture contamination was inevitable, compromising the seal and the outcome of the procedure. A comprehensive review of the subject⁸¹ of retrograde filling materials concludes, “The material to choose will depend on prevailing clinical conditions. For example, if there are difficulties in maintaining a dry surgical field, even if a material has excellent biological characteristics, it is unsuitable if it is sensitive to moisture.” Amalgam is no longer recommended as a material for root-end filling.

Guided tissue regeneration

There is ongoing debate as to the requirement or usefulness of guided tissue regeneration (GTR) in improving outcomes of PRS involving apicectomy procedures. From recent evidence, it would appear that the use of a GTR membrane with or without bone graft material has no additional advantage on the surgical outcome in the treatment of patients exhibiting large PRD lesions.^{82,83} Furthermore, the experimental use of bone-inducing substances, such as bone morphogenic proteins,⁸⁴ or clinical use of calcium sulfate,⁸⁵ has not displayed any enhanced benefits over natural healing. The one exception for this is in the case of a through-and-through lesion where a bone substitute and GTR may help to avoid healing of the defect site with granulation tissue (Fig. 18.22).

Wound closure

If MTA has been used as the root-end filling material, it must be carefully protected during the normal surgical practice of thorough irrigation, cleaning, and inspection of the surgical site before closure. Once the site has been inspected and any remaining foreign bodies removed, gentle bleeding may be encouraged by simple curettage to ensure a blood clot is present in the wound. The flap should be repositioned and compressed with a damp, sterile gauze swab. Once suturing is complete, similar firm compression should be applied for at least 5 minutes to ensure initial clot formation is undisturbed. To avoid wicking of bacteria into the operation site, silk sutures are not recommended for periradicular surgery. Synthetic or coated materials are preferred. Resorbable suture materials are not preferred unless the patient is unable to return for suture removal, as the rate of resorption is too variable. Interrupted or continuous sutures may be placed according to the operator’s preference. The disadvantage of the latter is that should one suture pull free from the tissue the entire flap may be loosened. Ideally the corners of the flap are initially sutured before the central part is correctly positioned and sutured. Following compression and confirmation of hemostasis, bupivacaine local anesthetic may be administered, taking great care to ensure that the flap is not disturbed in the process.



Fig. 18.21 Example of root-end filling material (IRM, Dentsply, UK).

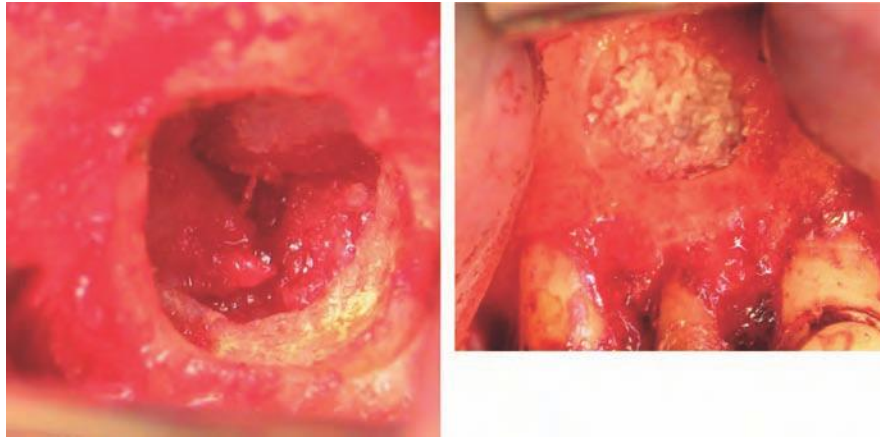


Fig. 18.22 Extensive periradicular through-and-through defect associated with tooth 22. The soft tissue of the palate can be seen through the defect (left). Placement of a barrier membrane and bone substitute to prevent scar formation and permit guided bone regeneration at the site following surgical root resection and soft tissue curettage (right).

Postsurgical management

An example of a postoperative instruction leaflet is given in Fig. 18.23. A cold compress may be used by the patient to help reduce postoperative swelling.⁸⁶ The patient should be advised of the possible development of a limited area of ecchymosis and instructed to continue regular use of chlorhexidine gluconate mouthwashes until suture removal. Sutures should be removed after 3 or 4 days by which time periodontal fibres at the gingival margin will have reattached.⁸⁷ A postoperative radiograph should be exposed at the suture removal appointment. Good clinical governance would involve exposing a further radiograph after 1 year to confirm healing and facilitate audit of clinical outcomes. The tooth should be monitored radiographically for up to 4 years.

Following PRS procedures, the majority of patients will experience moderate pain. However, this is of a relatively short duration and occurs within the ensuing 48 hours. Postoperative pain is more likely to occur when preoperatively there is pain, poor oral hygiene, and a smoking habit.^{88,89} For pain control, non-prescription NSAIDs such as ibuprofen, unless medically contraindicated, may be taken before or immediately following surgery and continued at regular intervals for 3 days.⁹⁰ The use of such drugs on an *ad hoc* basis renders them far less effective. Severe postoperative pain is unusual, especially if long-acting bupivacaine local anesthetic has been administered. However, some patients may require codeine or similar medicament. The prophylactic administration of antibiotics is contraindicated unless the patient has a specific underlying medical condition indicating their use.

Periradicular surgery outcomes

The success of PRS has been reported to vary between 25% and 99%.⁸⁶ A recent systematic review of PRS outcomes determined that the weighted average of success was 64%, whilst the outcome from 25.7% of

Endodontic surgery

Postoperative advice sheet

1. To reduce postoperative swelling an ice-pack should be applied to your face adjacent to the site of surgery for 15 minutes in each half hour for the 6 hours after the operation.
2. Minor bleeding may occur from the site of the operation which will color your saliva. Should bleeding appear to be excessive apply pressure to the area with a moist clean cotton cloth or gauze.
3. Anti-inflammatory painkillers, such as ibuprofen, should be taken regularly for the next 2–3 days. If you are unable to take these for medical reasons an alternative medication will be prescribed.
4. Continue using the chlorhexidine mouthwash twice daily until the sutures have been removed. However, only rinse gently for the first day after surgery.
5. Follow your usual oral hygiene habits with brushes, floss, and other aids, but avoid cleaning the surgical site and only brush the adjacent teeth gently. Do not lift your lip to inspect the wound or this may delay healing.
6. Try to eat only soft food for a few days and avoid chewing anything hard with the teeth involved in the surgery.
7. Do not smoke until the sutures have been removed.
8. Please telephone the surgery if you have any concerns, in particular if bleeding persists; you suffer pain that is not controlled by the analgesics; you develop signs of infection such as a raised temperature or swollen glands.

Fig. 18.23 An example of a postoperative instruction leaflet to be given to the patient.

cases remained uncertain and 15.7% of surgical cases were unsuccessful.⁹¹ Periradicular resurgery has been demonstrated to have a significantly greater failure rate than a first surgical procedure.⁹² Repeat surgery is reported to be associated with a weighted average healing rate of only 35.7% and a failure of 38%. Furthermore, the success rate of NSRCT retreatment of failed periradicular resurgery cases is lower than NSRCT retreatment of failed endodontically managed cases.⁹³ Nevertheless, periradicular resurgery remains a valid alternative to dental extraction.⁹²

Clinical success or failure appears to be associated with the operator and the types of materials used within the surgical procedure.^{91,94} As with successful outcome following NSRCT, surgical success is directly related to the size of the periradicular lesion.^{94,95} Lower success rates are also associated with inadequate root filling length⁹⁴ and pain at initial examination.⁹⁶ Maddalone and Gagliani⁹⁷ have demonstrated that periradicular microsurgical techniques can achieve success rates of 92.5% over a 3-year observation period. It is also established that the use of modern materials, magnification, improved surgical techniques, and better postgraduate training results in improved surgical success rates over traditional PRS techniques.^{37,98} Furthermore, modern surgical techniques result in less postoperative pain compared with conventional surgical techniques but this may be counteracted with more difficulties immediately postoperatively in mouth opening, the ability to speak, and mastication.⁹⁹

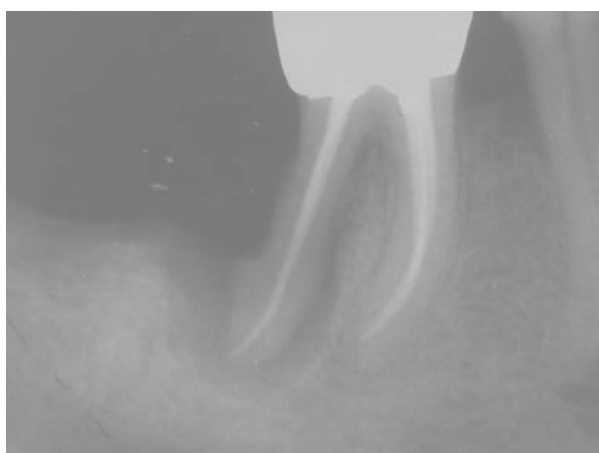
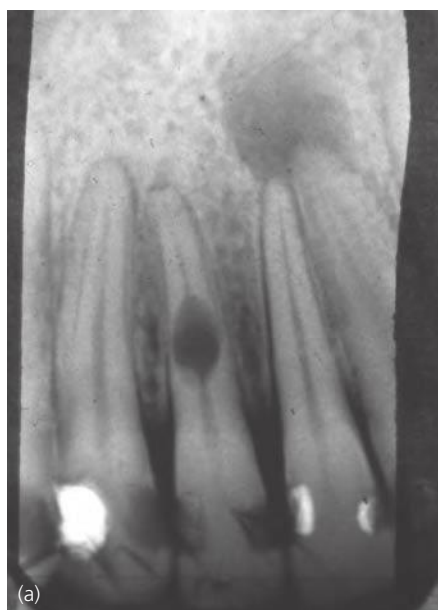


Fig. 18.24 A radiograph showing a persistent lesion on the distal root of a lower molar that has not responded to orthograde retreatment. This case may be suitable for hemisection.



(a)

Other surgical endodontic procedures

Hemisection or root resection

Occasionally, root canal treatment in a multirooted tooth may be successful in one root only. This scenario is observed in Fig. 18.24. If retreatment of the affected root is not possible it may be surgically removed. However, even following careful examination of the preoperative radiograph, the root morphology may be unclear. Unless the roots are adequately separated and not fused or joined together, removal of an individual root may prove impossible. If a suture needle is threaded through the furcation and the suture tied around the crown, the separation of the roots is confirmed and the path of resection clearly marked. Once the tooth has been sectioned with a high-speed bur the diseased root may be elevated. The remaining crown may be restored as it stands or, if sufficient periodontal bone support remains, the root may occasionally be used as a bridge abutment.

Perforation repair

Perforation of the root surface may occur as a result of internal root canal resorption, by iatrogenic damage during root canal instrumentation or during post-space preparation (Fig. 18.25). The prognosis for the repair of a perforation will depend upon its position on the root, evidence of chronic infection, and presence of any periodontal breakdown and epithelial downgrowth. In endodontic retreatment cases, two factors appear to significantly impact upon successful treatment outcomes: (1) the presence of a PRD lesion; (2) the presence of a preoperative perforation.¹⁰⁰

In the first instance, repair of root perforations should normally be attempted by an orthograde approach through a conventional access cavity and



(b)

Fig. 18.25 Perforations of the root surface due to (a) internal resorption and (b) iatrogenic damage during post space preparation.

the root canal. Once again, use of the SOM facilitates detailed examination and placement of MTA. If internal examination reveals that repair is not feasible via this approach then careful note should be made of the position of the perforation. Root perforations that are labial to a line drawn mesio-distally through the tooth may be amenable to a surgical procedure. Those that lie palatal to this line will require elevation of a palatal flap and access may be severely compromised. If an orthograde approach is not considered practical, careful use of parallax radiographs, or preferably 3D CBCT, will be necessary to accurately determine the position of the perforation.

A mucoperiosteal flap should be raised as previously described. Once identified, the defect is prepared and obturated as for root-end surgery. MTA is again the material of choice for restoration of the defect. However, if a coronal lesion communicates with the oral cavity and there is the possibility of the MTA filling washing out before it has set, glass ionomer cement may be the material of choice, providing that effective isolation and moisture control are achievable. Clinical results of compromised teeth exhibiting root perforations repaired with MTA have demonstrated good outcomes in enhancing the prognosis of perforated teeth that may otherwise have been extracted (Fig. 18.26).¹⁰¹

Intentional replantation

It is sometimes possible for a tooth with a persistent PRD lesion that is not amenable to surgical repair to be extracted and intentionally replanted.^{102,103} Careful assessment of the root pattern is necessary to ensure

atraumatic removal without causing significant damage to the periodontal ligament and root surface. Prior to tooth removal, all instruments and materials required for root-end resection and restoration should be laid out to facilitate rapid treatment. The root should be carefully held with gauze moistened with sterile saline and not allowed to become dry.

As soon as the apical procedure is complete (as described for apicectomy) the tooth is replanted, the bone gently digitally compressed and the patient instructed to occlude on gauze to maintain the tooth in position as hemostasis occurs. It may be necessary to adjust the occlusion for comfort and to avoid occlusal trauma. The patient should be advised to take a soft diet, in particular avoiding sticky foods, for at least 1 week. Intentional replantation may be considered a predictable treatment when performed with care in cases where conventional periradicular surgery is not feasible.¹⁰⁴ The prognosis appears to be most related to an atraumatic extraction and protection of the periodontal ligament with a minimum treatment time.

Informed consent

Informed consent must be obtained by the clinician responsible for the provision and arrangement of treatment from all patients for all clinical procedures. She or he should have undertaken detailed audit of their treatment outcomes in order to provide a confident prognosis of the outcome for each surgical procedure. Naturally, this may include reference to general studies reported in this chapter. However,

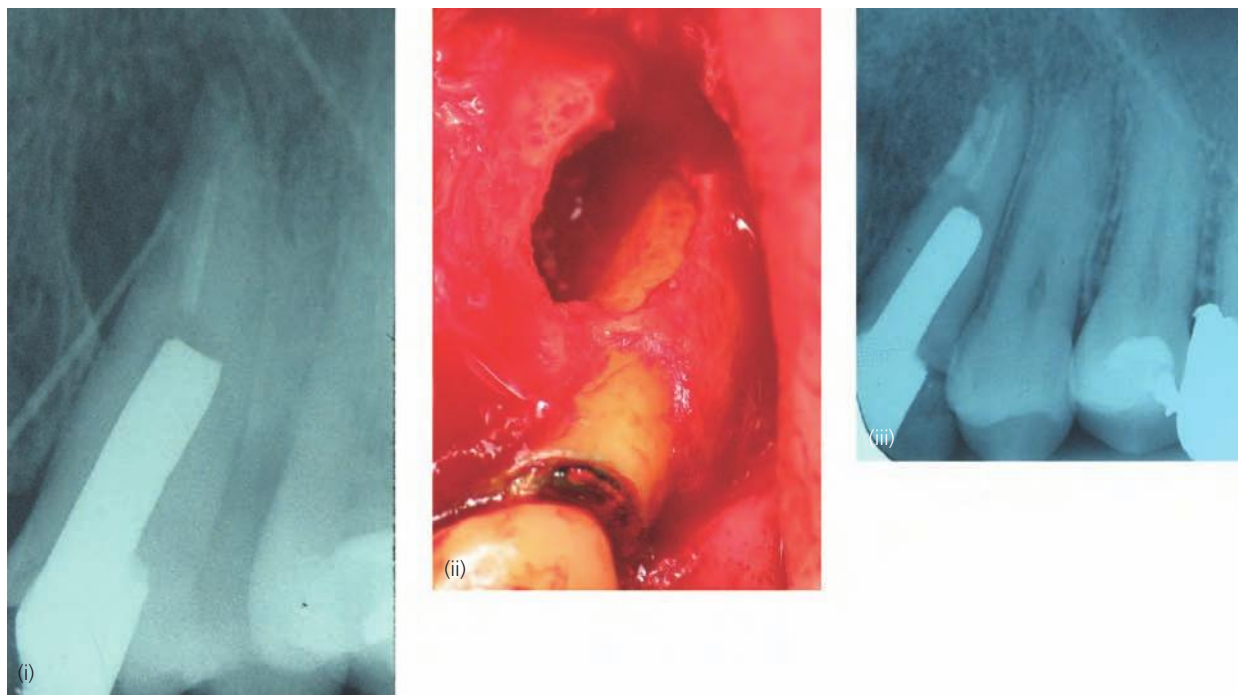


Fig. 18.26 Repair of a root perforation using MTA. (i) Preoperative radiograph with a gutta-percha point placed in the buccal draining sinus. (ii) A large defect is evident upon raising a flap. (iii) One-year follow-up radiograph of the healed site with MTA placed at the perforation.

the patient will understandably be most concerned with outcomes related to their own clinician's procedures and results.

A detailed knowledge and understanding of endodontic principles is therefore essential when advising the patient on the choice between orthograde retreatment or a surgical approach when a root filling has failed. Should the clinician fail to obtain informed consent and treatment not be successful, the patient may seek expert endodontic opinion as to the alternative treatments available, their indications, and their respective prognoses. Familiarity with current expert opinion and practice, particularly as taught in clinical centers of excellence, is essential for all seeking to practice safely within this field.

Endodontic surgery vs endosseous implants

The dental literature has recently seen an increase in the number of published papers on this subject. In particular, some clinicians appear to consign the entire concept of periradicular surgery to history, claiming that extraction of the diseased tooth and placement of a dental implant has a better prognosis and should be the treatment of choice. Amongst these reports are two quite conflicting papers in the same issue of the *Journal of Oral and Maxillofacial Surgery*. In the first, Ruskin *et al.*¹⁰⁵ concluded that, "It is thus possible to consider early removal of teeth and placement of implants and implant based restorations as a favourable treatment option compared with the majority of endodontically treated teeth." Conversely, the authors of this chapter consider that every reasonable effort should be made to retain the natural tooth with its periodontal support, function, and proprioception, and that an implant-retained restoration should be seen as the last possible treatment option. A similar opinion is held by Thomas von Arx,¹⁰⁶ the author of the second article in the *Journal of Oral and Maxillofacial Surgery*. He discusses how microsurgical techniques, as described in this text, have contributed to higher success rates, and states that "whenever possible, teeth should be salvaged". The controversy between endodontic treatment or implant placement will no doubt continue until high-quality research evidence, which is sadly lacking at present, is published. Those interested in taking this debate further should refer to two excellent review papers.^{107,108} Both papers have similar conclusions, that the outdated concept that periradicular surgery is the "junior partner" in revision of negative endodontic treatment outcomes needs to be reconsidered.

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Chapter 19

Preprosthetic and Oral Soft Tissue Surgery

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This chapter provides a comprehensive overview of various surgical procedures to improve soft tissue architecture and anatomy to improve oral function and optimize prosthetic rehabilitation in various ways, e.g. preparing a denture-bearing area and improving soft tissue conditions prior to dental implant treatment. Basic principles and recent techniques are presented.

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Overview

Background

Optimal oral rehabilitation of patients presenting with functional and esthetic problems is attainable by appropriate preprosthetic soft tissue surgical procedures. A variety of surgical procedures can be planned to correct or to improve soft tissue architecture and anatomy as well as to gain desirable soft tissue features either as a denture-bearing area over alveolar bone or around dental fixtures.

Irregular and frail soft tissues, both in fully dentate and edentulous patients, need to be diagnosed in advance to avoid undesirable changes, particularly in patients who are planned to receive dental implants. Surgical management of oral soft tissues is generally carried out for esthetic, functional or curative reasons. Oral soft tissue abnormalities may be due to acquired, pathological, traumatic, congenital or developmental factors.

Progressive alveolar bone loss following tooth extraction also has effects on the covering and surrounding oral soft tissues which undergo clinical changes. The changes may necessitate modification, alteration or replacement of oral soft tissues either to host or to facilitate optimal prosthetic rehabilitation.¹

Successful surgical management of soft tissue abnormalities requires thorough anatomic and functional assessment of the overall region in order to achieve appropriate outcome and fewer denture or implant-related complaints.² The appearance, texture, histological characteristics, and quantity of oral soft tissues should be carefully assessed. Deficient attached epithelium needs to be carefully evaluated to enhance the success of restoration, which may be a removable denture or an implant-supported prosthesis.

Keratinized epithelium may need to be broadened and improved through various procedures or sometimes may be surgically modified for enhanced structural benefit.³ The anatomic zones, transitions, and features of oral soft tissues have major roles in oral

rehabilitation by all means. Current trends and oral rehabilitation philosophies have changed immensely over recent years but mucosa-supported removable dentures in edentulous cases may still be the treatment of choice for several reasons.

Among these factors, individual financial status or health system regulations are the most notable ones. The quality and quantity of the denture-bearing mucosa is a major factor for favorable retention and stabilization characteristics. Mucosa-supported rehabilitation planning relies on stable and sound soft tissues covering alveolar bone for maximal retention and stabilization for improved prognosis. Neighboring soft tissues buccally, labially or lingually also have considerable influence on the success of prosthetic construction. Non-resilient, stiff soft tissues, and functional sulcus loss, may have consequential effects on prosthetic replacements.

The surface epithelium characteristics, in terms of both quantity and quality, dictate acceptable esthetics, function, and hygiene maintenance, and successful oral rehabilitation. Deviation from normal may result from either primary or secondary causes. Primarily the surface properties, topography, texture, and structural features of oral mucosa may complicate a desirable functional result or even inhibit prosthetic rehabilitation. In these cases a surgical plan may be needed, ranging from simple revision of a localized area to an extensive reconstruction of soft tissues on and around alveolar bone.

It is mandatory to be aware of the importance of the presenting clinical features of soft tissues so as to plan and/or to predict the rehabilitation outcome. The soft tissues should be as high quality as possible to improve the end result of prosthetic rehabilitation. Therefore presenting clinical properties should be known right from the start in order to minimize or avoid patient dissatisfaction. Recognizing and differentiating pathological conditions is also of particular importance so that a proper treatment protocol can be established.

Soft tissues may need to be converted or reconstructed by tissue transfers, where staged interventions may sometimes be needed for additional adjustments or improvements so that a better prosthetic device can be fitted. Where tissues from distant sites are brought into the mouth, original features may still be seen. Skin appendages may continue to exist for some time, although, in the majority of patients who received free or distant tissue transfers, these became acceptable, especially for dental fixture placements.

The attachment properties of the overlying mucosa and underlying muscular elements on to the bone may directly affect the success of oral rehabilitation. In some cases the attachment zones of the soft tissues may be distributed in rather broad form or they may be a localized suspender close to or over the alveolar crest. According to the area affected, surgical reconstruction is needed to reposition attachment zones.

The attachment of the mucosa and width of keratinized epithelium can also change due to resorption, pathology, previous surgery or trauma.

The pattern of changes of the oral mucosa may not require any intervention in fully dentate patients, but surgical management will have to be incorporated in the overall oral rehabilitation plan in certain cases. These may range from minor detachment of the localized areas to replacement or reconstruction of wide zones, particularly for functional reasons. Functional sulcus is an important element of the oral cavity: it contributes to oral hygiene maintenance, unrestricted movement of cheeks, lips and tongue, and phonation. The loss of this area complicates rehabilitation in edentulous patients.

It should be kept in mind that soft tissue anatomy may reflect many conditions, which may accompany underlying and supportive bony abnormalities. In these instances the procedure should include bone architecture improvement strategies. These strategies not only enable improvement of the supportive bony element but also improvement or correction of the overlying soft tissue envelope. These procedures can be employed simultaneously or in a staged fashion which may require multiple interventions.

Soft tissue problems that require a surgical plan should be employed with care as sometimes a hidden medical condition mimics an otherwise simple, localized architectural change. In recent years considerable progress has been made in the selection and inclusion of cases requiring preprosthetic surgery, where an almost complete shift to implant-supported restorative concepts has been observed. Surgeons now have more alternative soft tissue improvement techniques in addition to the earlier fundamental techniques, particularly for achieving more functional anatomy for denture-bearing regions. There are also several other procedures available to improve or optimize oral rehabilitation.

History

The early attempts in the history of preprosthetic surgery started with restoration of intraoral defects by grafting procedures at the end of the 19th century. Reverdin published the first paper on epidermal grafting in 1869.³⁸ After the Franco-Prussian War of 1870–1871, Carl Thiersch worked on epidermal grafting and was able to show viability of thin grafts by microscopic observations. Thiersch's discovery in 1874, that skin could be grafted to cover wounds, played an important role in the development of soft tissue grafting. In 1894, Schnitzer and Ewald applied a Thiersch graft in the buccal mucosa. Later, in 1915, Moskowicz was first to create a vestibule by intraoral skin grafting.⁴

Kazanjian, among one of the best known pioneers in preprosthetic surgery, demonstrated a crestally based mucoperiosteal flap for buccal site vestibulo-

plasty.⁵ Weiser was cited as the first to perform maxillary vestibuloplasty by skin grafting in 1918. Submucous vestibuloplasty was also recommended by Kazanjian but the technique was later found to be quite unsuccessful.⁶ Obwegeser described a method working successfully for maxillary submucous vestibuloplasty in 1959.⁷ Kazanjian, Clark, Obwegeser, Edlan, Rumpel, Szaba, and Godwin developed various techniques for vestibuloplasty by secondary epithelialization; however most of them had unpredictable results.⁴

Meanwhile it was demonstrated that supraperiosteal deepening without skin or mucosal grafting resulted in significant deterioration in vestibular or lingual sites. Trauner and Obwegeser popularized buccal and lingual sulcus deepening by skin grafts adapted to flanges of a denture.⁸ Split-thickness skin grafting was the mainstream preprosthetic procedure performed in the 1950s to the 1970s. The rationale of grafting lingual and buccal sulcus with skin grafts was based on three main issues: (1) mechanical resistance to displacement forces; (2) stable denture-bearing area; and (3) skin as a load-bearing tissue.⁴

A reduction in the use of skin grafts occurred after the introduction of mucosal grafts in the mid-1970s. Intraoral mucosal grafts have found worldwide acceptance in clinical practice, because skin grafts have major disadvantages and morbidity. In the 1990s, the International Research Group on Reconstructive Preprosthetic Surgery was founded to establish and promote basic and advanced techniques in this field. The first major meeting was organized in Copenhagen (1997) and the second in San Diego (1999). After each meeting, consensus statements were published as the main guidelines of preprosthetic surgery.⁹

Oral soft tissue anatomy and histology

The oral cavity is the upper end and the beginning of the digestive system and has a connection with the respiratory system.¹⁰

Lips

The muscles of the lips are the superior and inferior labial muscles and the orbicularis oris. Lips are the junction between the skin of the face and mucosa of the oral cavity. The vermilion zone of the lip is the red–purple area between these two areas. The corners of the lips are usually located adjacent to the maxillary canine and mandibular first premolar teeth.¹¹ This is an extremely important area, known as the esthetic zone, which determines distribution of dental implants in the arches during placement.

The inner surface, the labial mucosa, is covered by a thick, non-keratinized epithelium. The lamina propria is wider at that site where papillae are shorter

and irregular. The submucosa contains many minor salivary glands. Dense connective tissue binds the mucosa to the underlying orbicularis oris muscle.¹²

Cheeks

The cheeks extend intraorally from the labial commissures anteriorly to the ridge of mucosa overlying the ascending ramus of the mandible posteriorly. The mucosa is non-keratinized and tightly adherent to the buccinator muscle. The parotid papilla is visible at the level of the first and second upper molars.^{11,13} Sensory innervation of the cheeks is by buccal branches of the mandibular nerve; the cheeks are supplied by buccal branches of the maxillary artery.^{12,14}

Gingiva

The attached gingiva is firmly fixed to the underlying periosteum of the alveolar bone and to the cervical level of teeth. The free gingiva lies unattached around the cervical region of the tooth. The gingiva of the maxillary anterior palate is innervated by branches of the nasopalatine nerves, whereas the posterior palate is innervated by the greater palatine nerves. The labial and buccal gingiva of the anterior mandible extending to the premolar area is innervated by the branches of the inferior alveolar nerve. The buccal gingiva of the mandibular posterior area is innervated by the buccal nerve. The lingual gingiva is supplied by the lingual nerve and vessels.

The mucogingival junction is a connection region of the attached gingiva and the alveolar mucosa. The difference in appearance and color between alveolar mucosa and attached gingiva is determined by level of translucency and keratinization. The epithelium of the alveolar mucosa is translucent and the blood vessels lie superficially. The attached gingiva is specially keratinized tissue which serves as a masticatory mucosa.^{11,14}

The floor of mouth

The floor of the mouth is positioned above the mylohyoid muscle and beneath the tongue. It is covered by non-keratinized mucosa. In the mid-anterior area, the lingual frenum connects the inferior surface of the tongue and mouth floor; sometimes it extends to lingual cortex of alveolar bone.^{11,15} In severely atrophic lower jaw, the lingual frenum should be resected to eliminate the pulling effect of the tongue. The level of the floor of the mouth needs repositioning when lingual flanges sit on the genial tubercles, which become very prominent after extensive resorption. Resection of genial tubercles can be performed 3 weeks prior to lingual sulcoplasty.

The palate

The soft palate is lined with a non-keratinized mucosa which covers connective tissue with short and broad

papillae. Conversely, the hard palate has a typical masticatory mucosa with keratinized epithelium. In the mid-palate, submucosa is missing, thus the dense lamina propria joins to the palatal bone. The existing submucosa at the connection of palate and alveolar ridge contains the greater palatal neurovascular bundles. Many minor salivary glands and taste buds are dispersed through the mucosa of the hard palate. The soft and the hard palate are separated by a well distinguished color change of palatal mucosa.¹⁶

Oral mucosa consists of two distinct layers. The external layer is a stratified squamous epithelium. This layer includes keratinized mucosa that is subjected to chewing forces. The connective tissue and lamina propria are located under the epithelium. The submucosa consists of a looser connective tissue containing fat deposits and glands. Larger nerves and blood vessels run in the submucosa.¹⁷

Primary indications of surgical procedures

Preprosthetic surgical procedures originally emerged from a need for successful construction of removable full and partial dentures. Patients were only satisfied if stability, retention, function, and esthetics were all achieved together.¹⁸ The selection of surgical procedures and their success essentially depends on the preoperative evaluation of the supporting hard and soft tissue structures and their proper preparation.

Currently surgical interventions of this nature need to address both function and esthetics. In order to attain reproducible and predictable favorable outcomes, a problem list defining all soft and hard tissue abnormalities should be noted and be managed by various methods. These may be isolated soft tissue procedures, bony enhancement or reconstructive modalities, including augmentation of deficient alveolar bone, orthodontic tooth eruption, socket preservation, or guided bone regeneration to improve overlying or neighboring soft tissues.^{19,20}

Sometimes correction of underlying bony structures may have the advantage of rearranging or improving the overlying soft tissue configuration. Therefore some surgical techniques directed primarily to the bone may almost obviate the need for soft tissue surgical reconstructive techniques. On the other hand, soft tissue corrections have to be preceded by supportive tissue or underlying structural surgical interventions in order to achieve purposeful and significant results.

The relationship of anterior dentition to gingiva and upper lip is deemed important for desirable dental esthetics. It is often impaired by an unharmonious contour of the gingival margin in the anterior region, and a disproportional relationship between lip and clinical crown length. These often require different plans although they may present concomitantly.

Anterior esthetics with an unfavorable clinical presentation may or may not require a surgical plan that primarily involves teeth and gingiva. All that may be needed in some selected patients is to address a skeletal problem and esthetics are then corrected secondarily. In such instances orthognathic surgery is executed to refine and correct improper arrangement of these elements. However the problem can be solely related to an unharmonious or distorted gingival margin where procedures need to be employed to reverse the situation. In such cases solely dealing with soft tissue may not be the treatment of choice, and soft tissue contour can be improved by bony procedures such as orthodontic eruption of relevant teeth prior to prosthodontic treatment.

In addition, sinus augmentation, bone grafting or enhancement modalities and orthognathic surgical techniques can be employed singly or in combination to address hard and soft tissue deficiencies. Corrective attempts by surgical means may sometimes play a significant role in determining the success of implants and implant-supported rehabilitation through soft tissue healing. Preprosthetic surgical procedures can include repositioning the altered muscle attachments and correction of functional sulcus loss or deficiencies where several grafting modalities may also be required. This is particularly desirable for creating an appropriate environment for the placement of implants and dentures. To accomplish primary soft tissue closure, undermining, free grafting, pedicle or free flaps are the techniques of choice, and several techniques have been described.

Understanding the relationship between hard tissue, soft tissue, and tooth position is critical for optimal esthetic implant restoration. General biological guidelines have to be considered, however the individual condition of each case should be evaluated. Another important issue is the assessment of implant sites prior to and after loss of tooth to help maintain the site in a state of esthetic predictability. If the site is not ideal, a variety of procedures using orthodontics, hard tissue reconstruction, or soft tissue corrections should be instituted to enable optimal placement of an implant.²¹ These procedures facilitate the placement of a restoration that harmonizes with the adjacent dentition.

Implant site reconstruction in the esthetic zone is essential to create an inconspicuous implant restoration. If teeth are congenitally missing, or have been lost at an early age, the site may be underdeveloped. In addition, tooth extraction following extensive infection or pathology as well as trauma may lead to alveolar bone deformities. In such instances the goal is to create an enhanced site for the restorative clinician.

The treatment modalities must be appropriate for each diagnosis, based on residual ridge position, resorption pattern, and lip support. Preprosthetic surgery has been used for a considerable number of edentulous patients for optimization of prosthetic

rehabilitation. It should be borne in mind that there is also an increase in demand of natural oral and facial postoperative function and appearance.

After surgical management of oral tumors, preprosthetic procedures may be required to improve the patient's mood and to improve quality of life by allowing completion of reconstruction.²²

Biotype conversion around both natural teeth and implants has been advocated, and the resulting tissues appear to be more resistant to recession.²³ Subepithelial connective tissue or free gingival grafting at implant sites is used to achieve a more stable peri-implant tissue particularly in thin biotype situations. Facial gingival recession of thin periodontal biotype seems to be more pronounced than that of thick biotype. Unavoidable facial gingival tissue recession is still common, however, particularly at 1 year of function.

Implant-related indications and soft tissue interactions

Soft tissue integration

Soft tissue integration as a term is used to describe the process of soft tissue healing (formation, maturation, and reshaping/adaptation) occurring around the transmucosal part of endosseous dental implants and surrounding oral mucosa. The success of osseointegration is basically judged by the quality and quantity of alveolar bone, but the quality and quantity of overlying soft tissues have an unquestionable role. Every oral surgeon knows the basic steps for dental implant placement in ample alveolar bone. Management of soft tissue problems or related insufficiencies is the key to long-term success. Establishment of an esthetic and healthy peri-implant soft tissue interface should be the primary goal of implant therapy. Long-term predictability and patient satisfaction could be obtained by creating a stable peri-implant environment, using basic and advanced surgical procedures such as soft tissue augmentation, flaps, and periodontal plastic procedures.

Surgical management of peri-implant soft tissue

Implant-related indications

Preservation of healthy peri-implant soft tissue is an important and challenging part of dental implant treatment. It is achieved by tight adaptation of oral mucosa to the marginal section of a dental implant, forming a natural soft tissue barrier. The existence of any periodontal pathology will result in structural breakdown of this soft tissue barrier, and this promotes the onset of a peri-implant disease.

Careful evaluation of peri-implant soft tissues includes determination of quantity and quality of attached gingiva, which can be performed with a periodontal probe during bone mapping prior to implant insertion. Implant-related indications for soft tissue surgery include lack of soft tissue volume around implants, presence of hypertrophic gingiva, esthetic reasons, inadequate vestibular depth, and inadequate depth of lingual sulcus.

Mucosal or mucoperiosteal flaps are developed according to the type of existing soft tissue problem. The incisions should be placed at the crestal level in order to leave adequate amounts of keratinized and well perfused mucosa at both buccal and lingual sites. The criteria for incision and flap development vary according to whether the marginal bone level of the implant is submerged or non-submerged. A palatal incision is preferred to hide the incision line and prevent any mucosal fenestration of submerged implants during the osseointegration period (Figs 19.1 and 19.2). In contrast, a crestal incision is widely accepted to facilitate soft tissue adaptation around necks of non-submerged implants.

Papilla regeneration

The papilla regeneration technique is a simple and innovative method to form a new papilla between implants or teeth. The vestibular flap is used as a donor site, and a small semi-lunar flap developed from the vestibular flap is rotated mesially or distally



Fig. 19.1 Flap design for edentulous maxillary arch.



Fig. 19.2 Flap design for total edentulous mandibular arch. The dotted lines indicates the vertical releasing incisions if needed.

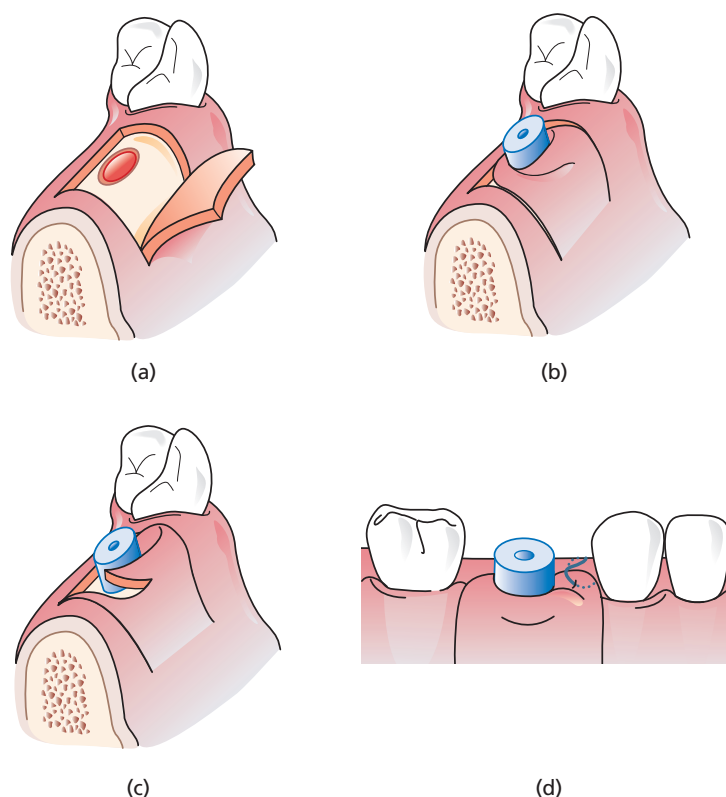


Fig. 19.3 The papilla regeneration technique described by Pallaci.

where formation of a new papilla is desired (Fig. 19.3). A small palatal or lingual crestal incision is made to uncover the implant and, immediately after a healing cap is inserted, a semilunar incision is performed and rotated.²⁴

Oral soft tissue grafting

Success of oral soft tissue grafting mainly depends on vascularization of a graft at the host site, graft immobilization, host site hemostasis after graft fixation, and graft thickness. Vascularization of full- or split-thickness soft tissue grafts occurs in two stages: plasmic imbibition and inosculation of blood vessels. Plasmic imbibition is the first process of graft healing in which plasma-like fluid accumulates under the graft and is absorbed by thin inner endothelial cells. This absorption continues over 48 hours, while a fibrin network and blood flow are established. In the second phase, vascular buds inosculation with arteries

and veins in the graft and anastomoses develop between the host and the graft.¹⁹

The host region should provide proper vascularization properties for graft integration. Exposed implant surfaces or thick scarred mucosa are poorly vascularized areas that should be covered with flaps retaining their blood supply, such as a vascularized periosteal connective tissue flap or a lateral advancement flap. Immobilization of a transplanted graft increases the chance of graft survival. Mobility of a graft prevents establishment of microcirculation between the host site and the graft. Active hemorrhage after graft fixation prevents formation of blood clots and adaptation of the graft to the host site.

The graft should be large and thick enough to cover the denuded region. The size of the graft should compensate for postoperative contracture. The thinner the graft is, the better it is incorporated at the host site. Thicker (full-thickness) grafts are suitable for closure of abutments or healing caps.

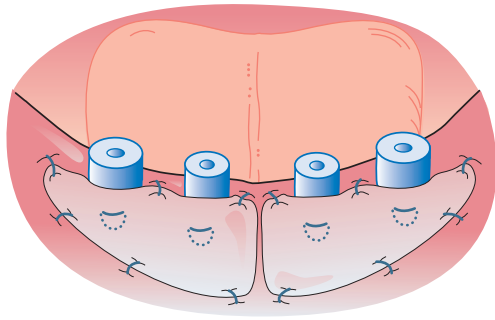


Fig. 19.4 Schematic representation of onlay palatal mucosa grafting to form peri-implant attached mucosa at the neck level of implants.

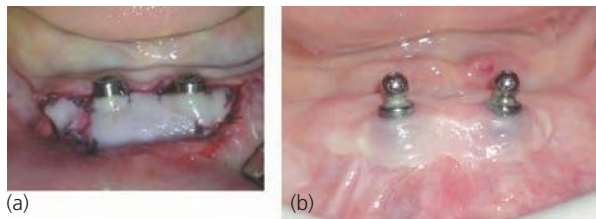


Fig. 19.5 (a) Palatal mucosal graft adapted to the host site. (b) Free palatal graft increased the width of labial attached gingiva and gained extra support against peri-implant flora and lip stretching.

Palatal mucosa grafting

Mucosal palatal grafts were used successfully for years in periodontal practice to cover denuded roots. A similar philosophy is applied to dental implants which have loose alveolar mucosa on their labial sides. The quality and amount of attached gingiva determines the predictability of peri-implant soft tissue inflammation. Restoration and maintenance of an acceptable band of attached gingiva around dental implants is essential for satisfactory outcome from implant therapy (Fig. 19.4).

The general technique for obtaining keratinized peri-implant soft tissue is application of palatal mucosal grafts which have superior resistance to inflammation at the crestal level. Split- or full-thickness palatal grafts can be harvested (Fig. 19.5). In severely atrophied jaws, gingival grafting should be performed prior to implant placement when the height of the mandible is less than 10 mm and has less than 3 mm attached gingiva.

Subepithelial connective tissue grafting

Ridge augmentation by connective tissue grafts is used for enhancement of esthetic appearance of soft tissue surrounding abutments. Biologically, subepithelial connective tissue grafts (SCTG) exhibit superior ability to heal in grafted sites since the graft is interposed between split-thickness upper layer and the periosteum so it receives its perfusion from both sites.²⁵

For convenient grafting, the SCTG should be 1.5–2 mm thick. Multiple pieces of varying size and shape could also be used for correction of soft tissue

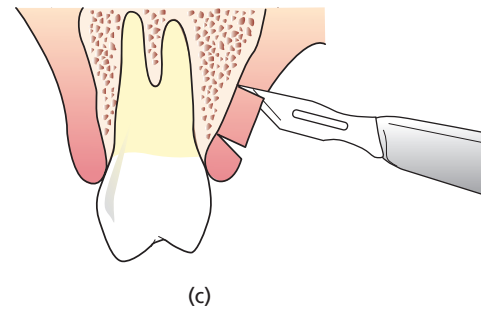
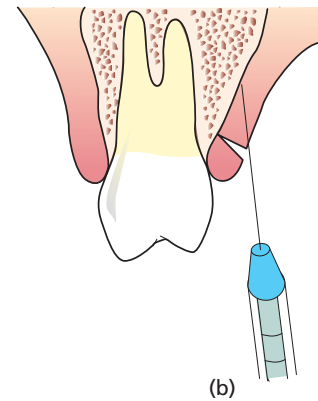
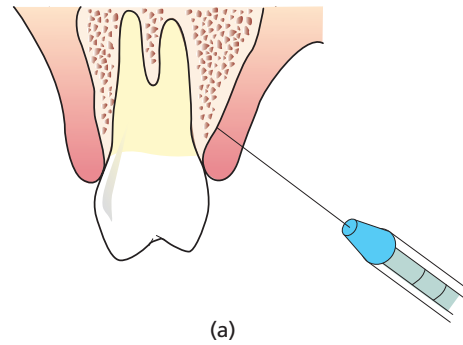


Fig. 19.6 The double incision technique to retrieve an SCTG from the palate. (a) Horizontal, initial incision. (b) Second, beveled incision. (c) Harvest of connective tissue with a scalpel.

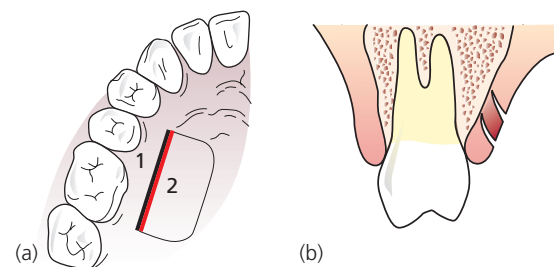


Fig. 19.7 Bruno's technique for harvesting an SCTG. (a) First short incision. (b) Beveled vertical incision to remove the graft from the base.

deformities. Several techniques have been described for SCTG harvesting, of which Bruno's method (Figs 19.6 and 19.7),²⁶ open door technique with broad-band pedicle or with narrow base (Fig. 19.8), and double-scalpel technique²⁷ are mostly used. Greater palatal vessels may be endangered during harvesting of connective tissue, therefore the donor site should not be prepared too far distally.

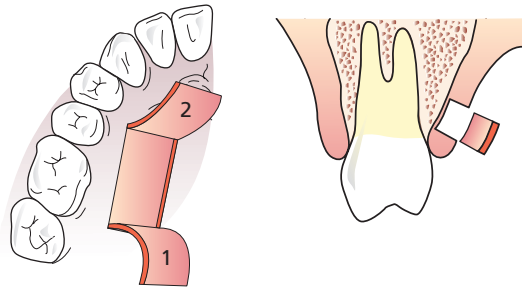


Fig. 19.8 The open-door technique for a SCTG harvest.

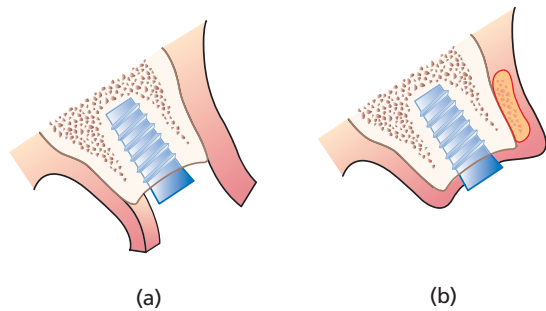


Fig. 19.9 Reconstruction of a soft tissue defect on the buccal side. (a) Creation of palatal and buccal flaps. (b) A connective tissue graft is inserted under deficient mucosa.

In Bruno's method two incisions are used:

1. Initial, paramarginal horizontal incision which is made down to the palatal bone.
2. Second, beveled or vertical incision in which scalpel touches palatal bone and goes 10mm deep parallel to the tooth axis (Fig. 19.6).

The periosteal elevator dissects the connective tissue with attached periosteum and primary closure of flaps is obtained at the donor site (Fig. 19.7).²⁶

After harvest of SCTG, flaps are prepared at the host site and the graft is submerged. At least 3 weeks are required for optimal healing before any prosthetic or surgical procedure is carried out. The grafting technique is described in Figs 19.9 and 19.10. Modification of this procedure includes rolling of a pedicled SCTG graft under a buccal flap.

Vascularized periosteal connective tissue flap

The vascularized periosteal connective tissue flap offers reconstruction of large soft tissue deficiencies



Fig. 19.10 (a) Mucosal depression on the buccal gingival band. (b) Design of an incision. (c) Harvest of a connective tissue graft from the palate. (d, e) Insertion of the graft and slight roll-up of the flap over the abutment. (f, g) Postoperative view of the grafted gingiva during prosthetic treatment.

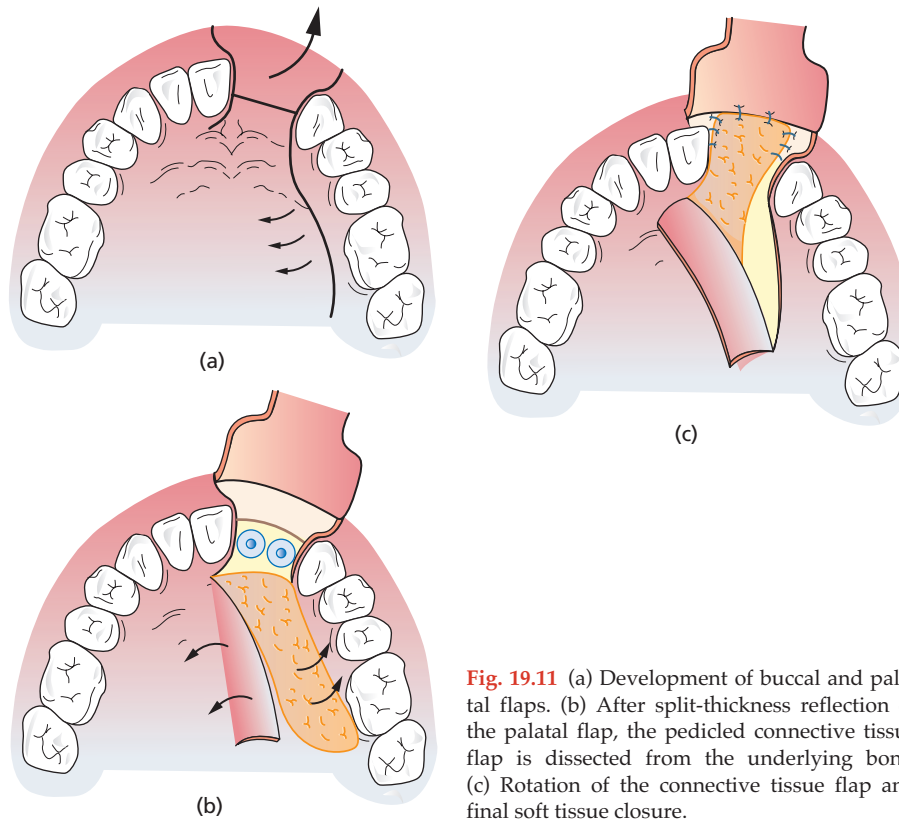


Fig. 19.11 (a) Development of buccal and palatal flaps. (b) After split-thickness reflection of the palatal flap, the pedicled connective tissue flap is dissected from the underlying bone. (c) Rotation of the connective tissue flap and final soft tissue closure.

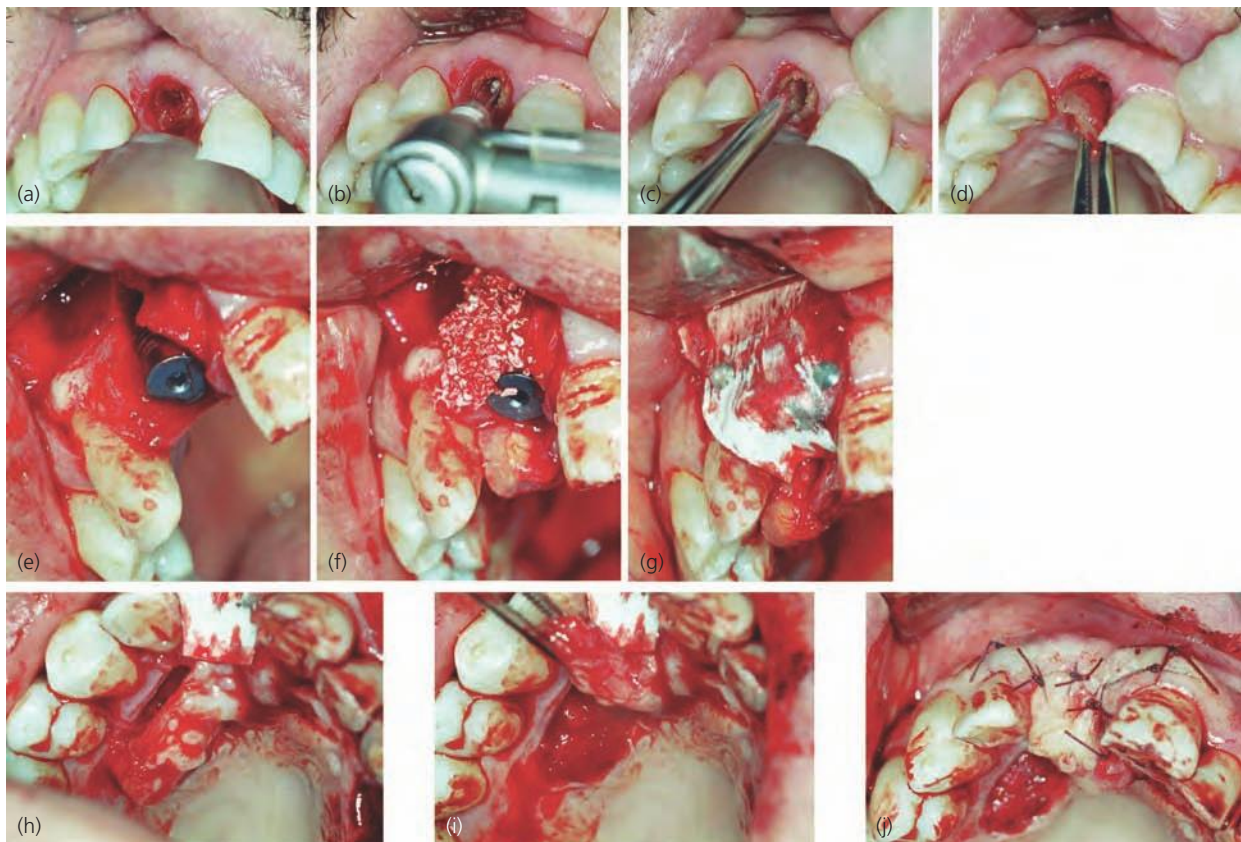


Fig. 19.12 Reconstruction of an extraction socket with guided bone regeneration, immediate implant placement and rotation of vascularized periosteal connective tissue flap. (a) The fractured root. (b) The socket preservation technique used with an implant drill. (c, d) Extraction of the root with forceps. (e) A fenestration-type bony defect at the buccal crest occurred after immediate implant placement. (f) Augmentation of the defect with allogenic particulated bone. (g) Application of a resorbable membrane over the reconstructed alveolus. (h) Development of a vascularized flap from the palate. (i) Rotation of the flap over grafted region. (j) Final view after soft tissue closure.

at the anterior maxillary ridge. The amount of transferred tissue is capable of reconstructing the compromised region in both the horizontal and vertical planes (Fig. 19.11). The size of pedicle is determined by the requirement of soft tissue reconstruction. The rotated flap brings its blood supply within the connective tissue envelope, thus perfusion is enhanced during the healing period (Fig. 19.12). The advantages of rotating a vascularized connective tissue flap are reconstruction of soft and hard tissue at the same time, short healing period, and treatment of scarred tissue in the esthetic zone.¹⁹

Minor preprosthetic procedures

Before fabrication of dentures, it is essential to evaluate skeletal discrepancies of alveolar or palatal processes. Any soft tissue impingement under flanges caused by irregular bony topography would create extreme difficulty for patients during oral functioning. The bony aberrances may be sharp-edged, lobulated or broad, and may disturb the health of overlying mucosa, compromising its structural integrity with progressive fibrotic transformation and irritation due to inflammation.²⁸

Tori on palatal and lingual sides may cause extra discomfort by preventing seating of dentures. Slightly larger and active frena on lingual and buccal sides may interfere with seating or dislodgement of dentures. Again, sharp-edged mylohyoid ridges or alveolar crest may be responsible for continuous irritation and pain during occlusal forces. Epulides on the maxillary vestibule are also sources for infection, irritation, and pain, and they must be removed before impressions are taken. The aforementioned situations are observed very often in clinical practice and it is imperative to resolve underlying bony or soft tissue problems before prosthetic treatment proceeds further.

Alveoloplasty along with tooth removal

Preservation of alveolar bone during extraction of a tooth is the main objective of a general practitioner. An attempt to prevent loss of marginal alveolar bone by grafting the empty tooth sockets and covering grafted sites with resorbable/non-resorbable membranes was practiced in the past, but it is not recommended due to its questionable success.

Extraction of fractured tooth apices is problematic because surgical removal will cause inevitable destruction of residual alveolar bone in the apical area. If the apex is uninfected and root is small it would be better to leave the root. Another problem mostly encountered after multiple extractions is postextraction irregularity of alveolar sockets. The irregular surface may be trimmed with a bur, a rongeur, a fine osteotome or a bone file. A horizontal

crestal incision with two releasing incisions is made to provide access. Great care is taken during flap elevation since soft tissue is tightly adapted to undercuts of irregular bone. Whenever possible, it is better to use a file or rongeur in minor discrepancies to prevent decrease of alveolar height. Excessive trimming may cause filling of sockets with mucosa which later results in decrease of bone height.

The maxillary alveolar process that is inclined buccally results in additional difficulties such as blocking the intraoral route of dentures. In severe lateral projection, mucoperiosteal flaps are developed from both buccal and palatal sites. After interdental septa are removed with a hard bur, two vertical bony cuts are made on the buccal alveolar wall and palatal sites which help to weaken the alveolar process. Unfortunately, removal of interdental septa inhibits growth of endosteal bone which is important to preserve the ridge form.

The alveolar process is squeezed with the fingers to create green-stick fractures on both sides. The flaps are sutured and the ridge is stabilized. In cases with less inclined maxillary ridges, it is logical to perform buccal wall corticotomy only on the buccal side. The edges of the mucoperiosteal flap are trimmed, reduced, and closed in primary fashion. Resorbable running or lock stitch suture could be used for closure.

Treatment of exostosis

Exostosis of alveolar bone is a benign osseous hypertrophic formation. The exostosis occurs more commonly in the maxilla. Large undercuts may also be present after extractions. Secondary alveolar recontouring is indicated in such cases. A mucoperiosteal flap is elevated after adequate infiltration of the mucosa. In large exostosis, an anesthetic block of the inferior alveolar nerve may be required. An envelope flap is developed with a vertical releasing incision not going too deep in the vestibule. The bone is resected and smoothed with an osteotome or a bur.

If implant placement is considered appropriately at crests with exostosis or undercuts, the defects under these irregularities are treated with autogenous bone grafts or guided bone regeneration.

Reduction of genial tubercles

The genioglossal muscle attaches to the genial tubercle located at the lingual aspect of mandible. As the mandible resorbs, the genial tubercle becomes prominent and the mouth floor ascends simultaneously with the resorption process. During fabrication of dentures, the activity of the genioglossal muscle and protuberance of the genial tubercle complicates wear of dentures by causing dislocation during function. Lingual flanges have to be made shorter to avoid direct pressure on the prominent genial tubercle, and short flanges result in inadequate prosthetic retention. Lowering of mouth floor procedures could also

be considered when the genial tubercle and genioglossal muscle interfere with retention and stability of a prosthesis, but this technique is no longer recommended.²⁹

A crestal incision is performed with a #15 scalpel along the anterior ridge. Subperiosteal dissection is continued under the lingual mucoperiosteum until the genial tubercles and muscles become evident. Sharp dissection of the genioglossal muscles is performed with monopolar cutting electrocautery in order to control any hemorrhage from the muscle. The genial tubercle is resected with a fissure or round bur. Strict hemostasis should be obtained prior to closure of the lingual flap otherwise life-threatening airway restriction may occur due to hematoma formation in the floor of the mouth.

Reduction of mylohyoid ridge

In extensive resorption of the mandible, the mylohyoid ridges become prominent and contribute significantly to denture displacement with strain of the mylohyoid muscles. Pain is felt because of mucosal impaction of dentures at the sharp mylohyoid ridges. With the introduction of dental implants, lowering of the mouth floor and reduction of the mylohyoid ridge became a historical procedure. However, in sporadic cases, indications still exist for surgical resection of sharp ridges.²⁹

After accomplishing lingual, inferior alveolar, and buccal nerve blocks, a crestal incision is made along the posterior mandibular ridge. Subperiosteal dissection is carried out to expose the mylohyoid ridge and muscle. The attachment of the mylohyoid muscle to the ridge is grasped with a curved clamp and the muscle is separated by electrocautery. The mylohyoid ridge is smoothed or resected with a bur. At this stage, if lowering of the mouth floor is going to be performed, the lingual flap is sutured percutaneous to skin of the submandibular crease with a Reverdin needle. If sulcular deepening is done on the buccal side, the lingual flap is sutured to the edge of the buccal flap. Lowering of the mouth floor is a historical procedure; it is not used routinely any more.

Removal of tori

Palatal and lingual exostoses are known as "tori". Lingual tori are generally bilateral and can be singular, lobulated or multiple. These benign osseous growths should be removed prior to dentures being contemplated. The lingual tori are accessed with horizontal crestal incision. Vertical incision must be avoided as much as possible since perfusion of thin overlying mucosa could be affected. The lingual torus can be removed with a bur or an osteotome. It would be of benefit to place gauze under the tongue to prevent hematoma formation.

Palatal tori of unknown etiology are observed mostly in the middle of the palatal vault. The inci-

dence in women is twice that in males. Palatal tori can be broad, lobulated, or nodular. Indications for removing the palatal tori are:

- large nodular and lobulated tori interfering with seating of dentures;
- torus with traumatized mucosal surface;
- torus extending beyond the palatal region;
- torus with deep undercuts and multiple nodules;
- torus that complicates speaking;
- psychological problems.

Before contemplating a torus operation on the palate, an impression is taken and a translucent surgical stent is fabricated. The patient should be informed about the possibility of formation of an oroantral fistula in the postoperative period, since the mucosa overlying the palatal torus could be very thin and susceptible to perforation.

Various incisions have been described for removal of palatal tori. A C- or U-shaped incision, open-door technique (double Y incisions over tori) or palatal flap technique could be chosen, according to the type and height of tori. After infiltration of the major palatal nerves and nasopalatine nerve, an incision is made and a subperiosteal flap is elevated. Small tori are removed with a round bur but large tori are sectioned first with a fissure bur and then removed with an osteotome or egg-shaped bur (Fig. 19.13). Extra care is taken to avoid extensive removal of palatal bone and subsequent perforation of the nasal floor. The flaps are sutured in a primary fashion after copious irrigation with saline. The surgical stent is applied with a tissue conditioner to avoid formation of a hematoma and dead space under the stent.

Recontouring of soft tissues

Maxillary tuberosity reduction

Enlargement of the maxillary tuberosity may occur due to soft tissue bulking. Increase in vertical dimension at the posterior ridge may limit the intermaxillary space required for denture construction. With enlargement of the maxillary tuberosity, pneumatization of the antrum may descend into the tuberosities. Pneumatization of sinuses must be checked with a panoramic radiograph prior to surgical reduction. Entrance to the maxillary antrum would not complicate the procedure.

A wedge-shaped resection of excessive tuberosities is performed (Fig. 19.14). Two elliptical incisions are made and a subperiosteal dissection is continued to the tuber maxilla. Excessive submucosa is undermined with sharp dissection and removed from the buccal and palatal flaps. Removing submucosal contents is important for approximation of flaps on both sides (Fig. 19.15). Antral communication during bone removal is not a problem if the antrum is free of any disease, infection, etc.²⁹ The surgical field is irrigated

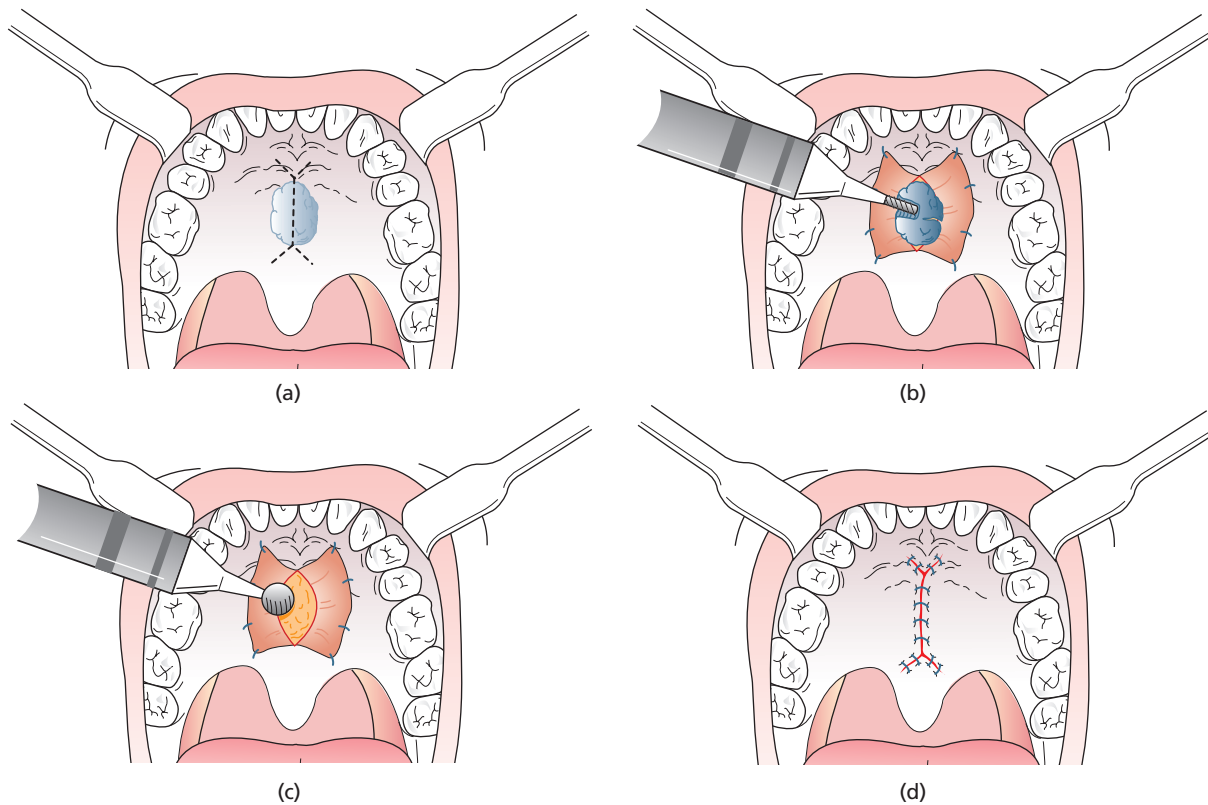


Fig. 19.13 Removal of a palatal torus. (a) Design of an open-door incision. (b) Sectioning of the hard nodules with a fissure bur. (c) Smoothing the palatal bone with an egg-shaped bur. (d) Final closure.

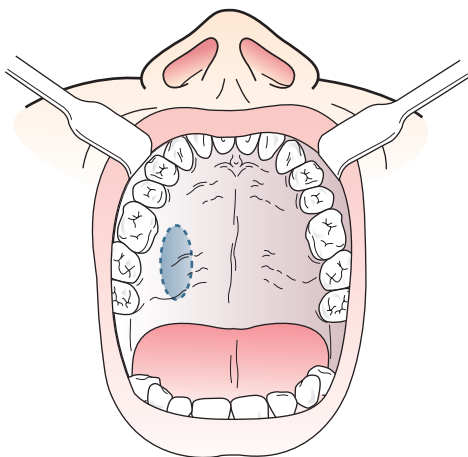


Fig. 19.14 A wedge-shaped elliptical excision for tuber reduction.

copiously, and buccal and palatal flaps are trimmed to prevent overlapping and closed primarily.

Frenectomy

Various techniques have been described for removal of active and strong frenum attachments. These are V-Y advancement flap, Z-plasty, and diamond-shaped excision.

The diamond-shaped excision is usually used and provides optimal results. Local anesthetic with epinephrine is injected into the mucosa adjacent to the frenum. Direct injection into the frenum is avoided since anatomical borders may disappear, and result

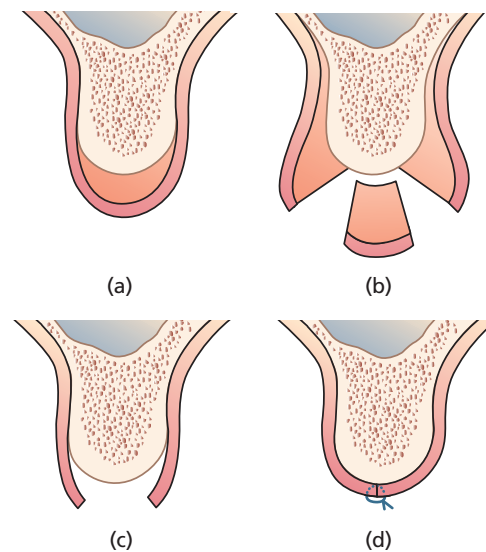


Fig. 19.15 Reduction of an abundant maxillary tuberosity. (a) Redundant soft tissues of the maxillary tuber. (b) The wedge excision. (c) Undermining the submucous tissues. (d) Final closure.

in excessive excision of mucosa. Eversion of the upper lip also helps to identify borders of a frenum. In the diamond-shaped excision technique, two hemostatic clamps are applied to the superior and inferior section of the maxillary midline diastema. With a #15 scalpel, the clamped tissue is resected immediately from the mucosa. The upper vertical incision is closed with simple sutures and the inferior part, localized at the attached gingiva, is left to heal secondarily (Fig. 19.16).

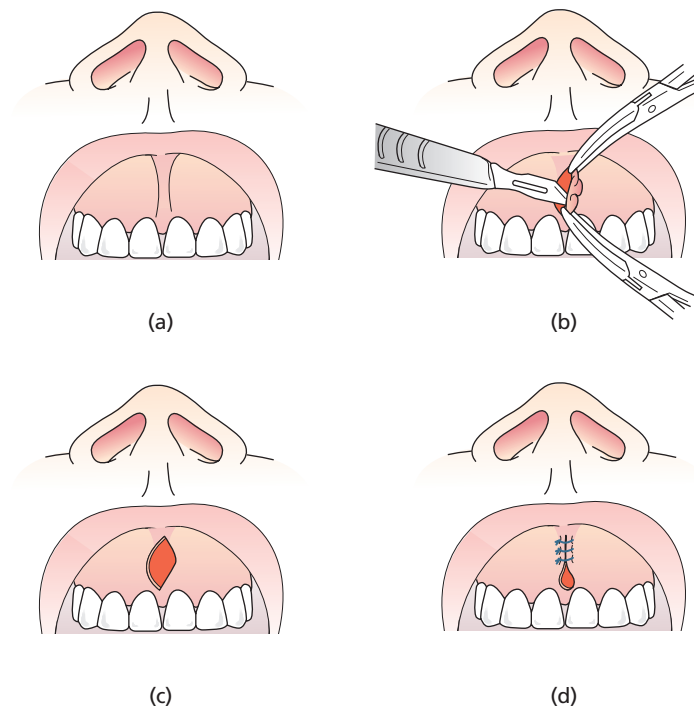


Fig. 19.16 (a) A labial frenum. (b) The excision is performed adjacent to a clamp. (c) Residual defect after excision. (d) Suturing upper end of the wound, the lower raw surface is left for secondary epithelialization.

The Z-plasty technique may produce less scar than the diamond-shaped excision. Z-plasty is usually indicated for a frenum with a wide crestal part and shorter alveolar height because it increases alveolar height. An incision is made directly along the length of a frenum. Superior and inferior incisions are placed to form triangles. After supraperiosteal undermining, the apex of the inferior triangle is rotated upwards and conversely the apex of the superior triangle is rotated downwards (Fig. 19.17).

If the frenum is broader, localized vestibuloplasty offers better results compared to the above techniques. An incision is placed at the base of a frenum and the frenal attachment is elevated with supraperiosteal dissection. The area is left to heal by secondary epithelialization.

Lingual frenectomy (tongue tie, ankyloglossia treatment)

In edentulous older patients, the lingual frenum may attach high at the crestal level. This attachment may cause speech difficulties and denture instability. The mouth floor is infiltrated with local anesthetic. A

suture is placed in the tip of tongue for reflection and exposure of the lingual frenum. The most important anatomical structures are the orifices of Wharton's duct and the vascular network of the mouth floor. Connective tissue at the base of the tongue is released with a scalpel or electrocautery and blunt dissection is performed with dissecting scissors to enable approximation of flaps. Fibrotic tissue starting from alveolar ridge to the base of tongue is then resected while additional attention is paid to tissue between sublingual carunculi. It is preferred to close the area with resorbable sutures.

Removal of palatal papillary hyperplasia

Papillary hyperplasia of palatal mucosa is a pathologic condition of unknown etiology and mainly occurs with ill-fitting dentures. Day-long wearing of dentures may be responsible for the condition, however the disease is observed also in patients who do not wear their dentures for 24 hours a day. Papillary hyperplasia is presented with reddish small nodular inflammation and edema of palatal mucosa. Mechanical irritation seems to be an initial factor,

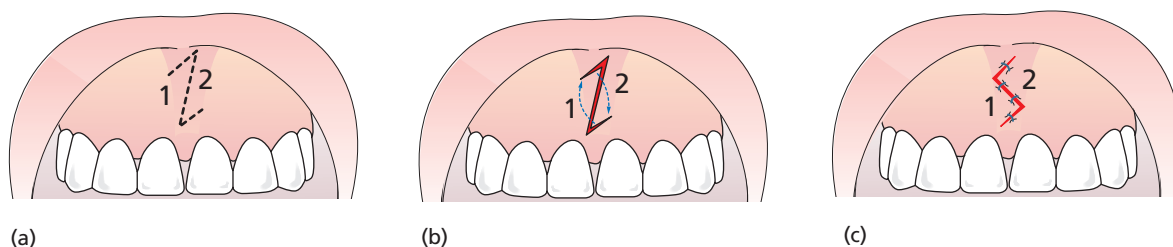


Fig. 19.17 The Z-plasty frenectomy technique. (a) Outlining of the Z flaps. (b) Creating the small triangular flaps. (c) Rotation of the flaps and final closure.

however isolated cultures from lesions demonstrate predominance of *Candida albicans*, a species that increases in mouths with poor levels of oral hygiene.

An initial therapy is removing ill-fitting dentures or relining dentures with tissue conditioners over a 6-month period. Anti-fungal ointments may be used to eliminate candidiasis. Nystatin or clotrimazole are drugs of choice. In advanced stages of papillary hyperplasia, surgical excision should be accomplished with the following methods: rotary instruments, debridement with scalpel, electrocautery, and laser resurfacing.

Vestibuloplasty

Preprosthetic surgery attempts to create an oral environment that can properly support a functional prosthetic appliance and should be considered as an essential component of prosthetic reconstruction. Difficulties faced in the prosthodontic rehabilitation of a patient are re-establishment of the best masticatory function combined with restoration or improvement of dental and facial esthetics.

The factors affecting bony and soft tissue changes are:

1. General factors – systemic bone disease, nutritional abnormalities.
2. Local factors – size, shape of alveolus before and after extraction, effects of ill-fitting prosthesis.

A thorough and comprehensive examination, diagnosis, and treatment plan will reveal that surgical intervention prior to commencement of prosthodontics can improve the prognosis of patients. Detailed intraoral examination and review of medical history, physical evaluation, discussion with the patient about his/her chief complaint, goals and expectations for long-term function and esthetics, psychological factors, and level of patient's surgical risk should be considered while developing a treatment plan. As a general rule the best procedure to choose is the least invasive one offering predictable clinical success.

Oral evaluation should include bony evaluation and soft tissue evaluation:

1. Bony evaluation: evaluate height, width, and general shape of alveolar ridge and underlying basal bone; locate undercuts, neurovascular bundle, and concavities.
2. Soft tissue evaluation: lesions which require biopsy, attached mucosa, flabby tissue, frenum attachments.

The main objectives, goals or premises of preprosthetic oral soft tissue surgery are preparation and enhancement of denture seating area by providing optimal soft tissue conditions and deepening of flange region that increases resistance to displacement forces. Types of preprosthetic surgery can be classified in a number of ways. One method is to categorize the

surgery as resection, recontouring or augmentation of bony or soft tissue.

Increase of the occlusal load-bearing region and reduction of vertical alveolar bone resorption should be measured as a secondary purpose in performing reconstructive surgery.

Preoperative evaluation

Psychological evaluation

Preoperative preparation plays an important role for the success of procedures. Patients who require vestibuloplasty often complain about lack of functional chewing and loss of significant retention of their dentures. Preprosthetic surgery helps when the problem with retention is confined to aberrant intraoral anatomical topography. An initial interview would be very beneficial to understand whether the surgery would meet the patient's expectations. It might be possible to determine if patients have maladaptive behavior or neurotic anxiety that could not be altered with a surgical intervention. In such cases, referral to a psychologist would be logical.

Radiographs

Panoramic and cephalometric radiographs would be beneficial to determine vertical alveolar bone height in the mandibular symphysis and corpus regions: 15mm or more of mandibular body is considered to provide the desired vestibular sulcus after vestibuloplasty.

Benign soft tissue lesions

Hypertrophic/exophytic benign soft tissue lesions should be treated prior to flange deepening. Papillary hyperplasia, epulis fissuratum or chronic traumatic ulcers are common pathoses and must be managed several weeks before the vestibuloplasty operation. Cryotherapy, excision, electrocautery, and laser ablation may be techniques of choice.

Management of sharp mylohyoid ridges and genial tubercles

A sharp mylohyoid ridge requires reduction during lowering of the lingual sulcus, however osseous resorption generally occurs simultaneously after dissection of the mylohyoid muscles. Resection of mylohyoid ridges should not be performed unless prosthodontic consultation is obtained, since the undercuts of mylohyoid ridges provide noteworthy retention for dentures. In the same way, sharp genial tubercles usually atrophy after dissection of genio-glossal muscle. These tubercles should be recontoured 2 months before a graft-combined-sulcoplasty procedure is performed.

Surgical techniques

Ridge extension can be achieved by numerous techniques as appropriate for the maxillary and the mandibular ridges. Both local and systemic factors affect choice of the right surgical technique. The condition of recipient and donor sites, height and shape of the underlying alveolar bone, and amount of surgical dissection determine the worth of mucosal or split-thickness skin grafting.

After the Consensus Conference in Berlin, in 1983, the necessity of covering the periosteum was no longer discussed.³⁰ Covering the raw vestibular periosteal surface with a skin or mucosal graft and screw fixation was considered as the main objective. Otherwise, complete relapse could occur during secondary healing with scar contraction and epithelialization of the vestibular periosteum.^{4,31} Fast growing interest in implantology has restored the importance of the pedicled flap vestibuloplasties used in combination with implants.^{32,33}

Procedures for sulcular deepening are listed as below:

- With soft tissue grafts: mucosal graft, palatal graft, split-thickness skin graft, meshed skin or mucosal grafts.
- With pedicled mucosal grafts:
 - submucous vestibuloplasty;
 - crestally or inferiorly pedicled mucosal flaps.
- Secondary epithelialization.
- Mouth floor lowering with mylohyoid and genio-glossal muscle repositioning. No longer a recommended procedure.
- Mylohyoid repositioning.
- Creation of post-tuberosity (hamular) notch.
- Any appropriate combination of aforementioned procedures for the maxilla and mandible.

Free grafting techniques

Split-thickness skin grafting

A split-thickness skin graft, which is totally excised from its underlying blood supply, is composed of dermis and epidermis. Full-thickness skin grafts are composed of dermis and all the epidermis, which contains hair follicles, adnexal structures (sweat and sebaceous glands), and skin capillaries. Split-thickness skin grafts are subdivided according to thickness of the dermis: thin, intermediate or thick grafts.⁴ The thickness of grafts is determined by settings of a dermatome at 10 to 25 thousandths of an inch, transparency of a graft, and bleeding of the donor site. The thinner a skin graft, the more rapid vascularization and the more contracture occurs.⁴

Split-thickness skin grafts are mainly used in combination with a vestibuloplasty, and were used in lowering of the floor of the mouth in the past.³⁴⁻³⁷ Skin grafting for vestibular deepening was presented first by Reverdin in 1869.³⁸ Trauner and Pichler pub-

lished a detailed article revealing many aspects of this procedure in 1930.³⁹ Schuchardt demonstrated labiobuccal skin grafting without creating a skin-lined pouch in the vestibular sulcus.⁴⁰

Full-thickness skin grafts in the oral cavity are not as ideal as split-thickness skin grafts because they frequently display hair growth and keratinization, and patients are often disturbed by their color, taste and smell.⁴¹

Surgery starts with a mucosal incision from the left to the right molar region. The muscle fibers in the subcutaneous tissues are dissected from the periosteum and the mental nerves are protected. Supraperiosteal dissection must be limited to two thirds of the mental muscles in order to avoid chin ptosis.⁴² At least 5 mm of muscular pedicle (or 10 mm of soft tissue) should be kept intact to the periosteum to prevent sagging of the chin.⁴³

The skin donor sites are the hip⁴ and the retroauricular area.⁴⁴ Larger grafts can be obtained from the hip. Fixation of the grafts is performed by surgical stents, sutures, and fibrin sealants.⁴⁵ The skin is adhered to the inner side of the sterilized stent with wet cotton applicators. Tissue conditioner, such as Visco-Gel, can be combined with the stent to achieve proper sulcular shape and faster healing. The stent was secured in the past by circumpalatal or circummandibular wire fixation; however that concept was terminated after the Consensus Conference.³⁰ The stent is generally removed after 7–10 days. During that period, antibiotic cover should be provided and intraoral saline irrigation should be continued for 2 weeks.

Palatal grafts

The most preferred intraoral soft tissue graft is the palatal mucosa which heals perfectly.^{32,46} Palatal grafts can be used as an alternative to skin grafts. The graft can be harvested with a #15 blade or a mucotome. A graft is sutured on to the periosteum with resorbable sutures. Four to six strips of palatal graft have to be removed for a total vestibuloplasty. The fixation of a splint is done in the same manner as for split-thickness skin and mucosal grafts.⁴⁷

Local anesthetic is injected supraperiosteally to the donor sites. With a #15 blade, the incision lines are outlined and cuts are performed supraperiosteally. Lamina propria and submucous fatty tissues are dissected from each other with the #15 blade or dissecting scissors. After the graft is taken out, it is thinned by removing excessive fat tissue and minor salivary glands. The graft is rolled up in a saline-soaked gauze. The donor site is irrigated and any bleeding from the major palatal vessels is investigated. If such a complication is present, hemostatic agents, “stick-tie” ligation or electrocautery is often effective and provides satisfactory hemostasis. The graft must be fenestrated at two to three sites to allow regress of seroma or hematoma which may prevent adherence of the graft to the periosteum (Fig. 19.18).²⁰

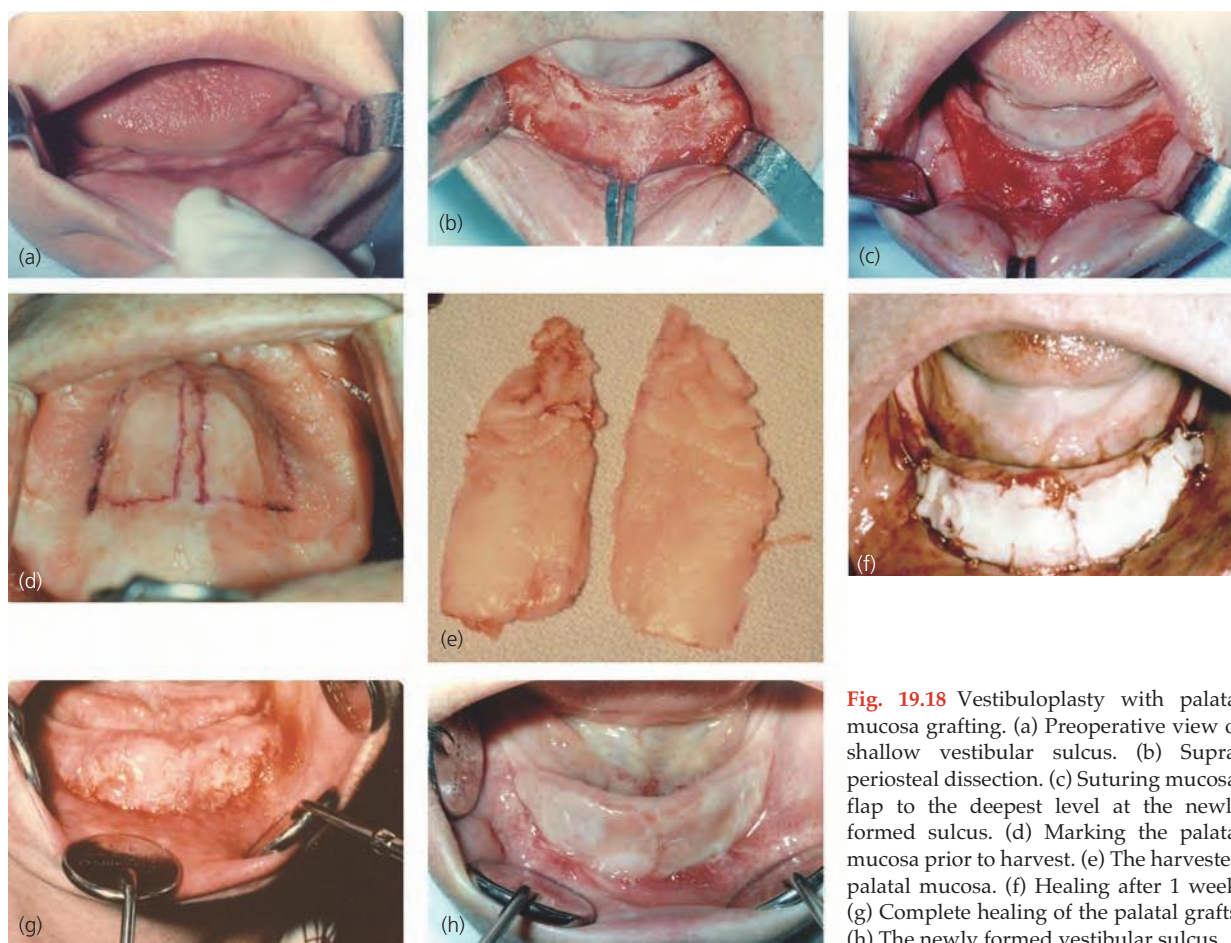


Fig. 19.18 Vestibuloplasty with palatal mucosa grafting. (a) Preoperative view of shallow vestibular sulcus. (b) Supra-periosteal dissection. (c) Suturing mucosal flap to the deepest level at the newly formed sulcus. (d) Marking the palatal mucosa prior to harvest. (e) The harvested palatal mucosa. (f) Healing after 1 week. (g) Complete healing of the palatal grafts. (h) The newly formed vestibular sulcus.

The advantages of intraoral palatal grafting are: (1) histologic similarity to adjacent intraoral soft tissues; (2) ease of harvesting; (3) lack of hair follicles or sweat glands; and (4) limited morbidity. The disadvantages of palatal mucous grafts are: (1) limited tissue; (2) bleeding from injured palatal vessels; and (3) donor side morbidity if dissection has gone too deep.⁴

Meshed skin and mucosal grafts

Meshing of split-thickness skin or mucosal grafts helps to cover large areas when donor sites are limited.⁴⁸ Meshing allows expansion of the graft up to nine times the surface area of the donor site. The slits in the meshed skin graft allow wound fluid to escape through the graft rather than accumulating beneath it and preventing adherence. Since introduction of machine-meshed skin grafts,⁴⁹ many other techniques and modifications have evolved to aid in the procedure.

After harvesting and thinning, the mucosal graft is meshed with a graft expander or manually. A common method of meshing a split-thickness skin graft is to make multiple cuts with a #15 blade to the graft laid on a hard, wooden board. The expansion slits heal by re-epithelialization and may contract significantly. When healed, the grafted site characteristically has a "crocodile skin" or "checkerboard" appearance.

Mucosal grafts

The donor site of mucosal grafts is the inside of the cheek. The submucosal fat and connective tissues are removed with scissors and tweezers. The cheek is closed primarily with a resorbable suture. The disadvantages of mucosal grafts are lack of attached mucosa that weakens resistance to infection and allows mucosal movement.⁵⁰

Pediced flaps

Kazanjian's vestibuloplasty

A transvestibular horizontal mucosal incision is made in the lower lip between the right and left premolars. The mucosa is dissected suprapariosteally to the alveolar crest, which is followed by suprapariosteal dissection of the mental muscles to the vestibular depth. The pedicled flap is sutured to the periosteum to the depth of the vestibular sulcus. The open wound in the vestibular sulcus and inner lip is left to heal by secondary epithelialization (Fig. 19.19). This method can be used with or without simultaneous insertion of dental implants.⁶ A novel modification⁵¹ was reported to eliminate formation of a V-shaped pocket in vestibular depth.

Although overcorrection has been recommended due to the relapsing tendency of Kazanjian's^{5,6} and Clark's vestibuloplasty,⁵² it is not always possible

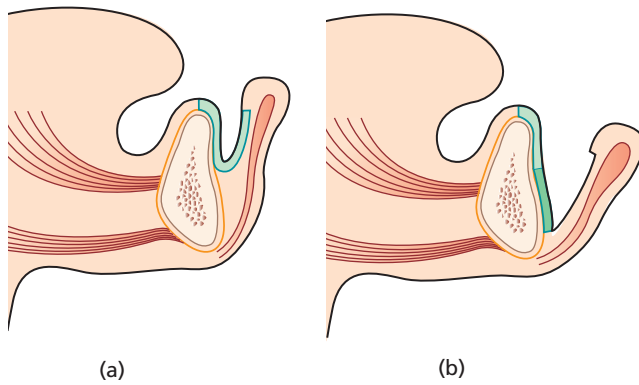


Fig. 19.19 Kazanjian's vestibuloplasty. (a) The crestal pedicled flap is raised from the inner side of the lip. (b) The flap is sutured over the periosteum to the deepest point of dissection.

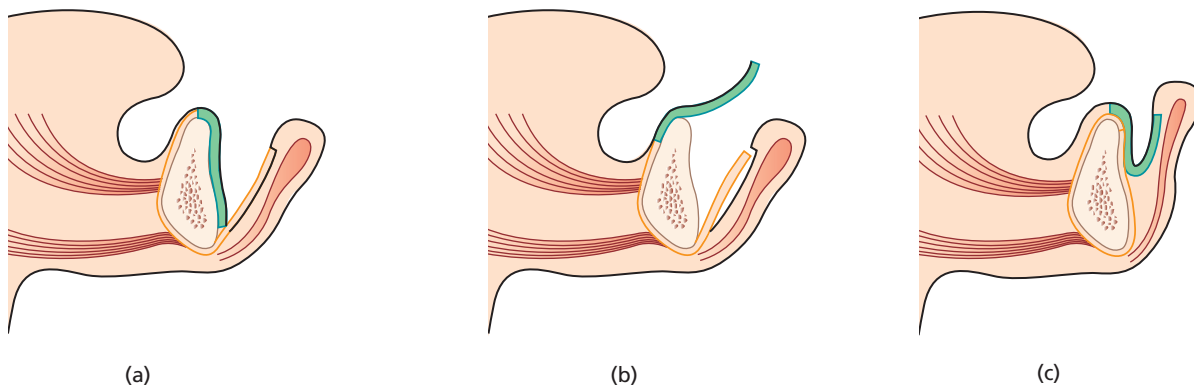


Fig. 19.20 Edlan's vestibuloplasty (lip switch technique). (a) The crestal pedicled flap is raised from the inner side of the lip. (b) The flap is sutured over the periosteum to the deepest point of dissection. (c) Final result after flap transpositioning.

to create a deep sulcus due to insufficient height of alveolar bone. Epithelial grafting of raw surfaces in the dissected vestibule decreases the secondary contracture and postoperative relapse during re-epithelialization.⁵³

Transpositional flap vestibuloplasty (lip switch or Edlan vestibuloplasty)

The lingual-based flap vestibuloplasty was first described by Kazanjian.⁵ The technique underwent a modification with covering the denuded region of lip that was left to secondary epithelization in the initial technique.⁵¹ This procedure is indicated primarily for the patients with insufficient mandibular vestibular depth and adequate lingual sulcus.⁵⁴

The standardized Edlan vestibuloplasty has an important role in preprosthetic surgery.²⁹ An incision is made through the mucosa from one premolar region to the inner lower lip and from there to the contralateral premolar region. The mucosa is prepared to the highest point of the alveolar ridge; the periosteum is incised at the alveolar ridge and prepared down to the required vestibular depth under direct vision of the mental nerve. The periosteum is sutured to the mucosal crest of the inner lip. The pedicled mucosal flap is sutured to the periosteum to the vestibular depth. This method is mainly used in combination with the insertion of implants (Fig. 19.20).

The lip switch technique could be combined with a surgical splint; however the patient's own denture could also serve as a retention splint if it is relined

with a soft tissue conditioner to obtain proper adaptation of vestibular sulcus. The disadvantages are scar contracture and unexpected relapses which occur in up to 50% of cases. Vertical resorption of the underlying alveolar bone is another complication with this procedure.^{4,31,55–57}

Submucous vestibuloplasty

Submucous vestibuloplasty was first described for the mandible; however the procedure was found to be quite unsuccessful.⁶ The submucous vestibuloplasty has been recommended for deepening the maxillary vestibule.⁸

Closed submucous vestibuloplasty technique (Obwegeser)

An incision is made at the midline of the maxillary arch and a layer above the periosteum and below the mucosa is dissected. The superficial plane is created anteroposteriorly with scissors until translucency of the buccal mucosa is observed. The length of the superficial dissection is carried out as far anteroposteriorly as the anatomy will allow. The secondary plane is formed with a periosteal elevator. The intermediate soft tissue is separated and moved superiorly (Fig. 19.21). Otherwise, wedge or complete excision could be performed. The thin mucosal flap is adapted directly over the periosteum and, with the help of a surgical stent, the flap is secured for a week. The

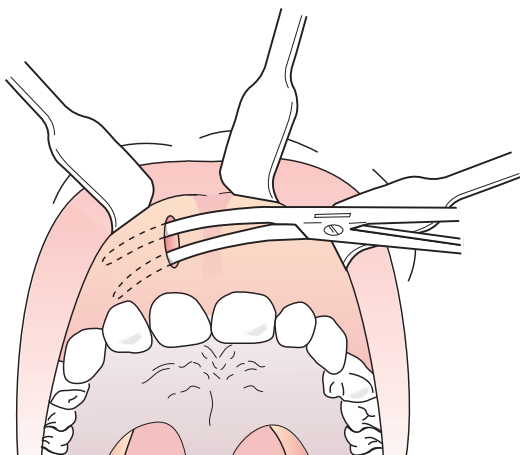


Fig. 19.21 Dissection of the superficial and the secondary deep plane is performed through a short vertical incision at the midline of maxillary vestibule.



Fig. 19.22 A surgical stent is secured to the upper arch with two titanium screws for 7–10 days.

sharp anterior nasal spine can be also smoothed with a rongeur or a bur. A figure-of-eight suture is generally made to elevate mucosa towards the nasal spine which is very effective in increasing the depth of the vestibule. Subnasal or cheek fullness, obtained by superior push of the intermediate submucosal tissue, satisfies the patient esthetically. A surgical stent, secured with titanium screws, could also be used to cover the operated region (Fig. 19.22).

Secondary epithelialization

Secondary epithelialization is an easy technique to accomplish though rapid and unpredicted relapses often prevent surgeons using this method alone. It could be performed when covering mucosa has an irregular surface and unhealthy status, such as presence of inflamed, granulation, ulcerative, and exophytic soft tissue lesions. The lesions could be removed several weeks before or at the same time as the secondary epithelialization vestibuloplasty.⁴ Secondary epithelialization was described by Kazanjian in 1935⁶ and Clark in 1953.⁵²

A crestal incision is made at the junction of the free and attached mucosa and dissection is deepened to the desired level. The muscle and submucous fat are dissected with scissors. The suprapariosteal surface should be cleansed as much as possible to prevent formation of reactive aberrant granulation in the early healing period (Fig. 19.23). The mucosal flap is sutured down to the junction of the newly formed sulcus and the periosteum. A surgical stent lined with a tissue conditioner could be modified in order to prevent direct pressure of the stent over the raw periosteal surface. Actually, the Clark's technique is preferred to Kazanjian's sulcoplasty since the raw periosteal surface in the latter results in more contracture.

Lowering of mouth floor

As the alveolar bone is resorbed, the attachments of the mylohyoid and genioglossus muscles may interfere with the lingual aspect of the denture causing an inadequate depth for prosthesis. Lowering the mouth floor can be done separately for each anterior and posterior region; however the lingual sulcus also could be deepened at once. This is no longer a recommended procedure.

Anterior lowering of mouth floor

The superficial and lateral fibers of genioglossal muscle are dissected from the genial tubercle and secured via percutaneous sutures to the submental skin fold or labial vestibule.

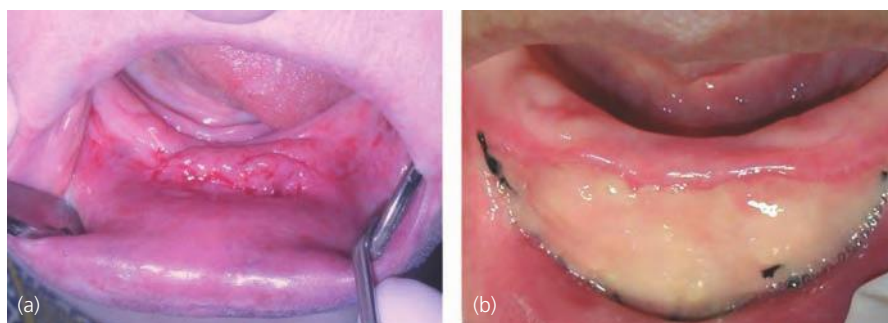


Fig. 19.23 (a) Preoperative intraoral view of a patient with a wide epulis fissuratum and shallow vestibular sulcus. (b) Ten days postoperative. The healing occurs by secondary epithelialization.

Posterior lowering of mouth floor

Procedures lowering the mouth floor were described by Trauner, Obwegeser, and Macintosh. The mandibular body should be at least 15mm in height. The main objective is to eliminate the rising effect of the mylohyoid muscle on the lingual flange. An incision is made at lingual sulcus parallel to the alveolar wall. Supraperiosteal dissection is progressed to the mylohyoid ridge and the mylohyoid muscle is separated. After the buccal flap is prepared, lingual and buccal flaps are secured with a Reverdin needle at the buccal side. If only lowering of mouth floor is performed, the flaps are anchored to a new lingual sulcus with non-resorbable percutaneous sutures at the skin crease of submandibular region. The periosteal surface is grafted with split-thickness skin or palatal grafts which are protected with a surgical stent.

Such a major procedure in geriatric patients is no longer recommended in the current practice of oral and maxillofacial surgery. Contemporary management of edentulous patients covers dental implant surgery with various prosthetic solutions. These options are more reasonable and effective for solving retention problems in extensively resorbed lower jaws.

Adjunctive and supportive surgical techniques

With the intense use of implants, bony augmentation and reconstructive soft tissue techniques have improved. Instead of simple functional rehabilitation, full esthetic and functional requirements of the patient are met by trying to return to the original quality and quantity of bone and soft tissues. In order to succeed in bone augmentation, there should be sufficient soft tissue to cover the new bone and the implant should be supported by at least 3mm of attached keratinized gingiva. That is why careful handling and management of the soft tissues during surgery determines the final outcome of the whole procedure.

Socket preservation

Micro or macro fractures occurring during dental extraction are avoided as much as possible when the alveolar socket is to be preserved following tooth removal. Conventional extraction methods may be traumatic and cause alveolar bone destruction in the healing period. In order to avoid postextraction bone and soft tissue loss, tooth removal may be performed by periosteal (Fig. 19.24). This tiny and sharp instrument helps to cut the periodontal ligament and forces the roots to the surface. The use of this method enables the practitioner to insert an implant at the same time as tooth removal, as the socket is not widened and/or fractured by conventional extraction methods.

Immediate implant insertion at the time of tooth removal was thought to be a socket preservation

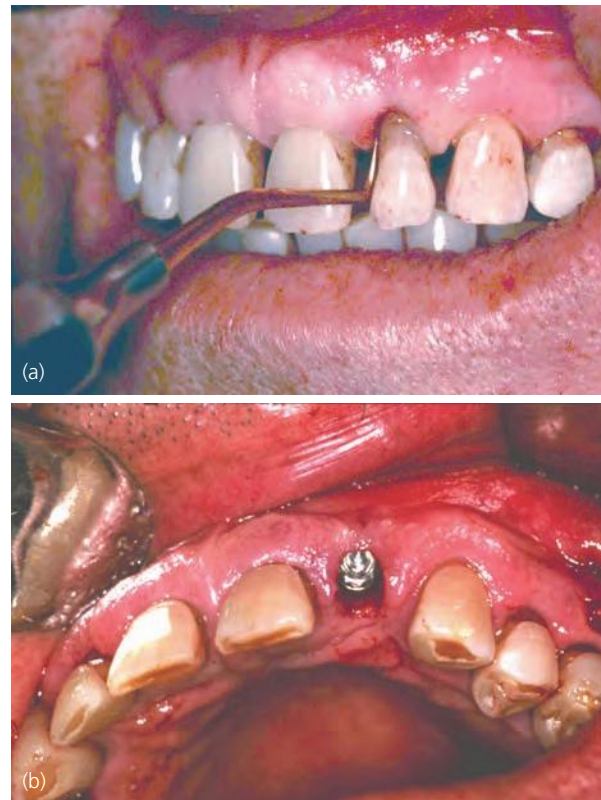


Fig. 19.24 Socket preservation. (a) Removal of the maxillary first molar by periosteal. (b) Immediate implant insertion.

method until recently. However, today it has been shown that bone resorption continues even after immediate implant insertion. Other methods such as filling the extraction socket with biomaterials and covering with membrane are not very commonly used techniques as they are expensive and results are not always satisfactory.

Other well known methods to preserve the socket are removal of roots after separating them by burs or removal of the roots following drilling the roots from inside, starting from the pulp chamber.

If a periodontally compromised tooth is removed, resultant alveolar width and height are mostly less than ideal for esthetic and functional reconstruction. If the residual defect is mild there are a few options to manage this problem; one of them is orthodontic tooth eruption, and the other one is tooth removal followed by alveolar guided bone regeneration (GBR) and/or augmentation.^{23,58}

Orthodontic tooth eruption

Orthodontic tooth movement is a technique that improves the dimensions of hard and soft tissues surrounding periodontally compromised teeth.^{59,60} This technique, also known as forced eruption, is a non-surgical treatment option that facilitates hard and soft tissue remodeling.⁶¹ Bone volume is increased due to the tension applied to the periodontal ligament by orthodontic forces. New bone is deposited by osteoblastic activity where the periodontal attachment

exists. The coronal shift of the tooth causes migration of the soft tissue and the bone in the direction of the movement.⁶¹ Orthodontic tooth eruption is also used to potentiate bone development at the implant site.^{62,63} Although it is a long-term therapy, forced eruption can result in surgical augmentation procedures being avoided.

First teeth should be treated endodontically and filled with calcium hydroxide-based root canal sealer. Direct bonding tubes are then bonded further gingivally, and occlusal reduction is performed to allow extrusion of those teeth. The patient should be examined every 2 weeks to monitor progress and to reduce the occlusal surface of the teeth being extruded.

Implant site development using forced eruption is an alternative technique that provides hard and soft tissue improvement.⁶⁴

Guided bone regeneration

GBR with or without bone graft substitutes is a very well known and commonly applied technique for periodontal defect repair and for mild alveolar defects. Although results are mostly favorable, volume gained by guided tissue regeneration is limited and the high cost of membrane and bone substitutes is another disadvantage.

Ridge splitting

Ridge splitting is another option for augmentation of horizontal defects, especially used in triangular, V-shaped crests with adequate length.⁶⁵ However U-shaped crests cannot be reconstructed by this technique.⁶⁶

Bone grafting

Onlay bone grafts are well protected since grafts are not disturbed with postoperative dehiscences and infection. In moderate defects bone grafting is the widely applied technique for ridge reconstruction. Intraoral bone grafts harvested from the ramus and chin area are closer to the operation area and have low morbidity. Additionally membranous bone grafts may be preferred because they have less resorption than endochondral bone grafts due to early revascularization, better potential for incorporation in the maxillofacial region because of a biochemical similarity, and greater inductive capacity because of a higher concentration of bone morphogenic proteins and growth factors. Cortical bone grafts maintain their volumes significantly better than cancellous bone grafts.

The mandibular ramus and chin are the most favorable donor sites.⁶⁷ Larger amounts of bone volume could be obtained from the ramus area and with less complications. Numb incisors and gum, donor site morbidity, and dehiscence are the main compli-

cations reported after symphysis graft harvesting. Augmentation of alveolar deficiencies both with autogenous bone grafts and alveolar distraction osteogenesis (ADO) are the most frequently used techniques in moderate defects.⁶⁷ However, despite various publications reporting favorable results of these two methods, controversies still exist about the ideal treatment modality. ADO offers significantly better results when the quantity and quality of new alveolar soft tissue are considered.⁶⁸

Alveolar distraction osteogenesis

In larger defects, ADO is an alternative treatment for bone and soft tissue reconstruction.^{69,70} This process is based on the concept of bone distraction along a vector that is transverse to the long axis of the bone, which results in bone formation.^{69,70} It was later applied to the human mandible, and more recent clinical reports have shown that ADO is effective for treating severe forms of alveolar ridge atrophy.⁶⁹ A primary advantage of distraction osteogenesis is that there is no need for additional surgery at the donor site. Another benefit is the coordinated lengthening of the bone and associated soft tissues.^{70,71}

In all these techniques (forced eruption, GBR, bone grafting, ADO, and ridge splitting) the bony volume is increased. In order to obtain successful results both hard and soft tissues should be lengthened and widened. However if open techniques are used, the vestibular depth is lowered and the amount of attached and keratinized gingiva is diminished. The defective alveolus mostly remains uncovered with deficient soft tissue pedicle which is also handicapped with anatomical landmarks limiting the extension of flaps. This may result in tension on the soft tissues during closure and may end with dehiscence and graft, bone substitute, and membrane removal. ADO is the best technique for soft tissue lengthening in a defective alveolus. Especially in moderate and severe defects, this technique enables restoration of soft tissues with desired quality and volume (Fig. 19.25).

Lasers in preprosthetic surgery

LASER is an acronym for light amplification of stimulated emission of radiation. Laser application in maxillofacial surgery practice covers control of leukoplakia, oral malignancy, preprosthetic or periodontal surgery, temporomandibular joint, uvulopalatotomy, and cosmetic procedures.

Clinical lasers are of two types; soft lasers are essentially an aid to healing with relatively few rigorous studies available to support their use. Surgical hard lasers, however, can cut both hard and soft tissues and replace the scalpel and drill in many areas. From initial experiments with the ruby laser most clinicians are using argon, CO₂ and now Nd:YAG

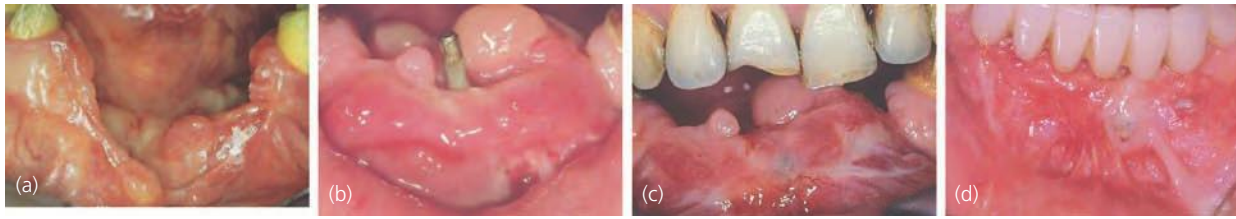


Fig. 19.25 Pre- and postoperative alveolar distraction osteogenesis of the anterior mandible. Note the significant augmentation of the defective mandible. Combination of bone grafts and membranes in similar cases would not provide the same outcome due to difficulty of obtaining soft tissue flap closure. Note that besides bone lengthening, the soft tissues are also lengthened with healthy keratinized and attached gingiva over the alveolar crest.

systems. Both the CO₂ and Nd:YAG lasers can be used for intraoral soft tissue surgeries.⁷²

The advantages of lasers include a relatively bloodless surgical and postsurgical course, minimal swelling and scarring, coagulation, vaporization, and cutting, minimal or no suturing, reduction in surgical time, and, in a majority of cases, much less or no post-surgical pain.

CO₂ laser

Since its development in the early 1960s, the CO₂ laser has been rapidly introduced into medical research and almost every dental and medical surgical specialty. In dental surgery it is mainly used for oral soft tissue applications. CO₂ laser light can be focused for cutting purposes or defocused to secure hemostasis. This laser can also be used to vaporize neoplasms.⁷³

The CO₂ laser offers the dental surgeon an improved alternative to the scalpel due to the advantages of relatively bloodless surgery, decreased postoperative discomfort, minimal swelling and scarring, and the laser's ability to coagulate, vaporize, or cut.⁷⁴ The laser tip does not touch the tissue, thus good access and visibility are obtained. As the CO₂ laser eliminates bleeding, placing sutures is unnecessary. In comparison with traditional electric scalpel, resective operations done with CO₂ laser are 10 times more precise.

The basic surgical procedures that can be completed efficiently with CO₂ laser radiation include:⁷³

- biopsy – tissue cutting and removal;
- removal of mild and early lesions: fibroma, papilloma, coccus, nicotine stomatitis, Wilson's lichen, erosion lichen, epulis, common warts;
- removal of malignant lesions;
- removal of excessive keratosis;
- frenectomy: along lips (along maxilla middle line); along tongue (ankyloglossia);
- gingivectomy, removal of hypertrophies (e.g. caused by dilantin, procordica, ciclosporin, idiopathic, cardizem);
- gingivoplasty;
- reduction of soft tissue tuberosity;
- operculectomy;

- removal of aphthae;
- improvement of hygienic conditions around crowns and bridges: elimination of 'swollen' lips, irregular lip line, removal of overgrown tissue;
- coagulation in transplant collecting sites;
- removal of vascular lesions: hemangioma, pyogenic granuloma;
- treatment of patients suffering from blood clotting disorders: hemophilia, thrombocytopenia, Sturge-Weber syndrome;
- internal ear vestibule shaping;
- implants, stage II, soft tissue removal.

Nd:YAG laser (neodymium)

The first dental laser based on an Nd:YAG engine provides handpieces of similar size to conventional instrumentation and, being fed by a fiber-optic 'cable', has the flexibility for intraoral use that the CO₂ lasers, widely used in oral surgery, lack.^{72,73} The Nd:YAG was the first laser used for its coagulating effect in surgical procedures in the oral cavity. It is used to excise pathologic lesions or for its sterilizing or sealing effect at lower irradiation doses. Since its absorption is 10000 times slower than Er:YAG it cannot be used for hard tissue procedures. The Nd:YAG laser is absorbed by hemoglobin and chromophores (melanin and other proteins). A photothermal effect occurs; after absorption of the laser radiation it is converted to heat, resulting in vaporization and tissue ablation.

The main advantages of Nd:YAG laser application in dentistry are: (1) dry operation field; (2) satisfactory results of postoperative treatment; (3) low invasiveness; (4) high precision; (5) procedure safety; (6) lower susceptibility to caries; (7) convenience for patients.

Parameters for Nd:YAG laser surgery could be chosen as follows:

- Implant release after surgery: power 2.5W, frequency 20Hz.
- Reduction of bleeding: power 3–5W, frequency 50Hz.
- Removal of fibroma/hyperplasia: power 2W, frequency 30Hz.
- Gingivectomy: power 3–8W, frequency 50Hz.

Er:YAG soft tissue surgery (erbium)

With a special surgical handpiece, the Er:YAG laser could also be used conveniently in oral surgery (Fig. 19.26). Frenectomy, gingivectomy, and treatment of stomatitis could be accomplished. Small and deep cuts are observed when a procedure is performed by Er:YAG irradiation. Shallow and wider cuts are obtained with defocused irradiation. Wound healing with minimal damage zone without carbonization takes a week. In small lesions, local anesthesia is usually not required. The treatment parameters are:

- Energy: 50–350 mJ.
- Frequency: 20–30 Hz.
- Pulse width: VSP (very short pulse), LP (long pulse).

Fibromas in the oral region could also be easily treated with Er:YAG laser ablation. The procedure is the same described for soft tissue procedures; however energy requirement differs and should be adjusted to 200 mJ with LP pulse width.

Removal of superficial oral lesions (leukoplakia, lichen planus, keratosis)

Er:YAG is an ideal laser type for ablation of white lesions on the buccal mucosa, palate, and mouth floor, since underlying tissue is not damaged. The lesion is peeled layer by layer like an onion to the required depth. Most cases need two or three appointments after which ablated lesions do not recur even after years.

- Energy: 800 mJ.
- Frequency: 8–10 Hz.
- Pulse width: SP (short pulse).

Emerging technology and the future

Tissue engineering, the science of growing living human tissue for transplantation, opens new perspectives in medical care and will have a positive influence in the field of preprosthetic surgery. Cell culture technology, originally described for cultured skin and mucosal grafts, has opened a new era in the field of oral reconstructive surgery.⁷⁵ The major advantage of cell culture is the expansion of a small biopsy specimen into transplantable mucosal tissue by two to three orders of magnitude within a few weeks. Oral mucosa substitutes are classified into three types:

1. Sheets of cultured autologous keratinocytes.
2. An artificial dermis composed of collagen and/or glycosaminoglycans.
3. A composite oral mucosa, containing both an epithelial and dermal layer.⁷⁶

De Luca transplanted cultured palatal mucosa to replace gingival mucosa and demonstrated that 4 months later the grafts had formed a well differentiated keratinizing mucosa similar to palatal mucosa *in situ*.⁷⁷ Cultured autografts were successfully used instead of split-thickness skin grafts to free the tongue after malignant tumor resection.⁷⁶ Although there were good clinical results with the tissue-engineered mucosa grafts in most of the cases,⁷⁸ some wound shrinkage was observed. This phenomenon might occur because cultured mucosa grafts consist of only a thin layer of keratinocytes without any submucosal connective tissue.⁷⁵

Two skin substitute methods have been frequently used in oral and maxillofacial surgery. One is a mucosal epithelial sheet fabricated by the technique developed by Rheinwald⁷⁹ who described a method for

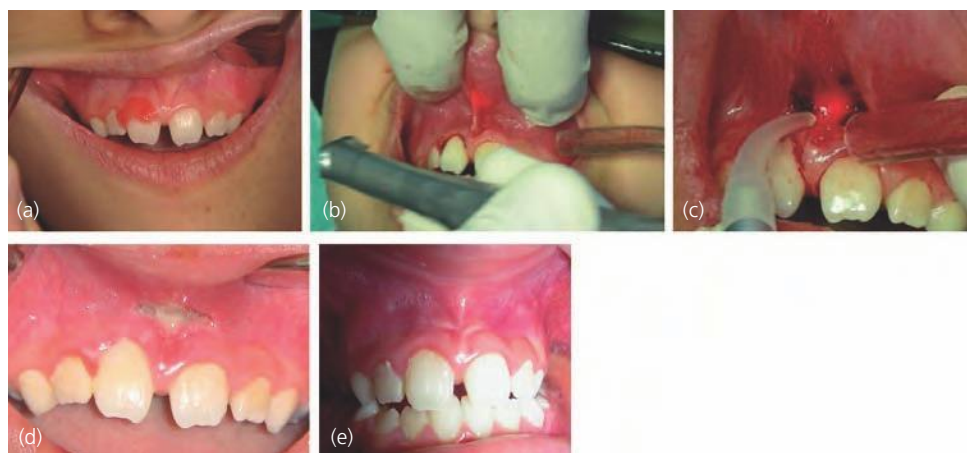


Fig. 19.26 Frenectomy performed with Er:YAG laser and Nd:YAG lasers. (a) Preoperative view of the labial frenum and the papilloma. (b) Frenectomy by Er:YAG surgery. (c) Coagulation of the operative field by Nd:YAG irradiation. (d) Early postoperative healing at day 3. (e) The final result.

serial cultivation of human epidermal keratinocytes. A second skin substitute technique uses a bi-layered artificial dermis that consists of collagen glycosaminoglycan/silastic sheet available at the time of surgery.⁸⁰ Its major disadvantages are difficulty with suturing and its susceptibility to infection.⁸¹ A dermal replacement (Dermagraft) can also be used instead of skin grafting.⁸²

Studies on production of *ex vivo* intraoral skin/mucosal graft gave promising results,⁸³ however the complexity of the procedure, time, and cost are questioned when simple and fast autogenous grafting techniques are possible. Absence of donor site morbidity should be considered as the most advantageous point of *ex vivo* produced grafts.

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Part 3: **Implant Surgery**

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Chapter 20

Implantology

Lars Rasmusson and Lars Sennerby

This chapter includes basic knowledge of osseointegration with a focus on the dynamics in tissue reactions following implant surgery. Indications and contraindications for implant treatment are discussed as is the importance of clinical research. Primary and secondary stability as well as loading effects on the bone tissue are analyzed and evaluated. The importance of careful maintenance procedures is emphasized. The difference in treating adolescents and children with oral implants is included. Finally the authors include a section on guided implant surgery technique and conclude that long-term clinical data on modern implant systems on the market today have in general shown high survival rates and minimal bone loss around the implants.

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Evidence-based oral implant therapy was presented to the dental community in the early 1980s.¹ Foreign materials have, however, long been used to replace missing teeth. For example, subperiosteal and blade implants had been used for some decades when pure titanium screw-shaped implants were introduced by the experimental and clinical work of P-I Brånemark.² Today, the use of osseointegrated oral implants is considered to be standard procedure for the treat-

ment of total and partial edentulism. Long-term clinical data on modern implant systems on the market today have in general shown high survival rates and minimal marginal bone loss around the implants.³ However, critical reviews of the scientific literature have concluded that some, often low-price systems, are not yet sufficiently documented in long-term clinical trials.

One prerequisite for successful anchorage and long-term function of titanium implants is that a sufficient volume of healthy bone is available in order to house adequate numbers and sizes of implants. The density of the bone is also a determinant of implant success, since failure is more common in bone with low mineral content.

Biological principles behind osseointegration

Implant stability and loading considerations

In the early days of implant dentistry, the use of osseointegrated implants always included a healing period of typically 3–6 months from placement to prosthetic treatment.⁴ The formation of direct bone-implant contacts was considered a prerequisite before loading could be commenced (Fig. 20.1). Today, immediate/early loading of dental implants is a clinical reality and numerous clinical studies have demonstrated as good results as those previously reported for two-stage implants.⁵ This has changed the understanding of what factors are important for a successful clinical outcome. In the early days, focus was mainly paid to the osseointegration process *per se*, whilst today a general stability concept is discussed where bone healing is one of several important factors.⁶

The clinical manifestation of a successful dental implant is the absence of mobility. Thus, achievement and maintenance of implant stability are prerequisites for a successful clinical outcome with dental implants.



Fig. 20.1 Light micrograph of an osseointegrated implant, retrieved from the posterior mandible of a patient 6 months after placement. The implant is surrounded by cortical and trabecular bone.



Fig. 20.2 Light micrograph of a clinically retrieved implant showing dense cortical bone in contact with the implant surface and with signs of ongoing remodeling.

The main determinants of implant stability are: (1) the mechanical properties of the bone tissue at the implant site and (2) how well the implant is engaged with that bone tissue.⁷ The first factor is determined by the composition of the bone at the implant site and is influenced by stage of healing, since soft trabecular

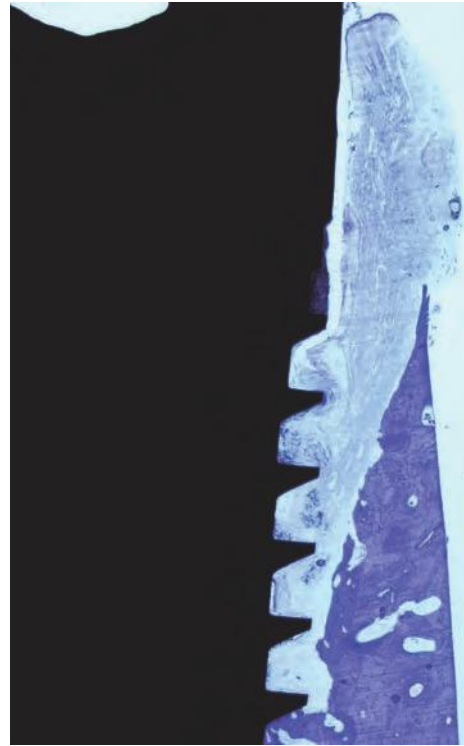


Fig. 20.3 Light micrograph of a clinically failed and retrieved implant. The implant is separated from the bone by a layer of fibrous scar tissue.

bone seems to be transformed to dense cortical bone near the implant surface. The second factor is influenced by the surgical technique, the design of the implant, and the osseointegration process. Successful healing results in bone formation that reinforces the interface zone and forms bridges and a direct contact between the implant surface and the surrounding bone (Fig. 20.2). Unsuccessful healing results in formation of an interface fibrous scar tissue (Fig. 20.3), which can be caused by infection or mobility of the implant after installation.

However, a clinically stable and successful implant also shows a certain degree of mobility on the micro-scale when a load is applied. For instance, if applying a lateral load (bending) to an implant in bone, the implant will be displaced but will return to its original position as soon as the load is removed (Fig. 20.4). Thus, a stable implant can display different degrees of stability, i.e. different degrees of displacement or resistance to load, depending on factors relating to the bone, the surgical technique, and the implant design. During clinical function, loading will be applied in axial, lateral, and rotational directions (Fig. 20.5). Furthermore, axial loads can be in push-in or pull-out directions. Lateral loads can principally be applied from any direction 360° around the implant. Rotational loading can either be clockwise or anticlockwise. Thus, the outcome of implant stability analysis depends greatly on the type of test used and in which direction the load is applied. In essence, stability measurements in bending give information about the stiffness of the implant–bone system, while

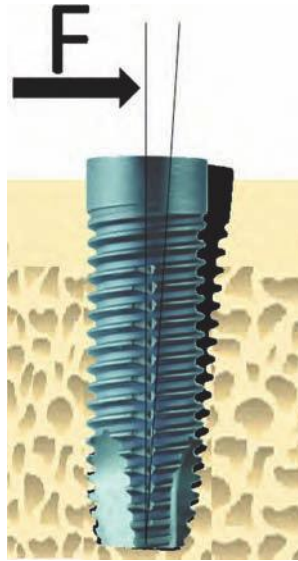


Fig. 20.4 Schematic showing displacement of an osseointegrated implant in the bone bed due to application of a lateral load.

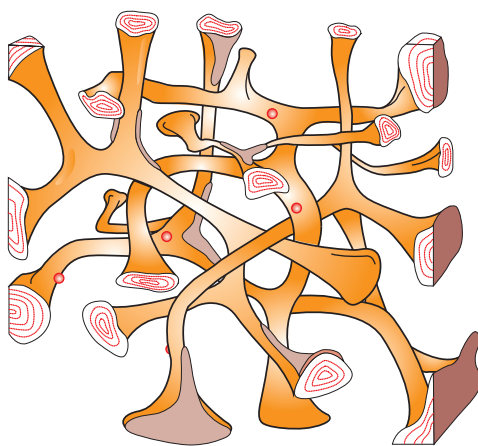


Fig. 20.5 An implant is subjected to loads in axial, lateral, and rotational directions.

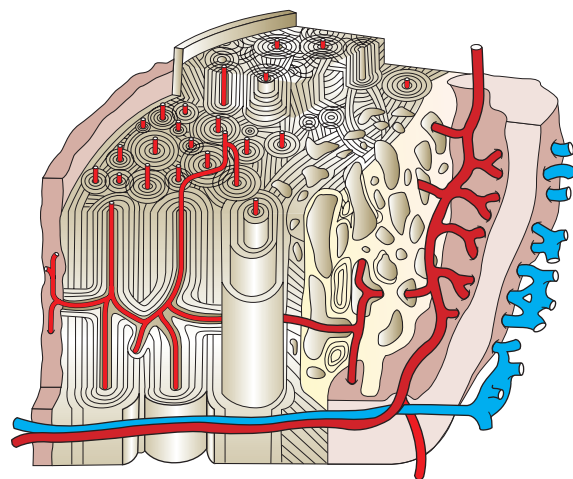
application of shear forces, with for instance a reverse torque test, measures the strength of the interface. This means that a newly placed implant can show a high degree of lateral stability but is easily removed when applying reverse torque since bone has not been formed and interlocked with the implant surface. With time, bone formation will lead to an increased interlocking with the implant surface and an increased strength of the interface, whilst the lateral stability may be unaffected. Since most implants will be connected with a framework, reverse torque tests are probably less relevant than measurements of lateral stability.

Primary stability

Implant stability is the result of contact between the implant surface and surrounding bone tissue. The degree of primary stability after installation depends on factors related to the bone, surgical technique, and implant design. The biomechanical properties of bone are determined by the ratio of cortical and trabecular bone at an implant site. Cortical bone is built up of densely packed mineralized lamellae, whilst trabecular bone is porous in structure and contains more soft tissue components than mineralized tissue (Fig. 20.6). For this reason cortical bone is 10–20 times stiffer than trabecular bone and provides a better support for an implant. The surgical technique can influence implant stability depending on the choice of drill diameters, depth of preparation, and whether pretapping is used or not. The implant itself also has impact on stability depending on its geometrical features. In a human cadaver study, it was demonstrated that bone density and implant design are important factors that influence primary stability as measured by resonance frequency analysis.⁸ They also found that 1° tapering of an implant dramatically increased the primary stability in poor bone. In continuing work, it was shown that primary stability also is influenced by drill diam-



(a)



(b)

Fig. 20.6 (a) Schematic of trabecular bone. One unit of trabecular bone tissue is mineralized to 40–60%. (b) Schematic showing cortical bone. One unit of cortical bone tissue is mineralized to 90–95%.

eter and whether pretapping is used or not.⁹ In essence, thinner drill diameters, omitting pretapping, and the use of a tapered implant will result in higher primary stability.

Secondary stability

After implant placement the bone tissue will respond to the surgical trauma, which with time results in: (1) a change of the cortical/trabecular bone ratio and (2) an increasing degree of bone–implant contact. The time needed for completion of bone formation and remodeling is in the range of 12–18 months. The impact of the degree of bone–implant contact for secondary implant stability is not known in detail, although it is generally anticipated that more bone contact means better implant stability. However, the change of the trabecular network into a more cortical bone in relation to the implant surface is probably more important. This means that the biomechanical properties of trabecular bone improve with time which leads to greater stability of the implant

(Fig. 20.7).¹⁰ Cortical bone already has favorable properties from an implant stability point of view. Therefore, histological changes of cortical bone will not necessarily lead to an increase in secondary stability (Fig. 20.8).¹¹

The time needed to achieve sufficient implant stability greatly depends on the density of the bone and thereby the primary stability, i.e. an implant with low primary stability needs a long period of healing whilst an implant with high stability needs only short or no healing. The host's ability to maintain and to increase the primary implant stability is also determined by the healing and remodeling capacity, which in turn is influenced by endogenous and exogenous factors such as health, the use of drugs, smoking, irradiation, etc.¹²

Maintenance of stability

When a crown/bridge or an overdenture has been connected to the implants, the conveyed loads and stresses will have an influence on the bone physiolo-

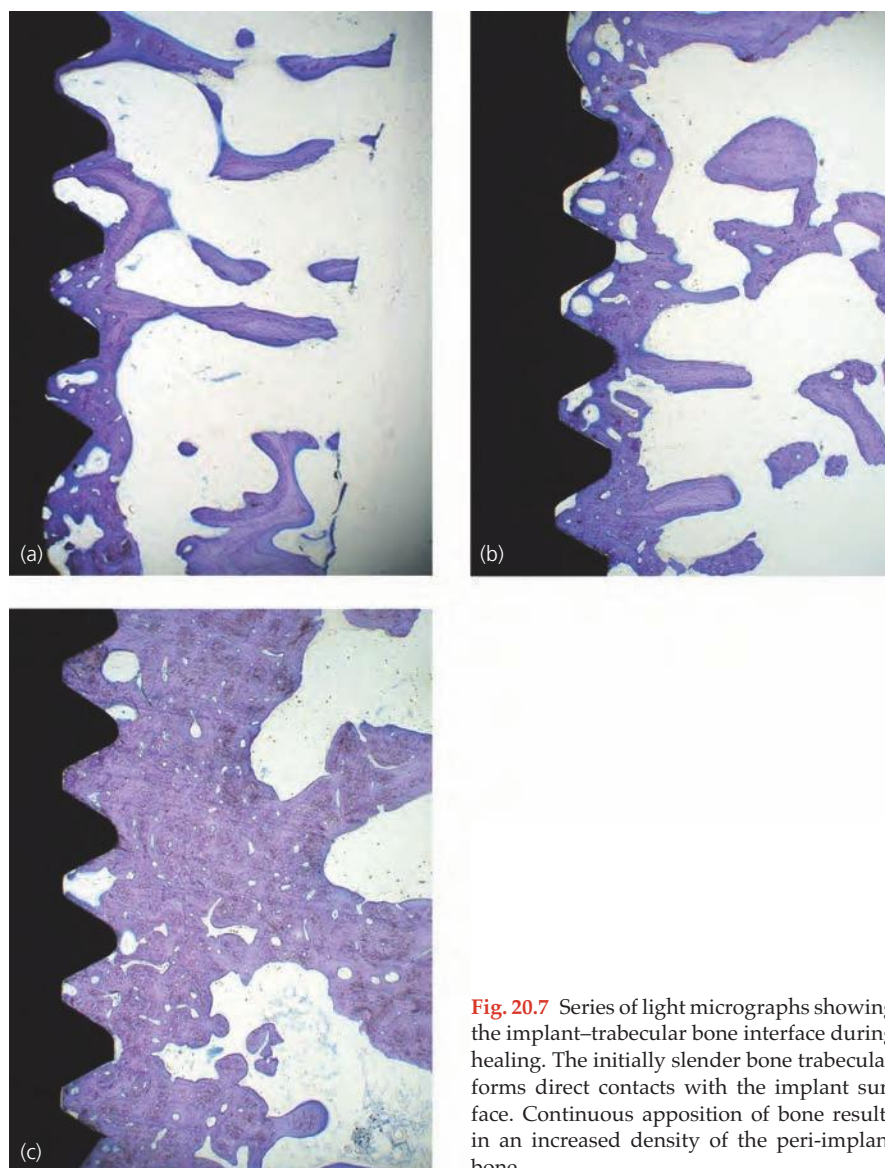


Fig. 20.7 Series of light micrographs showing the implant–trabecular bone interface during healing. The initially slender bone trabeculae forms direct contacts with the implant surface. Continuous apposition of bone results in an increased density of the peri-implant bone.

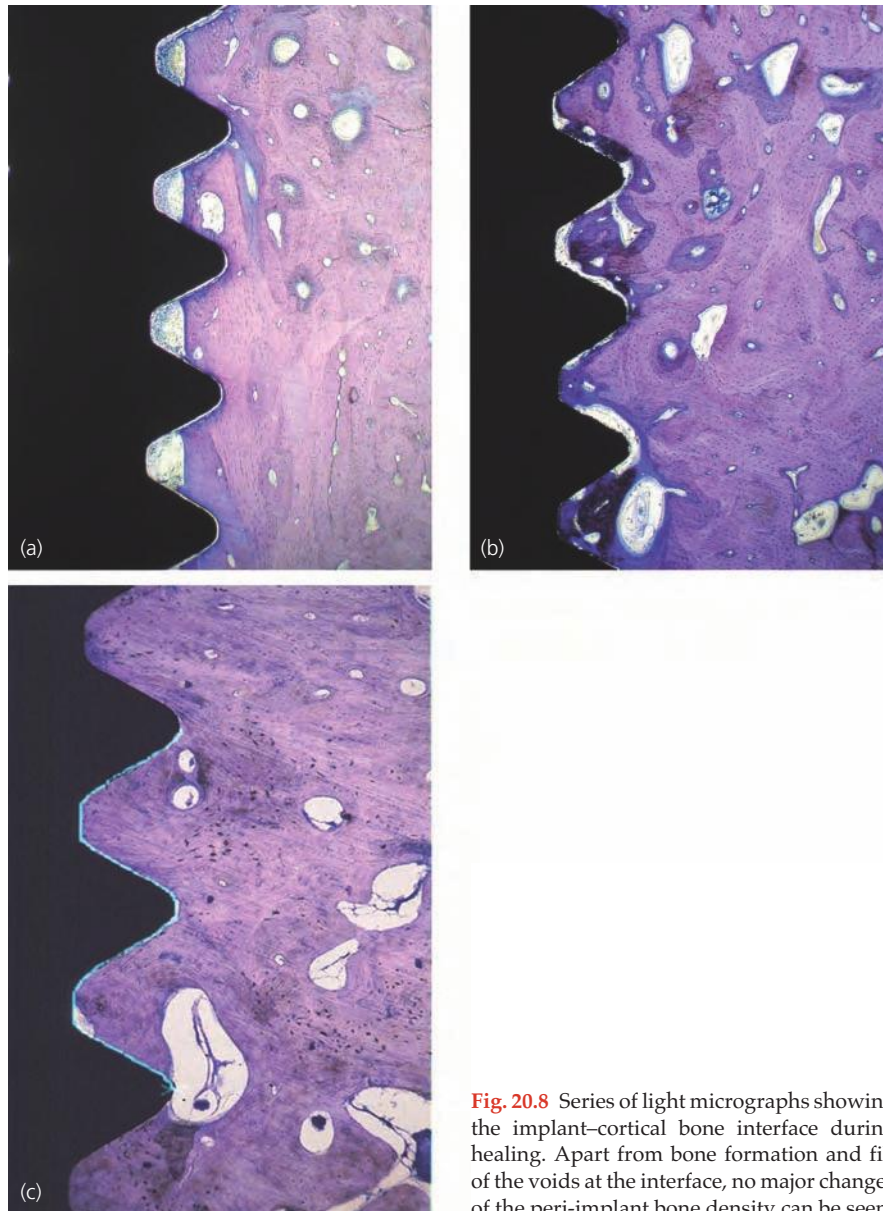


Fig. 20.8 Series of light micrographs showing the implant–cortical bone interface during healing. Apart from bone formation and fill of the voids at the interface, no major changes of the peri-implant bone density can be seen.

gy. In the early phase, the implant–bone system will be loaded while bone formation and remodeling induced by the surgical placement are still ongoing. It is reasonable to suggest that the bone tissue is more sensitive at this stage than after completed healing as most failures occur during the first year of loading. If the loads are excessive there is an obvious risk that this may lead to resorption, decrease of stability, and eventual loss of the implant. If the loads are within physiological limits it is probable that loading may stimulate further remodeling and adaptation of the bone to the present load situation. Overload has to be looked upon as a relative parameter since this describes an imbalance between loads and the degree of implant stability. In other words, for a given load the overload threshold is lower for an implant with low stability than for one with high stability.

Bone tissue responses to implants

The bone healing process around a dental implant resembles that of normal bone healing.¹³ The surgical trauma created during the insertion of an implant initiates an immediate and preprogrammed healing response at the bone–implant interface. This process involves the formation of a blood clot containing erythrocytes and biologically potent cells such as leukocytes and trombocytes. Cytokines and factors from these cells, the coagulation process, and injured tissues act as chemotactic stimuli on leukocytes and other cell types. The fibrin network within the blood clot provides an important scaffold for migrating cells involved in the formation of new tissues, such as vessels, extracellular matrix, and bone. Mesenchymal cells in the granulation tissue will differentiate to pre-

osteoblasts and subsequently osteoblasts and start to produce immature woven bone (Fig. 20.9). As will be described below, if a certain surface topography is present, new bone formation will occur directly at the implant surface (Fig. 20.10). Bone formation is also seen at the pre-existing bone surfaces facing the implant, probably as a result of activation of so-called resting cells and by populations of newly differentiated stem cells (Fig. 20.11). With time the new bone from adjacent bone surfaces will reach the implant surface and create bone–implant contacts and fuse with the bone initially formed in the granulation tissue near or at the implant surface. A rapid increase of new bone area and degree of bone–implant contact is seen during the early healing period (Fig. 20.12). Bone regeneration occurs in two stages and this is also the case around dental implants. The immature woven bone is replaced by mature lamellar bone through a

remodeling process. This is done by bone metabolizing units (BMUs), which contain osteoclasts, which resorb the woven bone, followed by a seam of osteoblasts, which lay down new layers of bone (Fig. 20.13). It is anticipated that the early bone formation takes about 3–4 months, whilst the remodeling process of the repair may take another 9–12 months in humans. However, physiological remodeling is a continuous process since it is part of the calcium metabolism system (Fig. 20.14).

The majority of early studies on implant integration used machined, minimally rough implants. Today, it is well known that implants with increased surface roughness can result in more rapid integration, with more bone contacts at an earlier stage as a result. Moreover, it seems likely the pathways of implant integration may be different for different surfaces and terms like “contact” and “distance”

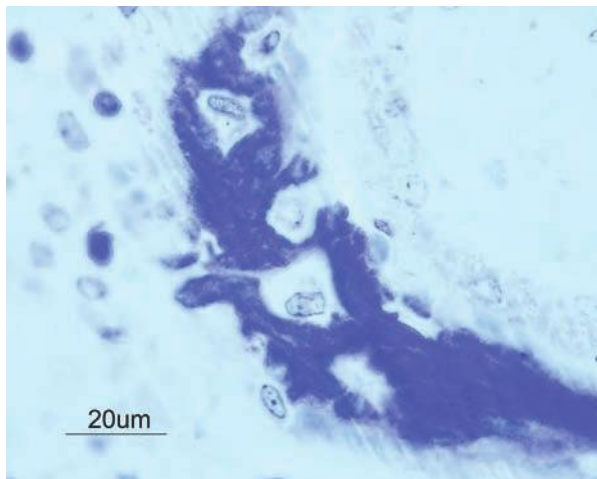


Fig. 20.9 Light micrograph showing osteoblasts producing woven bone in a loose connective tissue near an implant surface 7 days after surgery in the rabbit tibia.

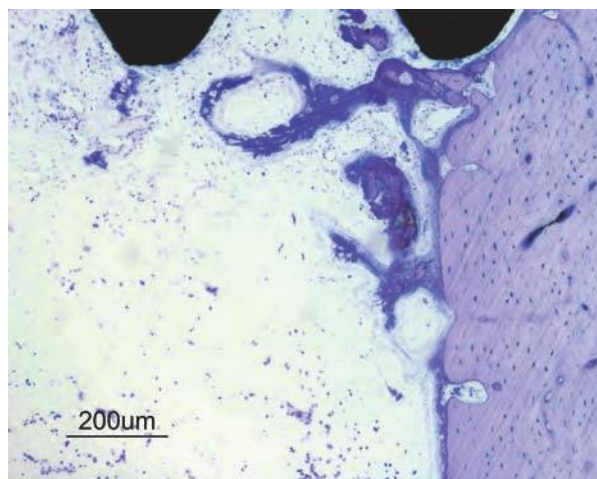


Fig. 20.11 Light micrograph showing formation of woven bone from the endosteal surface from the cortical bone towards an implant 7 days after insertion in the rabbit tibia.

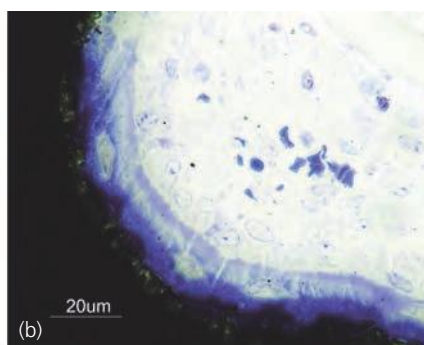
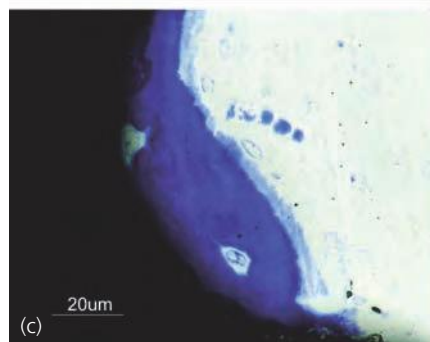
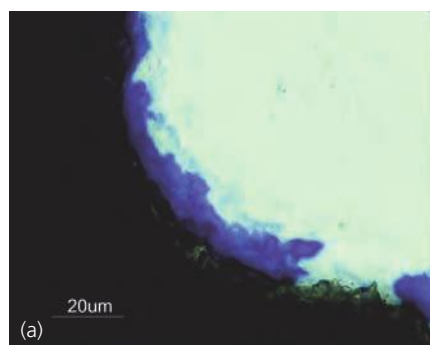


Fig. 20.10 Series of light micrographs showing bone formation directly on an implant surface in the rabbit tibia. (a) An acellular dark stained matrix is seen on the surface (7 days). (b) Osteoblasts producing osteoid are now visible (7 days). (c) Ongoing bone formation from the surface towards the adjacent tissues. Some osteocytes are seen near the surface (14 days).

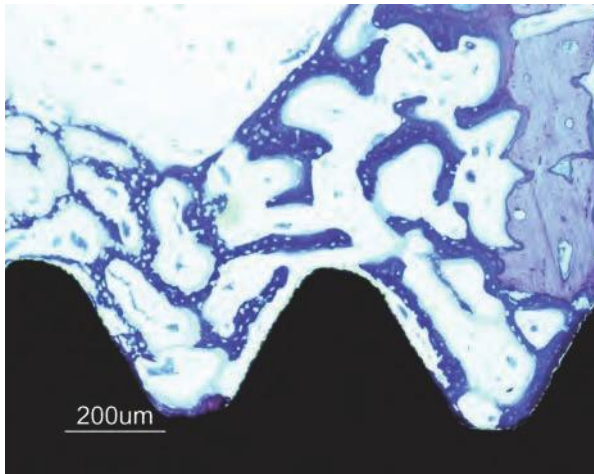


Fig. 20.12 Light micrograph showing extensive bone formation near and at the implant surface 14 days after insertion in the rabbit tibia.

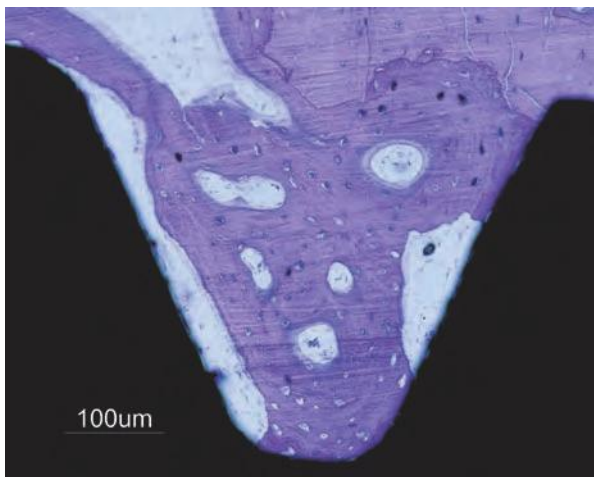


Fig. 20.13 Light micrograph taken 28 days after insertion in the rabbit tibia. Most of the thread is filled with new bone which is undergoing remodeling as indicated by the presence of secondary osteons.

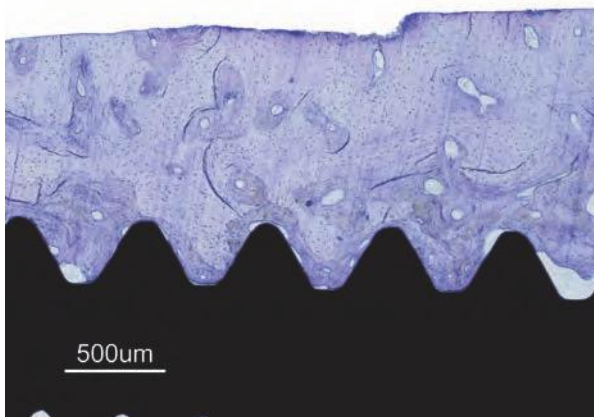


Fig. 20.14 Light micrograph of an implant that had been in clinical function for 9 months. Note the formation of secondary osteons indicating ongoing remodeling.

osteogenesis have been coined to distinguish between the two modes of implant integration.^{14,15} Contact osteogenesis means that the implant is integrated by formation of bone directly to the implant surface and distance osteogenesis means that bone is formed from pre-existing bone surfaces towards the implant sur-

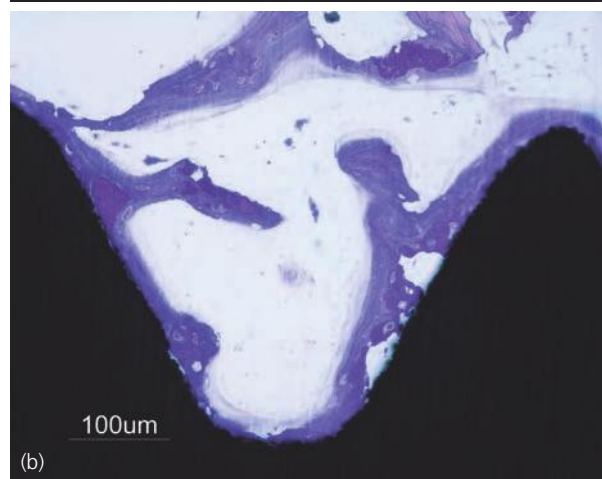
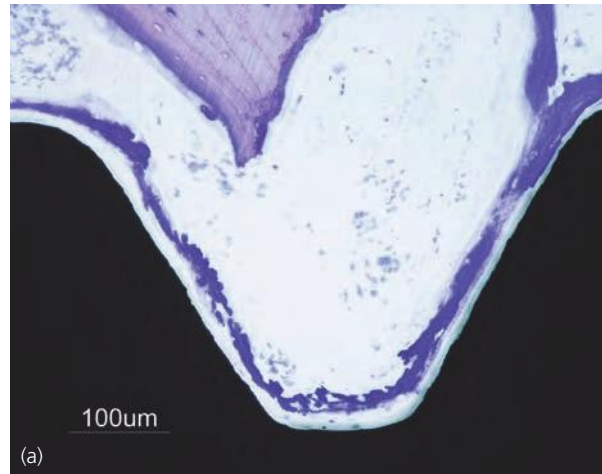


Fig. 20.15 Light micrographs demonstrating differences between a minimally rough and a moderately rough implant surface after 7 days of healing in the rabbit tibia. (a) Bone is formed at a distance from the turned and smoother surface. (b) Bone is formed at and along the surface of a moderately rough implant.

face. The differences between contact and distance osteogenesis were demonstrated in a kinetic study comparing oxidized and machined implant surfaces in a rabbit model after 7, 14, and 28 days.¹⁶ The surface-modified implants showed bone formation by osteoblasts directly on the surface, whilst bone formation occurred in the granulation tissue at a distance from the machined implant surfaces (Fig. 20.15). This may be because the initial blood clot retains better to the rough implant surface. The clot can then serve as a scaffold / matrix for migration of mesenchymal cells to the interface. At a smooth implant surface, shrinkage of the blood clot, which occurs after some time, will result in a gap at the implant interface (Fig. 20.16a), whereas the interface is maintained at a rough surface (Fig. 20.16b). Using the microimplant technique, Ivanoff and co-workers¹⁷ demonstrated more bone-implant contacts as well as more bone in the threads of implants modified by blasting or by anodic oxidation compared with machined controls. They observed bone formation at and along the surface of the modified implants whilst the machined implants seemed to be integrated by bone formed from the surround-

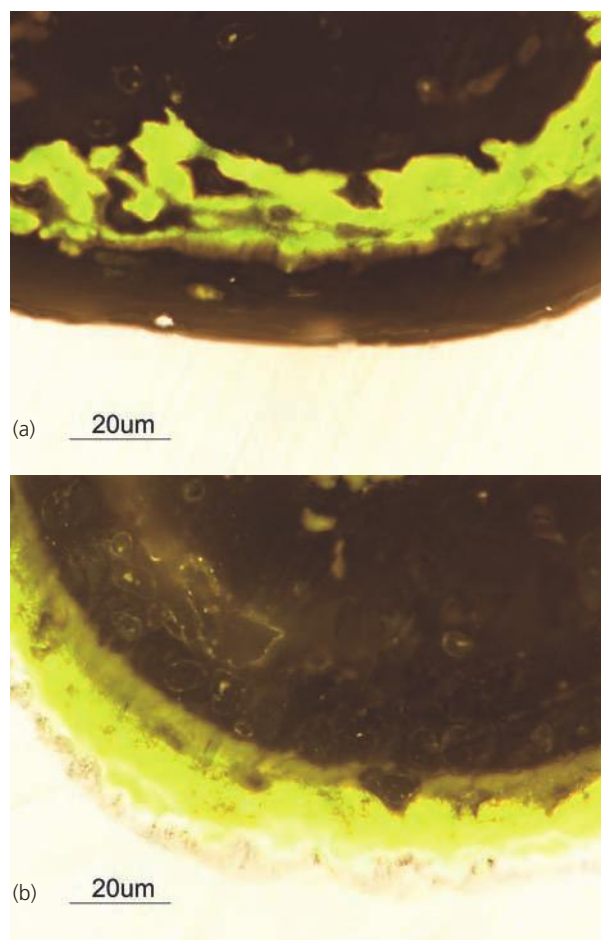


Fig. 20.16 Close-up of Fig. 20.15a, b. (a) Distance osteogenesis near a turned implant. (b) Contact osteogenesis at a moderately rough surface.

ing pre-existing bone walls. However, in spite of the biological benefits with surface-modified implants, randomized clinical studies with up to 5 years of follow-up have demonstrated no differences in normal patient populations.^{18,19} Having said this, the experiences with surface-modified implants in more challenging situations, such as bone grafting and immediate loading, indicate superior outcomes with surface-modified implants. The failure rate with machined implants in reconstructive cases was reported to be around 8%.²⁰ The same research group's experiences from the first 200 surface-modified implants, including varying reconstruction techniques, showed a failure rate of 0.5%.²¹ The results indicate that in biologically challenging situations surface modification may make a difference. Similar experiences have been noted when applying an immediate loading protocol, which is another example of a biologically challenging situation. Glauser and co-workers²² reported a failure rate of 17.3% for immediately loaded machined implants and only 3% for oxidized implants.²³ The histology of clinical oxidized implants, retrieved after 5–9 months of immediate or early (2 months of healing prior to loading) loading showed high degrees of integration.²⁴ Formation of bone directly at the implant surface (i.e. contact osteogenesis) could be demonstrated.

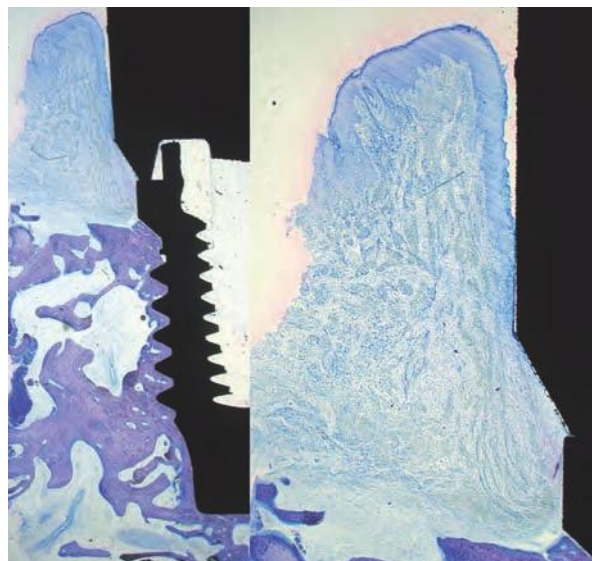


Fig. 20.17 Light micrograph of a clinically retrieved implant with mucosa penetrating abutment. The morphology of the implant–soft tissue interface resembles that of teeth.

The contact osteogenesis properties may have a significant impact on clinical results in terms of the integration of implants in situations where primary bone contacts are missing. The placement of implants in extraction sockets is one example of such a clinical situation. A study in dogs comparing the integration of machined and oxidized implants in bone defects demonstrated a more marked and rapid increase in stability for the latter as measured with resonance frequency analysis (RFA) after 4 and 12 weeks of healing.²⁵ The histological evaluation also revealed differences in favor of the surface-modified implants, since more bone was in contact with the oxidized titanium after 4 and 12 weeks.

The marginal tissues

The soft tissue barrier around an implant or abutment is important for maintenance of implant stability and long-term clinical function, since it protects the integrity of the bone–implant interface (Fig. 20.17). The morphology of the barrier resembles that of gingiva around teeth and contains a sulcus epithelium, a contact epithelium followed by a zone of connective tissue down to the marginal bone.²⁶ The presence of capillaries and postcapillary venules suggests that an effective defense mechanism is at hand as inflammatory cells can migrate into the interface for phagocytosis of bacteria and other foreign bodies. One major difference compared with gingiva is the direction of the collagen fibers which seem to run parallel with the implant/abutment surface, while perpendicular fibers are also present at teeth.

Some loss of marginal bone is expected during the lifetime of an implant. Studies have shown that the major changes, about 0.5–1.5 mm bone loss, occur during healing and the first year of loading.²⁷ The reasons are not well understood, but the initial bone

loss is probably related to healing and remodeling of the marginal bone following implant and abutment surgery as well as a response to loading. Mainly based on experimental dog studies, it has been proposed that a submucosal implant–abutment junction results in a micro-gap where microorganisms may be trapped and induce an inflammatory reaction and bone loss. However, follow-up studies have reported similar amounts of marginal bone loss around one-piece and two-piece implant systems, and thus do not support the earlier findings. Moreover, it is generally anticipated that surface roughness and geometric features such as micro-threads at the neck of the implant may minimize marginal bone loss. However, at present the results from clinical follow-up studies are not conclusive on this point. In general, well documented implant systems show minimal average bone loss over time. Having said this, some implants can show extensive marginal bone loss, with or without signs of infection. The incidence and causes of peri-implant bone loss are not well known and are under debate.²⁸

Implant components

Implant components are illustrated in Fig. 20.18:

- implant fixture;
- cover screw;
- abutments.

Indications for implant treatment

The indications for bone-anchored fixed or removable prostheses are edentulism, partial edentulism, and single tooth loss. In other words, any type of tooth loss is an indication for implant treatment, but



Fig. 20.18 (a) Cross-section of fixture and cover screw. (b) Cross-section of fixture and abutment + bridge screw for screw-retained construction. (c) Abutment in zirconium for a cement-retained crown.

it is certainly not the only or even the best solution for all patients. The clinician should always consider the individual situation for each patient and present different prosthetic alternatives together with treatment plans and estimations of costs. The overall goal with implant treatment is to achieve a long-term successfully stable construction. Criteria for successful outcomes with implant-supported prostheses have been proposed.²⁹ Implant therapy is prescribed to resolve prosthetic problems and such prostheses should meet the clinically evolved standards of function, comfort, and esthetics. The prostheses should also allow for routine maintenance and should permit planned or unplanned revisions of the existing design. Treatment outcome success criteria for implant-supported prostheses should also be assessed in context of time-dependent considerations for any required retreatment. The success criteria comprise the following determinants:

1. The resultant implant support does not preclude the placement of a planned functional and esthetic prostheses that is satisfactory to both patient and clinician.
2. There is no pain, discomfort, altered sensation or infection attributable to the implants.
3. Individual unattached implants are immobile when tested clinically.
4. The mean vertical bone loss is less than 0.2mm annually following the first year of function.

Contraindications

Medical contraindications

The absolute contraindications to implant treatment are few but similar to those for all other types of surgeries. As with any surgical intervention, the implant patient should be assessed preoperatively to evaluate his or her ability to tolerate the treatment. Absolute contraindications to implant surgery are limited to acute illness, terminal illness, uncontrolled metabolic disease, and severe bleeding disorders.

Systemic risk factors

Systemic or general risk factors such as age, smoking, and osteoporosis have been suspected to influence the treatment outcome. Follow-up studies have failed to detect any differences between patients under and over 60 years. Even the age group over 80 years showed high implant survival rates.^{30–32} It has been anticipated that osteoporosis/osteopenia may be a risk factor for implant failure, but a few clinical studies and several case reports have demonstrated successful clinical results in patients with diagnosed osteoporosis.^{33,34} So-called dual energy X-ray absorptiometry (DEXA) measurements have been used to assess the degree of demineralization of the skeleton

and it seems that there is little correlation between the bone quality in the jaws and other parts of the skeleton. It has therefore been suggested that the local bone quality as classified during implant placement is the best predictor for implant failure. Interestingly, failure of implants has been reported in patients treated with bisphosphonates due to osteoporosis. There is, however, little evidence yet that oral bisphosphonate medication in relatively low doses increases the risk for implant failure.^{35,36} On the other hand, intravenous administration of bisphosphonates in high doses due to skeletal malignancies may increase the risk of jaw bone osteomyelitis/necrosis. Additionally, the combination of bisphosphonate treatment and corticosteroids may increase the risk for jaw bone disease.

Patients with metabolic diseases are known to have an impaired bone-healing capacity but well balanced diabetic patients have shown a failure rate similar to that of healthy patients.

Studies have indicated that tobacco smoking is associated with poor peri-implant soft tissue health, marginal bone loss, and implant failure. Even though other clinical investigations have demonstrated little or no negative influence on the peri-implant soft tissues and marginal bone level, in general, implant failure is about twice as common in smokers as in non-smokers.³⁷ It is known that smoking has a negative influence on peripheral microcirculation and wound healing and it has been suggested that smoking influences bone quality, which in turn may lead to higher failure rate.

It has been suggested that ongoing chemotherapy could increase the risk for implant failure. Recent studies have not confirmed that, but of course ongoing treatment with chemotherapy should be considered as a risk factor.^{38,39}

Local contraindications

The primary local contraindication is irradiation to the head–neck region, ongoing and 1–2 years post treatment. There is a high risk of failure if implants are installed in irradiated bone tissue and there is risk of inducing osteoradionecrosis.⁴⁰ Other local contraindications are untreated periodontal disease in residual dentition, periapical lesions at adjacent teeth, other untreated jaw bone infections or cysts, and diseases in the oral mucosa.

Treatment of children and adolescents with oral implants

There are situations when prosthetic rehabilitation of children is needed. Trauma or congenital lack of teeth, such as in cleft patient or children with syndromes, may call for prosthetic treatment. The mouth is important for appearance and self-esteem in the growing individual. Missing teeth should primarily

be replaced by orthodontic spaces closure, composite retained onlay bridges or autotransplantation of teeth. Anchoring elements do not follow skeletal growth in space in the same way as biological teeth and therefore placement of osseointegrated implants is usually not indicated in children. During facial growth, the facial bones are displaced relative to each other and to the cranial base, while their surfaces undergo extensive remodeling. The midface grows generally in a downward and forward direction in relation to the cranial base. The mandible has a V-shaped growth pattern which implies a drift of the lingual and buccal cortical plates.⁴¹ Vertical growth is, to a considerable degree, a result of the development of the alveolar process, which in turn is essentially dependent on the presence of the erupting teeth.⁴² In experimental and clinical studies it has been shown that osseointegrated implants are stationary during growth and do not erupt with adjacent teeth, which subsequently leads to infra-occlusion for the implant-supported crown or bridge.⁴³ Additionally, a fixed prosthesis may impede normal skeletal growth. There are, however, situations when the demands for fixed prosthetic rehabilitation with oral or maxillofacial implants are high. The goal in such cases is not to aim initially at life-long function of the implants and superstructure in the growing patient but to temporarily rehabilitate and accept sequential installation of implants over time, as the maxillofacial anatomy matures.

A more common type of temporary anchorage element in children is the temporary orthodontic implant, which is used as an anchorage for orthodontic forces. This type of implant is pointed, does not require preparation and is inserted without raising a flap. It is inserted in alveolar bone or the palate and is usually not in place more than 12 months. It is simple to remove and local anesthesia is seldom needed at removal.

Treatment planning

Clinical examination

General medical evaluation

As with any surgical intervention, preoperative assessment should be carried out to evaluate the patient's ability to tolerate the procedure. Implant surgery may be associated with certain risks but since it is a relatively atraumatic procedure there are few immediate surgical risks. Absolute contraindications are, as described above, acute and terminal illness, uncontrolled metabolic disease, and severe bleeding disorders.

Oral health status

A thorough clinical examination is mandatory. Visual inspection and palpation of soft tissues and under-

lying bone will usually detect excess tissue and give preliminary information of bone shape and width. Time since tooth extraction and status of any remaining dentition should be taken into consideration. Signs of infection or ongoing periodontal disease are contraindications to implant placement. If immediate installation of an implant is planned, the status of the tooth that is to be extracted should be evaluated. Additionally, prosthetic aspects such as loading conditions, interocclusal distance, extension of the superstructure, and any need for cantilevers should be addressed at this stage. Esthetic demands are usually high in the anterior region and therefore the smile line, lip support, mucosal biotype, intercanine distance, and buccal corridors should be clinically evaluated if implant-supported crowns or bridges are planned in the anterior region. Additionally, a facial analysis should be carried out so that any asymmetry or disharmony of the face is noted.

Jaw relations

The sagittal relation between the jaws changes over time in edentulous patients since bone resorption leads to posterior drift of the maxillary alveolar bone and anterior drift of the crest of the mandible. Subsequently, a class III relation will arise. This is, of course, unfavorable both from a loading point of view and maybe also from esthetic aspects. Bone resorption also tends to make the crests narrower, which is why a transversal registration is also of importance. Correct placement of the implants can, at least to a certain degree, compensate for this. In severe cases of resorption, bone augmentation will be needed prior to or in conjunction with implant placement.

Radiographic examination

A comprehensive clinical examination, as described above, should always precede the radiographic examination, which should be done with techniques resulting in the lowest dose but still presenting all clinically necessary information. Failure to diagnose and treat pathologic conditions around remaining teeth and/or residual jaw bone can seriously compromise the results of implant therapy. Intraoral radiographs are still most common in dental practice and can be recommended if a paralleling technique is used. This way, a preliminary estimate of the vertical dimension of the implant site can be obtained. Panoramic radiographs are often used in the preliminary planning. Pathology in the jaws can usually be detected and available bone height can be assessed in both anterior and posterior regions. The best estimate of height and width, however, is obtained with cross-sectional tomography. This technique gives a higher radiation dose to the patient and should not be performed without a clear indication. There are some guidelines where indications for cross-sectional imag-

ing in implantology have been presented.⁴⁴ These are:

- when minimizing the risk of damage to important anatomical structures;
- to provide more information in cases where there is limited amount of bone height and/or width;
- to improve implant positioning that will optimize biomechanical functional and esthetic results.

Computed tomography (CT) and cone-beam computed tomography (CBCT)

Traditional medical CT scanners are big and expensive and have mostly been used for cranio-maxillofacial trauma surgery and for planning of treatment of malignancies in the head and neck region. CBCT scanners have been available for craniofacial imaging since 1999 in Europe and since 2001 in the US. These scanners use a cone-shaped X-ray beam rather than a conventional linear fan beam to provide images of the structures of the skull and maxillofacial bones. Conventional CT scanners use a single row of solid state detectors paired with a fan-shaped beam to capture the attenuated X-ray. CBCT scanners use a square two-dimensional array of detectors to capture the cone-shaped beam. This means that traditional CT provides a set of consecutive slices of the patient while the CBCT provides a volume of data. Both techniques make it possible to get a three-dimensional (3D) image of the anatomy when software is added to the equipment.

The CBCT scanner is smaller than a traditional CT scanner and the radiation dose is relatively lower, which makes it more popular in dental practice.

Radiographic classification of bone quality

Traditionally, classification of bone quality and sometimes also bone quantity has been registered during implant installation. A scale from 1 to 4 has been used, where 4 is the most demineralized type of bone and gives least mechanical support of the fixture at installation. Today, when tomography is getting more common, this rather subjective system can be replaced by the Hounsfield scale. The bone quality can be shown for each individual implant site (Fig. 20.19).

Hounsfield units:

- D1: >1250 HU;
- D2: 850–1250 HU;
- D3: 350–850 HU;
- D4: 150–350 HU;
- D5: <150 HU.

Model analyses

Model analyses are typically done on plaster casts and can be used for anatomic registration and for evaluation of occlusion and articulation. Implant

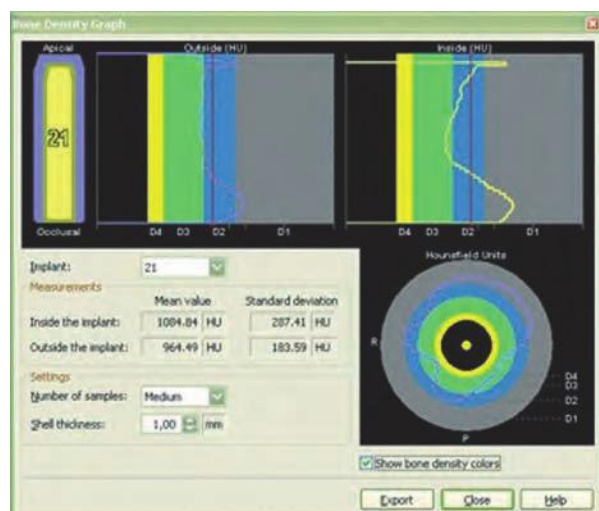


Fig. 20.19 Bone quality, Hounsfield scale.



Fig. 20.20 First, the planned position of the implant site is marked with a guide drill. This will provide information about the quality of the marginal bone and is also a start point for the first twist drill.

fixture position can also be planned on the model. Modern software and the use of 3D planning will, however, most likely put the plaster cast in the history books fairly soon.

Surgical procedure

Regardless of the preoperative planning and choice of surgical protocol, treatment includes site preparation and installation of the implant, aiming at good primary stability. Figs 20.20–20.26 provide a general overview of a standard implant site preparation with raised mucoperiosteal flaps.

If teeth have to be removed because they are impossible to use for prosthetic reconstruction, prepare for implant placement using an atraumatic extraction technique. The aim is to reduce unfavorable post-extraction ridge remodeling. Bone grafts and/or resorbable membranes may be used to preserve the ridge during healing, especially in the esthetic zone. Immediate installation of implants after extraction is



Fig. 20.21 Drilling should be in the planned direction to the appropriate depth under profuse irrigation. A secured indicator can be used to facilitate the direction of the subsequent drilling.



Fig. 20.22 Drilling is done stepwise to the planned diameter. Mesial-distal distance minimum 2 mm to adjacent roots and 3–4 mm between implants.

an alternative to a staged treatment protocol but there are some prerequisites that have to be fulfilled:

- Absence of active infection.
- Adequate residual ridge.
- Possibility for primary stability.
- Adjunctive use of bone grafting techniques to correct residual defects may be used, especially if there is a gap of >2 mm between the implant fixture and extraction socket.

Maintenance

Postoperative check-ups are mandatory and the intraoral radiograph is still the gold standard for evaluation of marginal bone level. The requirements for radiographic check-up have changed recently and today an individualized follow-up regimen is advised, which depends on factors such as patient cooperation and marginal bone level changes during the first year post-treatment. Annual radiographs are not recom-



Fig. 20.23 Drilling depth is verified before the implant is inserted.



Fig. 20.24 Implant installation with a contra angle at low speed (25 rpm) and maximum torque 35 Ncm.



Fig. 20.25 A two-stage procedure means that cover screws are installed and the flaps are repositioned tightly. This method is used when healing period without loading is needed, e.g. in soft bone or when the primary implant stability is poor.

mended if stable marginal levels have been established during the first 1–2 years of function. As a rule of thumb, radiographic evaluation of implants and surrounding bone should be carried out at delivery of the superstructure and then after 1, 3, 5, and 10 years in function. Clinical examination, including registra-



Fig. 20.26 One-stage procedure. Here, the flaps are sutured around the abutments.

tion of plaque and bleeding at probing around the abutments, should be carried out 3 months after delivery of the superstructure and, if without remarks, annually. Good oral hygiene is mandatory after implant treatment, especially if the patient has a history of periodontitis.

Computer-guided implant treatment

When a patient is considered suitable for implant treatment, computer-guided planning and treatment can be used. Computer-guided implant treatment implies that the patient has to go through a CT scan (or CBCT), preferably with a radiopaque scan prosthesis in place, before the data can be imported into a software planning program. There are several different software programs available on the market. To take full advantage of the concept it is recommended to define from the outset, the type of final restoration, the need for provisional restoration, and what surgical protocol to use. The software (Fig. 20.27) can either be used alone or as a base for manufacturing a surgical guide. If a surgical guide is used, it will be the link between the planning done with the software and the actual surgery. Cylinders in the guide replicate the

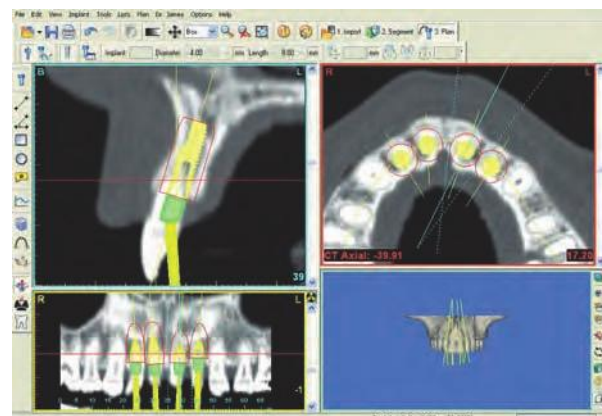


Fig. 20.27 Software for computer-guided implant planning.

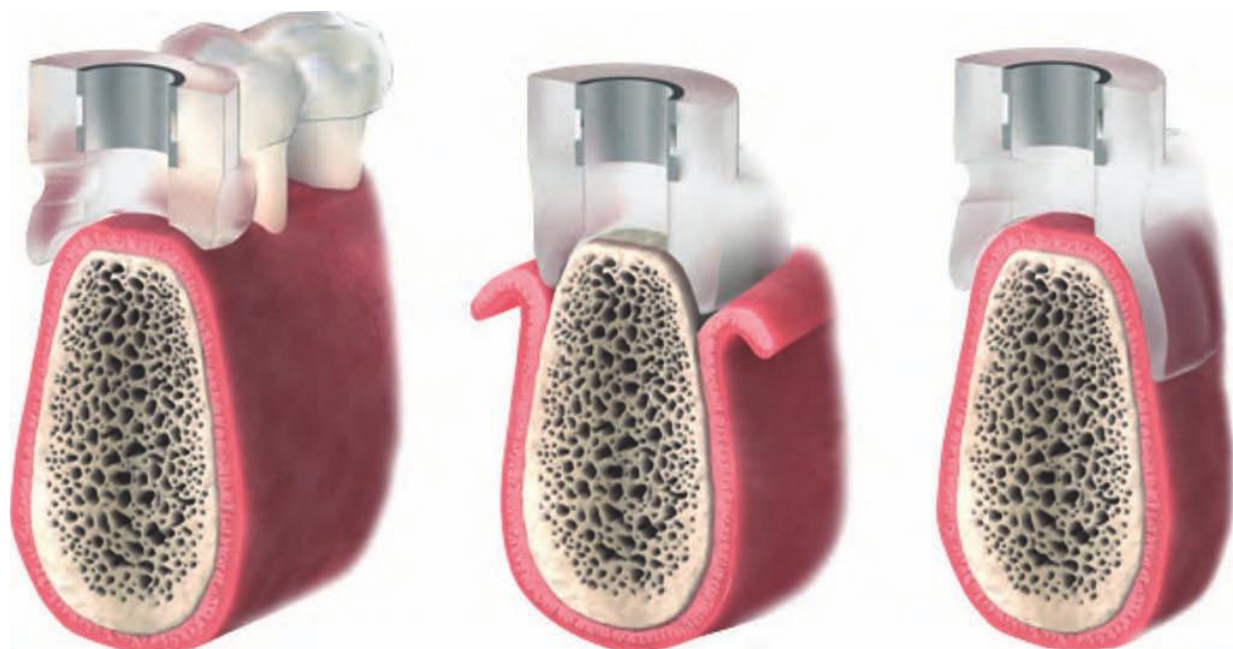


Fig. 20.28 Surgical guides: bone-supported, tooth-supported, and mucosa-support

plan by guiding the drilling and implant installation in the location and orientation defined by the software. The guide is produced using a biocompatible material, usually using a stereolithography process, and is custom-made for each patient. There are three different guides to be used: bone-supported, mucosa-supported or tooth-supported (Fig. 20.28). The bone-supported guide means that a flap has to be raised and the guide should be relatively stable when placed on the alveolar crest. The tooth-supported guide is also fixed and stabilized by the residual dentition and a flapless technique can be used. The so-called mucosa-supported guide means by definition that the surgery is performed without raising a flap. This type of guide will be difficult to keep stable in position during the preparation and placement of implants, so three to four horizontally inserted, stabilizing pins/screw are used to keep the guide in place. The interest in flapless surgery (also referred to as “minimally invasive surgery”) is increasing, since less postoperative swelling and discomfort have been reported.

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Chapter 21

Optimal Implant Placement in the Esthetic Zone by the Use of Guided Bone Regeneration

Christer Dahlin

The biological principles and indications for the use of guided bone regeneration (GBR) are described with specific focus on the esthetic zones in the upper and lower jaws. The efficacy of barrier membranes in conjunction with bone healing and reconstructive therapy is the result of mechanical, cellular, and molecular mechanisms. Basic studies on guided bone regeneration have shown the same sequence of healing occurring as in regular fracture repair. Different kinds of biological membranes, resorbable as well as non-resorbable, are described in the chapter. Clinical results are presented and a section on complications is also included. The author concludes that the underlying bone structure of the alveolar process plays a key role in the overall esthetic appearance.

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Clinical results of GBR

Biological factors influencing the reconstruction of alveolar bone

An absolute prerequisite for implant treatment is the availability of sufficient alveolar bone to support and retain the endosseous implant.¹ Factors such as infec-

tion, cystic lesions, tooth/alveolar trauma or congenital tooth agenesis cause a reduction of the alveolar ridge dimensions to a varying degree. With the increasing drive for optimal esthetic outcome of implant treatment, restoring both the hard and soft tissue levels is essential (Fig. 21.1).²

Tooth replacement in the anterior maxilla is a demanding treatment, since the absence of, or poor, preoperative planning, or the choice of an inappropriate treatment approach can lead to everything from esthetic shortcomings to real disasters.

Esthetic complications can be related to malpositioned implants and the choice of inappropriate prosthetic components. The most critical factors, however, are the anatomic causes that include bone deficiencies in the horizontal or vertical dimensions, and often a combination of the two. This is not infrequently associated with soft tissue defects of the alveolar ridge. Alveolar atrophy and anatomic alterations will have a negative influence on the proper buccal-palatal position of the implant.^{2,3} Malposition of the implant may have effects on the shape, emergence profile, and interproximal contour.

It is important for the clinician to understand that the anatomic contour of the ridge comprises the soft tissue and the underlying supporting bone tissue in all directions. Hence, the soft tissue contour is heavily influenced by the bone anatomy present.

The concept of the so-called biological width has increased the knowledge and understanding of the interaction between the different tissue types and different biomaterial surfaces.⁴ In brief, the soft tissue demonstrates relatively constant dimensions in thickness; the peri-implant soft tissue thickness is about 3–4 mm. It is slightly thinner on the buccal aspect and more pronounced at the interproximal areas. The soft tissue is also slightly thicker in the anterior maxillary area in contrast to the posterior region of the mandible, which demonstrates the thinnest portion in the oral cavity.



Fig. 21.1 (a) Clinical case demonstrating a trauma at young age and subsequent tooth loss (21). Note the buccal atrophy and concavity (arrow) of the hard and soft tissue. Due to the loss of tissue optimal restoration is not possible from an esthetic point of view. The patient has been temporarily restored with a Maryland bridge while waiting for skeletal growth to be completed. (b) Healing following implant placement and simultaneous GBR procedure. The buccal bone has been augmented with BioOss® and a Bio-Gide® membrane. Note the regained harmony of the contour of the keratinized gingiva of the soft tissue. (c) Ample time should be provided for the soft tissue to mature and stabilize prior to the finalization of the tooth. A combination of a permanent abutment in a biocompatible material and a high-quality temporary crown is provided for at least 2 months. (d) Final restoration of tooth 21 with optimal esthetic result. Note the harmony of the hard and soft tissue in the region providing an ideal emergence profile for the final crown.

Due to the relatively constant dimensions of the soft tissue, it is inevitable that the underlying bone structure of the alveolar process plays a key role in the overall esthetic appearance. This is particularly pronounced in the anterior region of the maxilla. This area is also defined as the esthetic zone.

The resorption pattern of the alveolar process following single or multiple tooth loss has been studied in detail and this knowledge is important in the treatment planning of esthetic implant dentistry.⁵⁻⁷ Frequently, loss of the buccal bone plate (horizontal resorption) is seen in trauma patients (Fig. 21.1a). This is most pronounced during the first 6 months following the injury. Another important anatomic structure to consider is the interproximal crest height since it plays a significant role in maintaining the peri-implant papillae. Today it is well accepted that vertical resorption of more than 5 mm from the top of the bony crest to the contact point reduces the probability of an intact papilla by approximately 75%.³ This is particularly the case in situations with multiple tooth loss.

Today's state-of-the-art esthetic treatment not only involves optimally designed crown and bridge solutions. It also involves restoration of the adjacent tissues (Fig. 21.1). Based on the discussion above, a solid

base needs to be created. Thus, reconstruction and correction of bone defects are needed. Numerous methods have been used in an attempt to overcome this problem. Previously, one of the most common methods involved the harvesting and implantation of fresh autogenous bone grafts. However, this involves an extra surgical site, and the morbidity for the patient following such an event should not be ignored. Numerous different types of bone substitute materials have been developed and recent advances are of great interest.

The materials can be divided into three categories:

- **Allografts.** This group of materials is derived from an individual of the same species and contains no viable cells.⁸ The principles for incorporation of allografts follows the same principles as for fresh autogenous bone grafts but the incorporation process probably proceeds more slowly due to the absence of living cells which are both osteoinductive and responsible for triggering the inflammatory process. Frozen or freeze-dried, mineralized or demineralized bone, demineralized dentin, and antigen-extracted allogenic bone (AAA) are all examples of allograft materials.

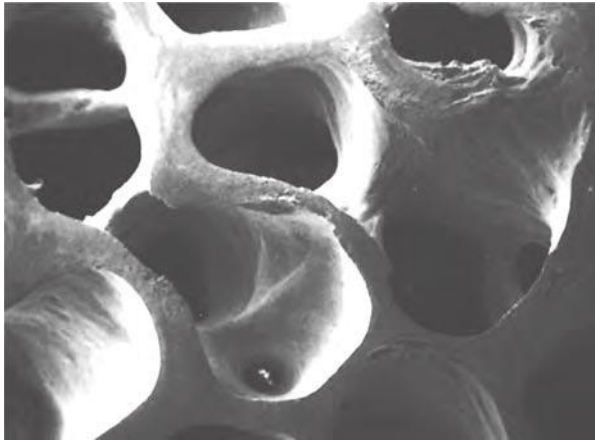
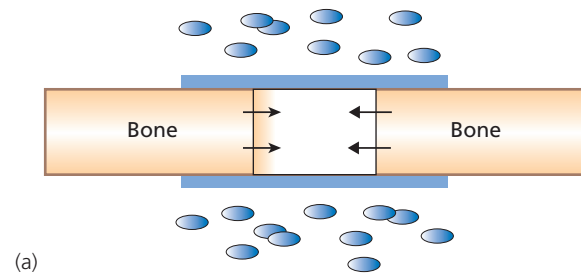
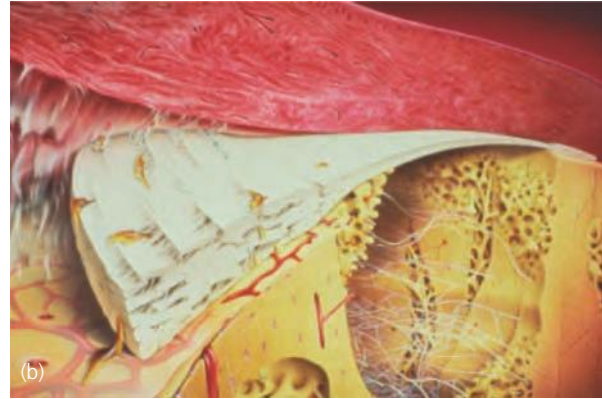


Fig. 21.2 Scanning electron microscopy image of the bovine hydroxyapatite (BHA) structure of BioOss® after deproteinization. The crystalline structure of BHA is very similar to that of human bone.

- **Alloplastic grafts.** These are developed and derived synthetically and follow the same principles as for allografts, except that the materials contain no proteins and have osteoconductive properties only.⁹ Hydroxyapatite (HA) is probably the best known material in this family. Bioactive glass ceramic is another member of this family that has been tested and demonstrates active bone formation. Calcium sulfate (plaster of Paris) has also been evaluated as a grafting material.¹⁰ Recently great interest has been shown in tricalciumphosphate (TCP) materials. This material is osteoconductive and has the capability to be slowly resorbed and eventually replaced by new bone.¹¹
- **Xenografts.** A xenogeneic graft is derived from bone tissue originating from various species. In order to avoid an immunologic response and subsequent rejection after implantation, the proteins have to be eliminated. The osteoinductive capacity of the material therefore disappears and this type of graft material only acts as an osteoinductive scaffold. During the last decade a bovine hydroxyapatite (BHA) (Bio-Oss®, Geistlich, Wolhusen, CH) has been developed and widely used in conjunction with implant treatment. It is by far the best documented material available on the market at present. The material is quite similar to human bone in its configuration and is considered to be osteoconductive (Fig. 21.2). However, there is a discussion about whether BHA is resorbable, or is slowly degraded, or is phagocytal or non-resorbable. Recent findings claim that virtually no resorption occurs even after several years.¹² The biological impact of these findings remains to be fully understood. The fact that BHA particles are incorporated into newly regenerated bone could actually be of an advantage in esthetic situations where the material will act as “reinforcement” of the newly formed bone tissue and thereby prevent early resorption of the regenerated bone.



(a)



(b)

Fig. 21.3 (a) Schematic drawing illustrating the principle of guided bone regeneration. Barrier membranes are placed in direct contact with the bone surface thereby protecting the defect from interfering soft tissue cells. A secluded environment is created into which only cells from the adjacent bone can migrate. (b) Illustration showing the principle of an e-PTFE barrier membrane. Note the more open portion in the periphery of the barrier, designed to allow more rapid ingrowth of the soft tissue. This will stabilize the membrane and minimize micro-movements. Also note the more cell-occlusive portion placed over the defect.

The biological principles of guided bone regeneration

Over the last two decades, the development of the technique of guided bone regeneration (GBR) has had a significant impact on esthetic reconstruction in conjunction with implant therapy. This technique involves the use of physical barrier membranes during the healing phase in order to avoid ingrowth of undesired tissue types into a wound area.¹³⁻¹⁷

During the 1980s the principle of guided tissue regeneration (GTR) was developed for regenerating periodontal tissues lost as a result of inflammatory periodontal disease. A series of studies documented the possibility of excluding undesirable cells from populating the wound area by means of membrane barriers. This favors the proliferation of defined tissue cells to produce a desired type of tissue (Fig. 21.3).

The principle of physical sealing of an anatomic site for improved healing of certain tissue types is by no means new. During the mid-1950s attempts were made for neural regeneration by the use of cellulose acetate filters.¹⁸

GBR refers more precisely to the goal of the membrane application than guided tissue regeneration. This concept promotes bone formation by protection

against an invasion of competing, non-osteogenic tissues. To this end, bone defects are tightly covered by a barrier membrane of defined permeability and excellent biocompatibility.

Experimental studies have proven that certain tissues within the body possess the biologic potential for regeneration if the proper environment is provided during healing. The ultimate goal of GBR is to use a temporary device to provide the necessary environment so the body can use its natural healing potential and regenerate lost and absent tissues.

The efficacy of barrier membranes in conjunction with bone healing and reconstructive therapy is probably the result of a combination of different mechanisms – mechanical, cellular, and molecular. Examples of these are:

- prevention of fibroblast mass action;
- prevention of contact inhibition by heterotopic cell interaction;
- exclusion of cell-derived soluble inhibitory factors;
- local concentration of growth stimulatory factors;
- stimulatory properties of the membrane itself.

The basic studies on GBR have shown that the sequence of healing occurring in regular fracture repair follows the same basic pattern that is found in osseous lesions during GBR therapy. Based on the scientific evidence available, it can be stated that certain conditions must be met for new bone formation to be predictably accomplished by GBR:

1. There must be a source of osteogenic cells. Viable bone must be present adjacent to the defect where regeneration is desired.
2. An adequate source of vascularity is essential. This supply originates mostly from the adjacent bone surface (Volkmann's canals and marrow compartment).
3. The wound site must remain mechanically stable during healing.
4. An appropriate space must be created and maintained between the membrane and the parent bone surface.
5. Soft connective tissue cells must be excluded from the space created by the membrane barrier. The structure of the material used must be able to accomplish this.

The general function of a membrane used for GBR therapy is to create an environment that will allow the normal healing process to form bone in a defined region (Fig. 21.3). Hence, the host tissue–biomaterial interaction should not interfere with bone formation and maintenance to a clinically significant degree. Biomaterial chemistry and structure should result in minimal foreign body response. Optimal bone–biomaterial interaction characteristics are also desirable. A GBR membrane that allows close adaptation of

bone tissue will allow more complete fill of the space defined by the membrane and stabilization of the membrane within the overall system.

Tissue integration

The phenomena of ingrowth and surface bonding of tissue to a biomaterial are termed integration. Surface and microstructural characteristics are usually responsible for these events (Fig. 21.4). The clinical benefits of GBR membranes that have the capacity to integrate with surrounding tissues are a result of a more mechanically stable (and therefore predictable) wound healing environment. While tissue integration appears to be necessary for optimal performance of a GBR membrane, chemical and structural properties that encourage tissue integration must be balanced with the overall functional needs for alveolar ridge augmentation (Fig. 21.4).

Membrane design criteria and material selection

The acceptance of membrane-assisted regeneration of osseous lesions in the oral cavity has introduced reconstructive dentistry to new therapeutic procedures and biomaterials. Clinicians are being exposed to an increasing number of membrane materials used, or proposed for use, in GBR. In order to select the best material for a specific clinical indication, it is impera-

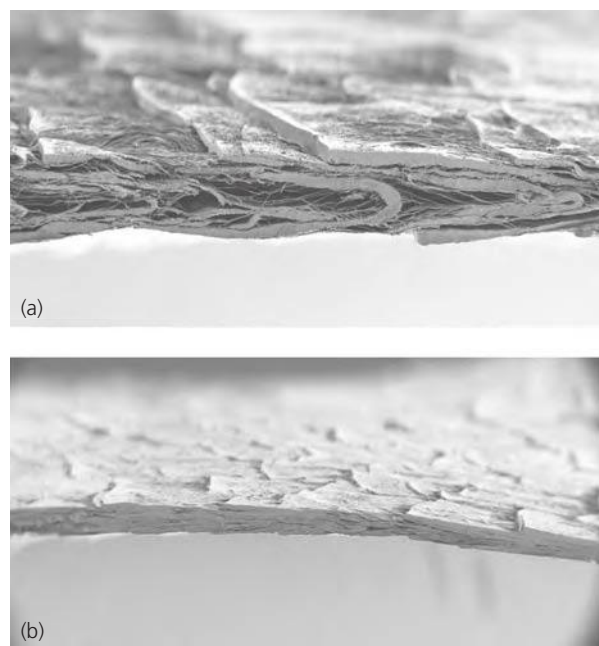


Fig. 21.4 (a) The outer surface of the GTAM (Gore-Tex Augmentation Material) is designed to create a barrier to passage of cells and to allow connective tissue attachment. The material is 50% air by volume and 25 μm between nodes. (Scanning electron microscopy image.) (b) The inner surface of the GTAM is designed for connective tissue attachment. It is 30% air by volume and less than 8 μm between nodes. (Scanning electron microscopy image.)

tive to understand the functional requirements demanded of membrane barriers for GBR procedures.

If the only requirement of a membrane material used in GBR were to provide a barrier to the proliferation of fibrous connective tissue, any suitable biocompatible material in the form of a cell-occlusive film could be used in clinical practice. However, a membrane that is used for alveolar ridge augmentation must meet a number of requirements in addition to acting as a passive physical barrier:

1. The membrane must be constructed of acceptable biocompatible material. The interaction between the material and the tissue should not adversely affect the surrounding tissue, the intended healing result, or the overall safety of the patient.
2. The membrane should exhibit suitable occlusive properties to prevent fibrous connective tissue (scar) invasion of the space adjacent to the bone and provide some degree of protection from bacterial invasion should the membrane become exposed to the oral environment (Fig. 21.4b).
3. The membrane must be able to provide a suitable space into which osseous regeneration can occur. Spacemaking provides necessary volume with specific geometry for functional reconstruction.
4. The membrane should be capable of integrating with or attaching to the surrounding tissue. Tissue integration helps to stabilize the healing wound (Fig. 21.4a). It helps to create a "seal" between the bone and the material and prevent fibrous connective tissue leakage into the defect, and retards the migration of epithelium around the material should it become exposed.
5. The membrane must be clinically manageable.

Two different types of membrane are the most commonly used on the market. Non-resorbable, e-PTFE (expanded polytetrafluoroethylene) membrane was the first material to be successfully applied for GBR. The use of this material requires a removal procedure once the healing is completed. Although this material demonstrates superior biological response, the extra steps in clinical handling led to the development of biodegradable membranes such as collagen or synthetic polymers. Furthermore, attempts have also been made using other types of barrier membranes such as lyophilized dura, calvarial bone, and peritoneal tissue. However, the results regarding these latter materials are still somewhat limited in applications related to implant treatment.

Biocompatibility

When discussing the clinical outcome of membrane materials, biocompatibility is a fundamental requirement for acceptable function of any implantable medical device. Although this requirement is often taken for granted, tissue interactions involve many

application-specific factors that are governed by complex mechanisms.

A classical definition of biocompatibility by Williams¹⁹ is "*the state of affairs when a biomaterial exists within a physiological environment, without the material adversely and significantly affecting the body*". This should be interpreted with regard to biomaterial used, the indication, and the environment in which the material is placed and maintained. For example, degradable materials are clearly affected by the environment of the body; however, safe degradation is one of the primary intended functions of this class of biomaterials. Biocompatibility is a relative term. All implanted materials interact with the host tissue to some extent. Biomaterials with dissimilar chemical composition or biomaterials with the same chemical composition but with different macro- and micro-structure will demonstrate different cellular or systemic responses.

Non-resorbable membranes

As previously described, non-resorbable membranes were the first materials successfully used in GBR. The best documented material is e-PTFE (expanded polytetrafluoroethylene) (Gore-Tex Augmentation Material, W.L. Gore & Ass. Inc., Flagstaff, AZ, USA). Originally used in medical applications such as synthetic vascular graft and heart patches, the material has an extensive documentation with regard to tissue response and safety. In the 1990s, barrier membranes with special characteristics for GBR were developed and became commercially available (Fig. 21.4). Substantial experimental and clinical data are available for this type of membrane. The biological response is near to ideal and the e-PTFE membrane is still considered the "gold standard" in membrane technology.²⁰ The material is made up of carbon and fluorine chains strongly bonded to each other. This creates a highly chemically stable and hydrophobic material which is ideal for biocompatible tissue interaction. The material has an open outer structure for early tissue integration and stabilization and an inner portion which is responsible for the occlusive properties of the device (Fig. 21.4).

Biodegradable barrier membranes

Collagen membranes are resorbed by enzymatic degradation, while synthetic polymers are resorbed via degradation into lactic acid and water.

Bio-Gide[®] was the first collagen barrier membrane designed for GBR and is by far the best documented product in the literature. It is made from native, non-cross-linked collagen types I and III and consists of two functional layers. The compact layer is cell occlusive and fulfills barrier function, whilst the porous layer allows tissue integration (Fig. 21.5). The membrane has hydrophilic properties, which enable self-adherence to the bone surface, thus providing easy

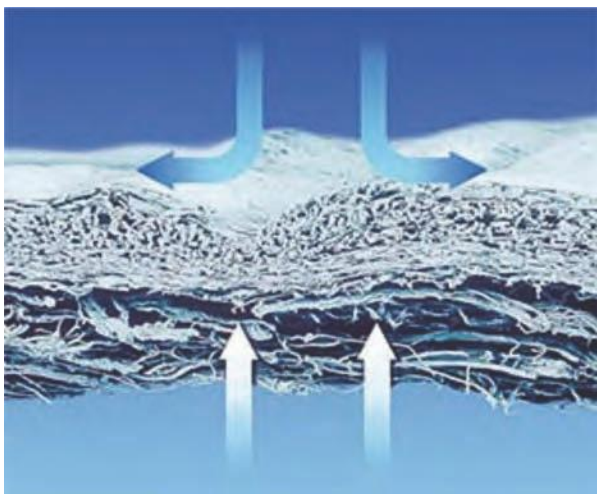


Fig. 21.5 Scanning electron microscopy image showing cross-section of a biodegradable Bio-Gide[®] collagen membrane. The double layer design is created in order to stabilize the material and prevent soft tissue penetration.

clinical handling. Due to the lack of stiffness, collagen membranes are usually used in combination with bone chips or bone substitutes.

Pure synthetic biodegradable membranes are also available on the market. These materials usually consist of a combination of PLA/PGA (polylactide and polyglycolide). An example of such membranes is RESOLUT (W.L. Gore & Ass. Inc., Flagstaff, AZ, USA).²⁵ Although this demonstrates excellent biological behavior in experimental studies, the amount of clinical data is still somewhat limited.

Indications for GBR treatment

When a defect in the alveolar bone is present, three basic situations can require the use of a regenerative procedure:

- Bucco-palatal bone thickness is reduced, but still allows placement of the implant although with the result of either a dehiscence or fenestration-type defect (simultaneous approach) (Fig. 21.6a).

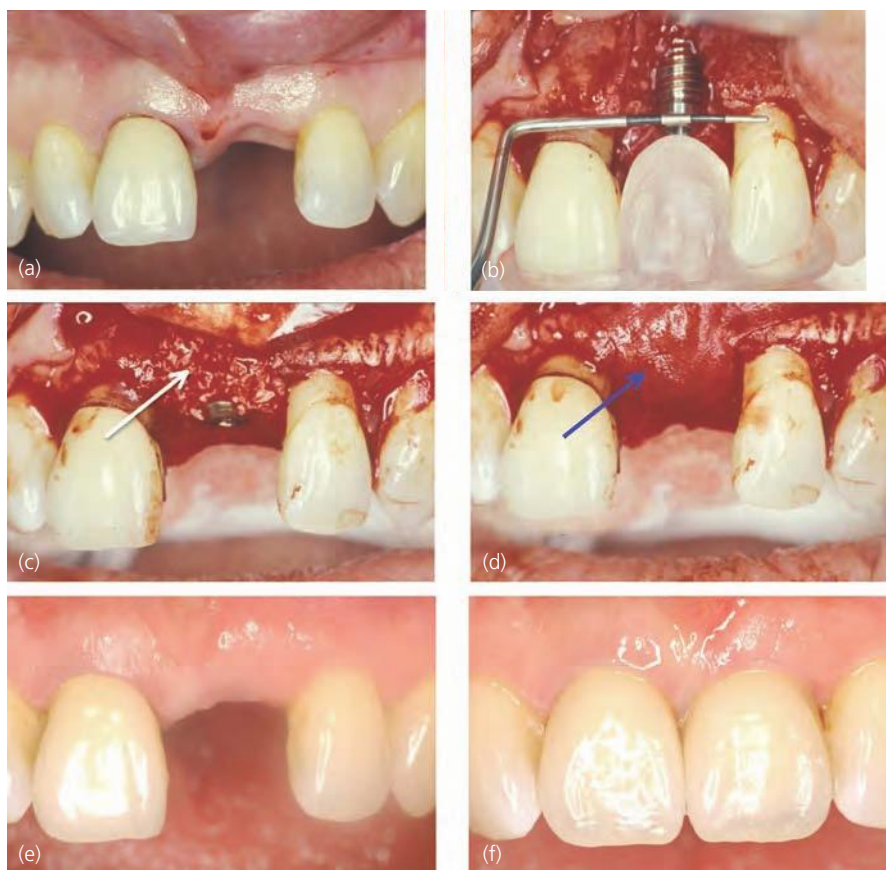


Fig. 21.6 (a) Buccal concavity in combination with minor vertical bone loss due to earlier trauma. The bone tissue is not sufficient either for optimal implant placement or support of the soft tissue. (b) Buccal mucoperiosteal flap raised. Note the design of the flap including one extra tooth on each side of the implant site. The implant is placed in optimal position according to the preoperative prosthetic planning using a surgical stent. Lack of buccal bone and several exposed threads of the implant are evident. (c) BioOss[®] particles mixed with autogenous bone particles harvested from preparation of the implant site are placed in the defect area and also used to cover the surface of the implant. (d) A Bio-Gide[®] membrane is trimmed and placed over the defect area. Note that the membrane is allowed to extend into the periphery from the graft material. Also note the trimming which leaves approximately 1 mm of space between the membrane and the adjacent root surfaces. (e) Clinical photograph showing uneventful healing after 6 months. Note the harmony of soft tissue contour compared to prior to treatment (Fig. 21.6a). (f) Completion of the treatment. Final crown (Procera[®]) in place. The soft tissue contour including the height of the papillae is evident.

- Horizontal bone thickness is reduced in such a way that optimal implant placement with regard to prosthetic planning is not possible (simultaneous approach).
- Bone thickness is so reduced that implant placement is not possible with proper primary stability. In this situation, ridge augmentation is required prior to implant installation (staged approach).

Basic surgical technique of GBR

Preoperative antibiotics

The same antibiotic prophylaxis is recommended as in conjunction with implant placement according to standard protocol. Usually a 1-day regimen including 2 g fenoxymethyl-Pc $\times$ 2 is enough. For advanced GBR cases, 1 week's antibiotic coverage postoperatively is recommended.

Flap design

A full-thickness crestal incision, slightly buccal but still within attached mucosa, is performed and extended mesially and distally to the adjacent teeth. Diverging releasing incisions are then performed buccally (Fig. 21.6b). Full-thickness mucoperiosteal flaps are elevated.

Site preparation

Based on the available amount of host bone present, simultaneous implant placement can be performed. If optimal position and direction and satisfactory primary stability cannot be obtained, a two-stage procedure should be considered.

The bone surface in the augmentation area should be carefully debrided in order to remove all remnants of soft tissue. If implant placement is performed it should be performed according to the protocol of the implant system used, aiming at a prosthetic-driven position (Fig. 21.6c).

Prior to placement of graft material and the barrier membrane, the buccal bone plate in the defect area must be perforated to create access for multipotent cells and blood vessels emanating from the marrow cavity. This can be performed either with a spiral or a round bur with a dimension of approximately 1 mm. This surgical procedure is believed to stimulate osteogenesis by activating a cascade effect of growth factors. Furthermore it allows the formation of an appropriate coagulum which will act as a matrix for the initial bone formation.

Graft material

The use of spacemaking materials underneath the membrane has been proven to provide a more pre-

dictable regenerative result and is today considered state of the art. Many different materials, including autogenous bone chips, freeze-dried-demineralized, deproteinized bovine bone, and synthetic graft materials such as TCPs, have been tested with varying results. The best documented filling material with predictable outcome is a combination of deproteinized bovine bone (Bio-Oss, Geistlich, Switzerland) and autogenous bone chips mixed in a ratio of 1:1. The addition of Bio-Oss has been shown to minimize resorption of the newly regenerated bone (Fig. 21.6c).

Membrane selection and positioning

The anatomic shape of the defect to be regenerated dictates the choice of membrane material. The following protocols can be recommended.

Biodegradable membranes

Most defects can be treated with resorbable membranes together with autogenous bone chips alone or in combination with bone substitutes. Usually the autogenous bone chips are placed in contact with either the bone surface or the exposed parts of the implant and then covered with a layer of Bio-Oss. Alternatively the two filling materials are mixed together in a ratio of approximately 1:1.

The membrane must be cut and trimmed to adapt to the anatomy of the ridge and applied over the defect in order to cover the bone graft. Due to the hydrophilic properties of the Bio-Gide membrane it will "stick" to the bone surface once wetted either with saline or blood. Hence no fixation screws or tacks are needed for stabilization in most cases (Fig. 21.6d).

Non-resorbable membranes

When a large volume of bone (outside the bone envelope) must be regenerated, the use of non-resorbable e-PTFE membrane is indicated. The preparation of the augmentation site is identical to that described above. However, extra attention must be paid to the membrane adaptation and fixation. The membrane must be cut to extend at least 4–5 mm beyond the filling material to avoid interference with the surrounding soft tissue. It is important to avoid creating sharp edges since they can increase the risk for membrane perforation during healing. A critical note is to trim the membrane so a distance of 1–2 mm is maintained from the root surface of the neighboring teeth. This is to avoid contamination due to bacterial downgrowth along the root and also to enhance periodontal reattachment.

Finally, the membrane should be fixated using either micro-screws or specially designed tacks. It is practical to start this procedure on the palatal side

prior to the placement of the bone graft material (Fig. 21.7). A critical technique is the adjustment of the flaps prior to suturing. A completely tension-free environment must be created by performing periosteal releasing incisions at the base of the buccal flap.

Suturing

Suturing is recommended using non-resorbable sutures in a biocompatible material. A double suture layer should be created with a combination of horizontal mattress sutures (4/0) (on top of the crest) followed by single interrupted sutures (5/0 or 6/0) for mucosal closure.

Follow-up

Due to the compromised wound, the recommendation is to maintain the sutures in place for at least 14 days. The patient should receive systemic antibiotics (amoxicillin) for 5–10 days when they have undergone a more advanced GBR procedure. In addition, the patient should rinse with chlorhexidine solution for 3 weeks after placement of the GBR barrier. This could thereafter be switched to a 1% chlorhexidine gel which is gently applied in the wound area only once daily.

The e-PTFE membranes are removed after 6–8 months (either at the time of fixture installation or abutment connection). During this healing period, the patients are checked once a month for plaque removal and any complications.

Temporary dentures

Implant treatment and related bone augmentative procedures are usually associated with a situation where the patient needs a temporary solution during the respective healing phases. Clinical studies have demonstrated a clear correlation between membrane exposure and pressure from temporary dentures in the wound area. Hence, strict rules apply for the design of the temporary solution in conjunction with GBR. Ideally, fixed solutions such as Maryland bridges or conventional temporary bridges are the first choices if possible. If a temporary removable denture is necessary, it should be designed in such a way that no contact is present between the base of the denture and the soft tissue covering the GBR membrane. Furthermore, occlusal support of the denture is mandatory in order to prevent a pumping pressure when chewing.

Membrane removal

A non-resorbable barrier membrane is removed under local anesthesia. Technically the easiest way to approach the membrane is from the lateral aspect and to dissect it free from the covering soft tissue layer. Following this procedure, it is usually easy to remove the barrier from the underlying bone tissue. Great care should be taken to remove the entire membrane material. This is usually performed in conjunction with either implant placement or abutment connection. Biodegradable barrier membranes do not usu-

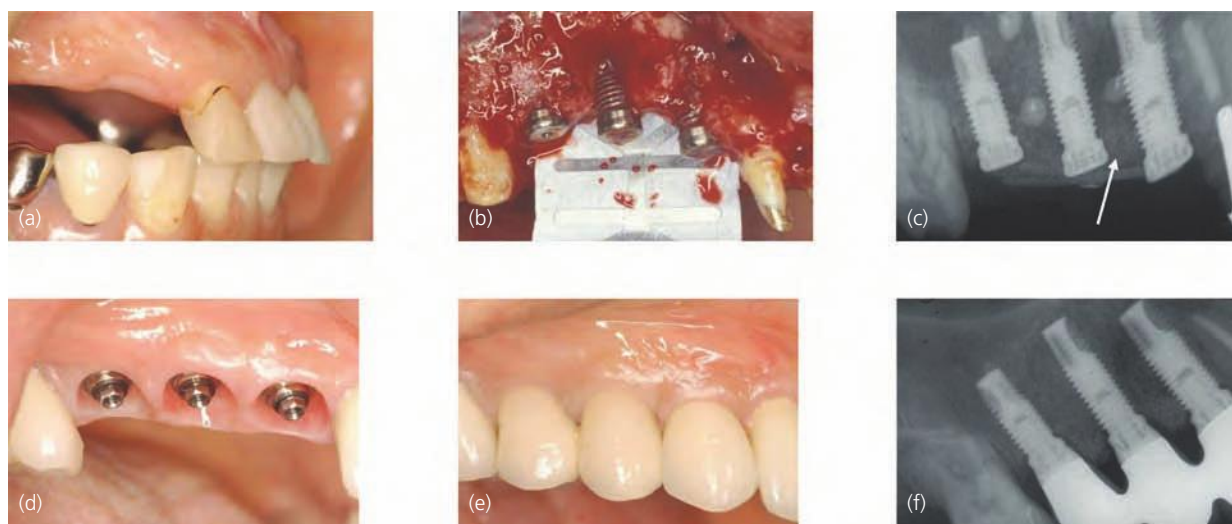


Fig. 21.7 (a) Edentulous area in the posterior maxilla. Both horizontal and vertical bone loss are present. Without bone augmentation in conjunction with implant treatment, a less than ideal esthetic outcome regarding the length of the clinical crowns would be evident. (b) Esthetic-driven implant placement. Implants are placed according to prosthetic planning transferred via a surgical stent. Note the level of the present bone in relation to the placement of the fixture heads. A titanium-reinforced GTAM has already been secured palatally by means of titanium fixation tacks. The defect area will subsequently be filled with a mixture of BioOss® and autogenous bone particles. Finally the membrane will be wrapped over the graft buccally and sealed with micro tacks. Suturing is according to the described protocol. (c) Radiograph showing the augmented area after 6 months of healing. Note the newly formed bone between the implants (compared to Fig. 21.7b). The tacks used for membrane fixation are also visible on the radiograph. (d) Clinical photograph after abutment connection and subsequent healing. (e) Final reconstruction: ProCera® crowns placed. Note the optimal length of the clinical crowns that matches the neighboring teeth. (f) Radiological follow-up after 1 year in clinical function. Note the bone height is maintained in between the implants. The bony support allows a stable soft tissue level around the superstructure.

ally require this procedure. Most biodegradable membranes on the market are designed with a resorption pattern of less than 6 months.

Clinical results of GBR

A variety of techniques and materials has been used to establish the structural base of osseous tissue for supporting dental implants. GBR is a surgical concept which has been in clinical use for well over two decades. It has undergone several developments and improvements and is nowadays considered a predictable treatment modality, once the previously described issues have been taken fully into account. Doubts have previously been raised regarding the quality and lasting capability of membrane-regenerated bone when being put into clinical function. Previous experimental studies have clearly shown the positive dynamics of this type of bone over time. Recently this has also been confirmed in several clinical studies. In a recent review study by Aghaloo and Moy,²³ the GBR technique was compared to different types of grafting procedures such as autogenous onlay, veneer (OVG), interpositional inlay grafting (COG), distraction osteogenesis (DO), and ridge splitting (RS). The data originated from a database search which identified 526 articles. Finally 335 articles met the criteria. Implant survival rate was 95.5% for GBR technique, 90.4% for OVG, 94.7% for DO, and 83.8% for COG. Hence GBR technique performed better or equal to more advanced bone grafting procedures using autogenous bone.

Another interesting clinical finding is that it seems slightly easier to augment bone in the maxilla compared to the mandible. The use of provisional restoration during the healing period seems to improve the result. Early implant placement also seems to be preferable if possible, due to alveolar ridge preservation, more favorable defect morphologies, and probably a higher regenerative capacity of the adjacent bone.²¹

Bone augmentation of atrophic jaw bone and, particularly, in the esthetic zone in the maxilla is a delicate and technique-sensitive procedure. The principle of GBR offers an alternative that is less resource demanding and also results in less morbidity for the patients. Predictable results can be obtained if a thorough understanding of the biological principles is applied in the clinical setting (Fig. 21.7).

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Chapter 22

Implant Placement in the Posterior Mandible

Bo Rosenquist

This chapter is a comprehensive overview of alternative methods to rehabilitate edentulous areas with compromised bone situations in the posterior lower jaw. The anatomy, with the mandibular canal as a limiting structure, is carefully described. Different methods to solve problems, including short implants, surgical positioning lateral to the nerve bundle, nerve transposition, and nerve lateralization, are described in detail. Alternative surgical methods to improve the bone situation like onlay grafting and distraction osteogenesis are also discussed. The author emphasizes the importance of a careful, skilful surgical technique and includes a section on complications that may arise with this type of surgery.

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Anatomy of the posterior edentulous mandible

Normal topography

The curvature of the alveolar process of the mandible is narrower than the body itself; the posterior parts of the alveolar process are positioned much more lin-

gually than the mandibular body. In the second and third molar area the bony substance of the oblique line, running from the coronoid process, is superimposed on the outer alveolar plate because of this divergence of the alveolar process and mandibular body. This gives an impression of a thick alveolar plate. Lingually, the temporal crest from the medial part of the coronoid process follows the course of the oblique line. Behind the third molar these two lines form the retromolar triangle which continues anteriorly as the alveolar crest. To accommodate the comparatively thicker roots of the molars the alveolar crest is wider and the crest flatter in the posterior than in the anterior parts of the mandible. Lingual to the second and third molars the downwards sloping mylohyoid line adds to the thickness of the superior parts of the alveolar crest. Hence, a cross-section of this part of the mandible is angulated lingually.

In the first and second molar area there is a shallow concavity in the lingual surface, the submandibular fossa. Further anteriorly, in the area corresponding to the premolars, the more distinct sublingual fossa is found.

As the oblique line continues anteriorly and inferiorly it gets less pronounced which gives the impression of a thinner mandible. The orifice of the mental canal, the mental foramen, is found in the buccal cortex, between the roots of the first and second molar, usually a couple of millimeters below the apices. As the curvature of the mandible increases in this region, the anterior rim of the orifice is sharper than the posterior.

The mandibular canal

The mandibular neurovascular bundle enters the mandibular canal through the mandibular foramen. This is a wide opening situated approximately in the center of the lingual surface of the mandibular ramus, hidden behind a thin, bony process, the lingula. During the first 8–10 mm the canal runs close to the lingual cortex. As it descends it moves to a more central position in the bone in a smooth curve downward

and forward into the mandibular body some 6–8 mm from the mandibular base. The curvature of the canal is less pronounced as it continues in the mandibular body. This means that the canal runs continuously closer to the mandibular base until it reaches the area between the first and second molar from where it moves upwards to the mental foramen in an increasingly sharp curve. As the canal moves inferiorly it moves lingually and reaches its most lingual position at its most inferior position, between the first and second molar. From there it runs closer to the buccal cortex. When the mandibular canal forks into an incisor canal, containing the anterior plexus of the nerve, and a mental canal (usually called the mental foramen), containing the mental nerve, the canal is positioned approximately 3–4 mm from the buccal surface. What is known as the mental foramen is actually a canal through which the first 2–4 mm of the mental branch of the mandibular nerve run before it leaves the mandibular body apical to, and between, the first and second premolar. However, in a few cases its location may vary from the canine to the first molar.¹ The mental canal usually runs perpendicular to the sagittal cardinal axis of the head. Thus it runs slightly posteriorly to the surface of the mandible after leaving the mandibular canal but the pattern of emergence seems to vary between population groups.² The posterior direction may be readily seen in lateral radiographs and has been interpreted as a loop.^{3–5} The length of the loop has been measured at between 0 and 7.5 mm.^{6–8} However, others have shown that the loop is a radiographic artifact rather than an anatomic structure and that it should have no impact on implant surgery.^{5–7,9} The anterior and posterior walls of the mental canal are frequently not parallel, but converge towards the foramen, which may explain the differ-

ence in findings between radiographs and clinical findings.⁷

As the upwards curve of the mandibular canal is accentuated close to its ramification, the mental canal reaches the mental foramen slightly from below. The incisor canal continues anteriorly after the furcation of the nerve. The size of the canal anterior to the furcation varies considerably; from being the same size as the main canal to a diminutive, almost invisible canal. However, even in cases where the canal is initially rather wide it soon narrows as it runs anteriorly.

The expression "mandibular canal" may give a false impression of a rather robust structure with thick cortical walls. In a few cases this may be true (Fig. 22.1a) but in most cases the walls are fairly thin, in some cases they seem almost non-existent (Fig. 22.1b). Normally the walls of the mandibular canal are perforated by small vessels and nerve fibers leaving the neurovascular bundle to the teeth. It is not known whether these perforations eventually disappear when the vessels and nerve fibers atrophy after teeth have been extracted. The mental canal seems to be usually surrounded by solid cortical walls, which are a continuation of the buccal cortex.

The periosteum that covers the exterior surface of the mandible continues into the mental canal and along the mandibular canal as an endosteum covering the surface of the walls of the canals.

The mandibular neurovascular bundle

The inferior alveolar nerve, sometimes called the mandibular nerve, is the third branch of the fifth cranial nerve, the trigeminal nerve. The mandibular

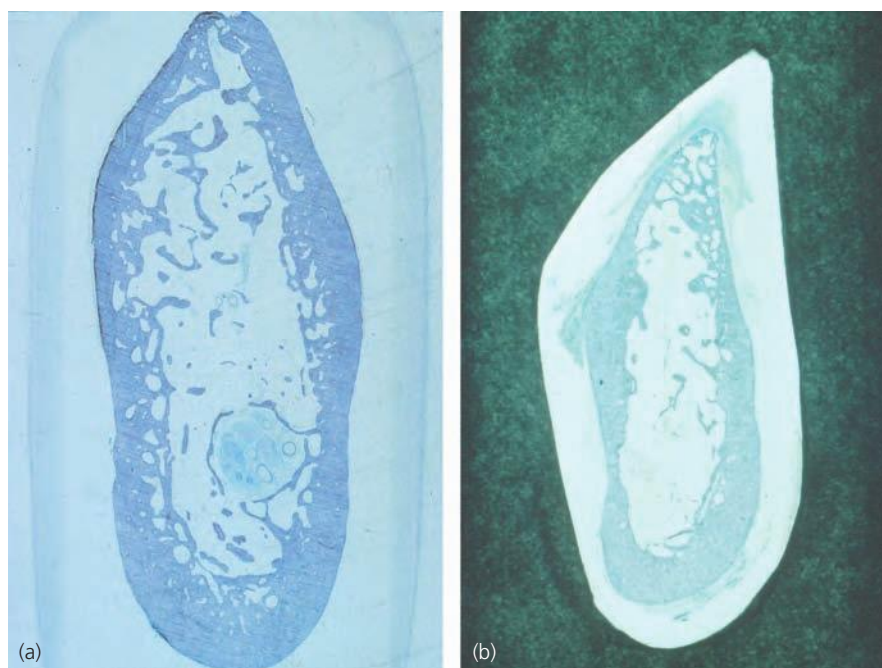


Fig. 22.1 (a) Cross-section of the mandible. Mandibular canal with a thick cortical wall. (b) Cross-section of the mandible. Mandibular canal with an extremely thin cortical wall.

branch is a sensory, polyfascicular nerve. This means that all information initiated by a stimulus on a specific area of the lip, the mentolabial fold, the chin, the teeth, the alveolar mucosa or the buccal soft tissues close to the mental foramen is transmitted from the periphery via the Gasserian ganglion to the nucleus in the brain by a specific nerve fiber. The nerve fiber is surrounded by a sheath, the endoneurium. Nerve threads from receptors in a certain area and their endoneurium are grouped together and surrounded by a sheath of dense connective tissue with thick collagen fibers, the perineurium, to form fascicles. These fascicles are embedded in loose connective tissue and surrounded by another sheath made up of thick collagen fibers, the epineurium, which constitutes the surface of the nerve. Internal blood supply is delivered by vessels in the loose connective tissue surrounding the fascicles, and external blood supply is from vessels running along the nerve outside, but sometimes integrated in the epineurium. While in the bony mandibular canal, the nerve is surrounded by the endosteum. Along the mandibular canal, dental and interdental terminal branches, the dental branches, enter the teeth and receive information from the pulps while the interdental fibers perforate the interdental septa and supply information from the periodontal ligaments and the gingival papillae.

At the furcation, the dental and mental nerve fascicles are separated into two nerve trunks; the dental fascicles continue anteriorly as the anterior plexus, while the mental fascicles, which were positioned below the dental fascicles in the alveolar nerve, form the mental nerve and leave through the mental canal. At the orifice of the canal, the mental nerve usually appears as two or three branches that are further divided to serve the skin and mucous membrane of the medial and the lateral parts of the lip, and the mucosa of the labial alveolar surface.¹⁰

As the nerve is sensory, damage cannot lead to motor impairment. However, it may result in sensory deficiencies in the whole or parts of the reception area of the nerve, depending on the number of and which specific fascicles have been damaged.

Resorption patterns of the alveolar ridge

As the alveolar crest is wider in the premolar and molar area than in the anterior region, vertical resorption is slower in the posterior parts. On the other hand, molars and premolars are usually lost earlier than incisors. Thus when patients seek implant placement, the posterior regions are usually equally or more resorbed than the anterior.

In the posterior molar area resorption usually results in a wider arch and a wider crest as resorption reaches the oblique and mylohyoid lines. In the first molar and especially in the premolar area, two different resorption patterns are seen, although combina-

tions of the two are common. Whether the resorption mode is due to the angulation of the alveolar crest or genetically determined is not known.

The usual pattern is vertical resorption. This results in a flat and rather wide crest. If the resorption is moderate, leaving more than 10mm height of bone superior to the mandibular canal, implant placement is usually uncomplicated. However, as resorption continues, the height above the mandibular canal is reduced and in advanced cases, where resorption reaches the level of the mandibular canal, the foramen is found on the top of and even slightly lingual to the top of the crest with part of the alveolar nerve positioned under a thin layer of bone or even directly under the alveolar mucosa.

The other, less frequent, resorption pattern is lateral resorption. This results in a narrow crest, in advanced cases in a very high, thin ridge made up of cortical bone which is totally unsuitable for implant placement.

General considerations

Placement of implants posterior to the mental foramen presents problems not usually found in the anterior parts of the mandible. Modern implantology as presented by Brånemark was developed for placement of implants between the mental foramina in totally edentulous mandibles. In this area, the mandible consists of thick buccal and lingual cortical plates with only a narrow space of cancellous bone in between. Implants can thus usually engage and be stabilized by both the buccal and lingual cortical bone as well as the cortex of the base and of the crest. Excellent initial stability is usually obtained.

The situation posterior to the mental foramina is different. The mandibular body is usually wider and the upper, lingual, and buccal lateral cortices are thinner. Thus, stability offered by the upper cortex is often inadequate and in most cases stability cannot be expected from the lingual and/or the buccal cortex as the mandible is too wide. The mandibular neurovascular bundle necessitates the use of shorter implants which cannot be secured in the cortex of the base of the mandible. In addition, the cancellous bone seems to be substantially less dense and varies more in density than is the case between the foramina. Thus, initial stability comparable to that obtained in the anterior region cannot be expected and it follows that the success rate is lower.¹¹

The surgical technique has to be adapted to save the thin upper cortex. Where counter-sinking is part of the procedure this may have to be done very gently so as not to destroy the cortical fit of the implant. In cases where the height of a very thin alveolar crest has to be reduced to allow the placement of implants, the reduction may have to stop short of the width equal to the diameter of the implant or no cortex may be left to stabilize it.

In a clinical situation it is easy to misjudge the inclination of the lingual surface of the mandible. The surgeon has to be aware of the risk of perforating the lingual cortex when drilling the implant seats. Due to the anatomy of the mandible this risk is especially high in the premolar and second/third molar areas. The lingual vessels should normally be positioned halfway between the lingual cortex and the midline of the floor of the mouth but the floor of the mouth is richly vascularized with minor vessels and severe, even fatal, bleeding has been reported after perforations of the lingual cortex.¹²

There is a persistent rumor among surgeons that if you have a very gentle hand when drilling the implant seats you can feel when you reach the roof of the mandibular canal, and that implants may obtain extra stability by being secured into the cortex of the roof of the canal. Any such procedures should be strongly discouraged; the thickness of the roof varies but even in the most extreme cases is not thick enough to bring about any sensation of increased resistance (Fig. 22.1a, b). Another rumor is that if infiltration anesthesia is given to the patient instead of a mandibular block, the surgeon could be warned when drilling close to the canal in the posterior part of the mandible. Again, this is a highly unpredictable procedure not supported by any studies, and is not recommended.

Usually, the molars and premolars are lost before the incisors and canines. After a long period of edentulism in the posterior mandible, extensive alveolar resorption may have occurred. This has two clinical implications; the different levels of the crest make esthetic reconstruction difficult without bone onlays or distraction osteogenesis, while elongation of the antagonists in the maxilla may make these procedures impossible because of lack of intermaxillary space. In extreme cases, elongation of the antagonists may make implant therapy impossible without a segmental osteotomy of the premolar/molar area in the maxilla.

Another difficulty in the posterior part of the mandible should be mentioned. To increase stability of the prosthetic reconstruction, the implants should be placed in the form of a curve. In the incisor region this automatically follows the anatomy of the mandible, but in the posterior area implants will tend to be placed in a straight line. The surgeon should consider alternative techniques to increase stability by varying the positions of the implants.

Implant placement superior to the mandibular canal

Conventional implant placement in the posterior mandible may be associated with the risk of nerve damage.^{13,14} However, as long as the distance between the superior cortex and the mandibular canal is 13 mm or more, implant placement in the posterior mandible is little different from elsewhere if the difficulties

mentioned in the preceding section are observed. The distance between the surface of the alveolar crest and the roof of the mandibular canal can relatively accurately be calculated by means of a panoramic radiograph once the position of the mental foramen has been clinically established. When the distance is less, more accurate measurement becomes imperative. Numerous methods to establish the location of the mandibular canal have been presented.^{15–17} When extreme accuracy is needed, a surgical guide based on a three-dimensional (3-D) model should be made and used during surgery.¹⁸

Nerve damage may occur even if the canal is not directly traumatized during implant surgery. The implant seat is usually drilled 0.5–1 mm deeper than the length of the corresponding implant. When working close to the canal it is tempting to be cautious and drill less deep. This may result in the implant being placed a little deeper than the seat allows, compressing the apical cancellous bone, and, indirectly, the roof of the canal, without the surgeon realizing. This results in pressure on the neurovascular bundle. If the compression is minor, only a part of the sensory area may lose sensation temporarily. A more severe compression may lead to compartment syndrome in the nerve and permanent nerve damage with anesthesia, paresthesia or hyperesthesia. Should the surgeon observe any such symptoms postoperatively, the pressure on the nerve should immediately be eliminated, either by unwinding the implant one or two rounds or by open exploration of the nerve.

The use of wider implants

To compensate for the reduced bone-implant contact area when shorter implants are used, wider implants may be considered. These also have an esthetic advantage in the posterior area; the wider neck of these implants mimics the size of the cervix of the molars. Both the esthetic and functional results may therefore be enhanced.

When using wider implants the surgeon should be aware that with increased diameter the speed at the periphery of the drill and implant are exponentially increased. Gentle drilling and placement of the implant are imperative to avoid failures due to overheating of the bone.

Placement of implants lingual to the neurovascular bundle

To allow the use of longer implants, placement lingual to the mandibular canal has sometimes been recommended. The impression that the implants are placed lingual to the neurovascular bundle and secured into the cortex at the base of the mandible may be given by illustrations such as Fig. 22.2. However, as can be seen in Fig. 22.3, the bone lingual



Fig. 22.2 Nerve transposition has been performed on the right side. As bone chips have been placed between and lateral to the implants the former alveolar canal can no longer be seen across the implants. As the buccal cortex has healed, a new “retromolar” foramen has been formed posterior to the implants on the right side. On the left side a so-called “lingual placement” of the implants has been performed. Note that the canal can still be seen across (buccal to) the implants. A false impression is given that the implants are secured into the base of the mandible but in reality are placed into the floor of the mouth.



Fig. 22.3 Set of cross-sections of a mandible from the mental foramen (upper left) to the third molar area (lower right). The amount of bone available for implant placement lingual to the neurovascular trunk is insufficient.

to the mandibular canal is too thin to allow insertion of implants. To obtain a result as shown in Fig. 22.2 the implants have to be angulated to perforate the lingual cortex immediately above or at the level of the canal and the implants are thus inserted into the floor



Fig. 22.4 Lingual placement of an implant. Note how the implant has been unfavorably angulated to avoid interference with the neurovascular bundle.

of the mouth. This may have the advantage of double cortical anchorage but the method is highly dangerous and may even be life threatening. As has been discussed above, fatal incidents have been reported after accidental perforations into the floor of the mouth. Due to the resilient tissues in the floor of the mouth, bleeding may pass unnoticed until it suddenly manifests itself as an airway obstruction.^{12,19} As the implant seat is drilled without visible or easily calculated reference points, laceration of the neurovascular bundle is also highly probable.

Of course, it may be argued that this technique allows for a bicortical anchorage of the implant, and in a few selected situations this technique may have its place. However if the distance to the mandibular canal is short, the implants have to be angulated in a way that makes it difficult to achieve a satisfactory esthetic and functional prosthetic reconstruction (Fig. 22.4); in cases where the height above the canal is sufficient, a safer choice would be to choose to place a shorter and maybe wider implant above the canal rather than taking the risk of placing it lingual to the nerve bundle.

Crestal split

Sometimes an advanced lateral resorption pattern of the alveolar crest results in a high but very narrow crest unsuitable for implant placement, even if implant perforations both lingually and buccally are accepted. In such cases, the intermaxillary space permitting, a so-called crestal split may be considered.^{20–26} Typically the method is used in the anterior maxilla but it can be adopted for use in the posterior mandible as well. The crest has to have a wide base containing cancellous bone that runs well into the top of the crest and the cortex must not be too thick. In these cases success rates in the range 85–95% have been reported.^{20,21} However, in the posterior mandible very few cases qualify for this procedure. The bone has to be elastic to permit bending or a controlled breaking. Thus the patient has to be young. In most cases the thin crest is made up of a unified lingual and buccal

cortex without any cancellous bone in between. This makes a split highly unpredictable or even impossible. In most cases other methods are to be preferred.

Surgical technique

After an incision has been made along the top of the crest, a lingually based flap is raised (Fig. 22.5). A buccal flap only about 2mm wide is raised, to preserve the blood supply to the buccal cortical plate. A thin groove is drilled along the top of the crest until cancellous bone is reached. A chisel is cautiously driven well into the groove by a mallet while care is taken not to interfere with the neurovascular bundle. The chisel is then gently rotated until the gap widens and, in most cases, a green-stick fracture is produced at the base of the buccal cortex which is nourished by the attached periosteum. If the cortex is thick or only a short length of the crest is involved in the operation, additional vertical cuts may have to be made at both ends. Vertical incisions for these cuts should be placed anterior and posterior to the bony cuts and narrow vertical flaps raised to expose the area planned for the cuts. The implants can then be inserted in the gap. In some cases it is not even necessary to prepare a seat.

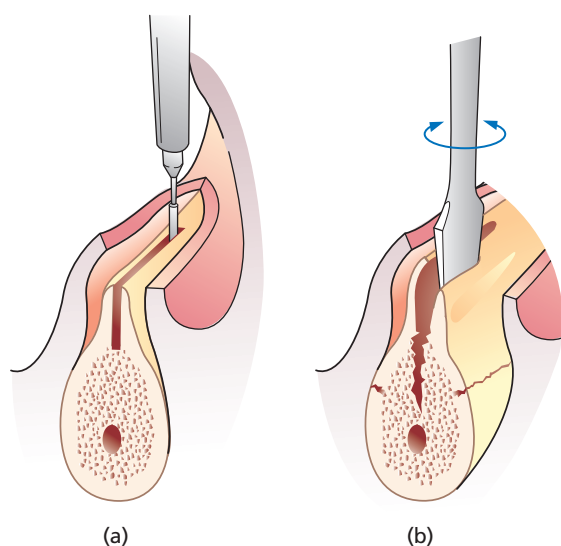


Fig. 22.5 Schematic sketch of a crestal split. A groove is made with a fissure bur along the crest into the cancellous bone, and a chisel is gently inserted and twisted until a space wide enough for insertion of the implants is created.

The gap between the implants may either be covered by a membrane and / or the space filled with a particulate bone graft. If the buccal bone has fractured it has to be secured by lag screws into the lingual cortex to provide initial stability for the implants. Soft tissue covering is produced by the lingual flap; this may sometimes be difficult. Second-stage surgery can be performed after 5–6 months.

Onlay augmentation in the posterior mandible

Indications for augmentation of the posterior mandible may be esthetic as well as functional. An esthetic problem is found in cases where the edentulous posterior part of the mandible is resorbed to a level well below the marginal level of the anterior, dentulous area. A residual crest thinner than 4mm or with a residual height above the mandibular canal less than 7–8mm presents a functional problem. In both cases, augmentation could be one of several possible solutions.

Onlay grafts and implant placement can be performed as a one-stage or a two-stage procedure, with autografts, allografts, xenografts, mixtures of these and various bone cements.^{27–30} Block grafts or particulate grafts may be used. At present, autogenous cancellous bone grafts seem to be the “gold standard” against which the merits of other grafts are measured.³¹

Vertical onlay

There are two limitations associated with augmentation of the posterior mandible; the antagonists in the maxilla may have elongated, leaving too little intermaxillary space for a prosthetic reconstruction. As has been discussed earlier, a segmental maxillary osteotomy with superior displacement of the osteotomized segment may be considered in these cases. The other is the problem associated with covering a vertical graft while preserving the depth of the vestibule. The vestibule is usually shallow in the molar area, even more so after alveolar resorption extensive enough to make an onlay graft necessary. When an onlay graft is covered there is a tendency for the ves-

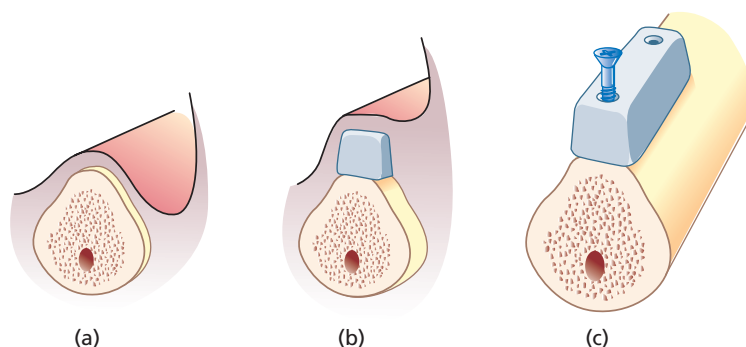


Fig. 22.6 Schematic sketch of a horizontal onlay graft. The depth of the vestibule has been reduced.

tibule to be reduced even further; in extreme cases it can be totally lost leaving the augmented mandible lying in the buccal soft tissues (Fig. 22.6). A second procedure, either a preoperative tissue expansion or a postoperative vestibuloplasty with grafting of attached gingival, should thus be included in the therapy plan when augmentation is planned in the molar area.

A cortico-cancellous block is usually preferred when the height of the posterior mandible is to be reconstructed. Adequate volume of this material is easily available from the iliac crest and, in contrast to most autogenous and allogenic alternatives, it offers stability to implants during the time it acts as a scaffold for new bone formation. The drawback with this material is that the degree of resorption is unpredictable. It is imperative to carve the transplant to an exact fit and to secure the transplant firmly to the recipient. As the residual amount of bone is unpredictable a two-stage procedure is preferred where the implants are inserted about 4 months after the transplantation.

In a few selected cases another approach may be chosen; if adequate stability can be obtained by inserting the implants one third to two thirds of their lengths into the mandible, compressed cancellous bone mixed with 20–30% allogenic bone substitute may be added around the implants to form a crest of adequate size for the implants. In this case the allogenic bone is used as a temporary filler, to increase the volume of the bone and, more importantly, to reduce the speed of resorption to allow adequate bone formation. The use of a contoured titanium mesh membrane to reduce the pressure on the graft is highly recommended in these cases. Alternatively,

the implants themselves may reduce external pressure on the graft by acting as “tent poles”.

Lateral onlay

There are three principle methods for widening a thin alveolar crest. Usually cortico-cancellous block grafts from the chin or lateral part of the corpus/ramus are used. Again, the graft should be contoured to a perfect fit to the recipient surface and secured by screws (Fig. 22.7a). If the cortical plate is thin, the vascularization offered by the recipient is usually less than when onlays are used to increase the vertical dimension. Hence, the risk for resorption may be higher and the “take” of the graft is usually slower and more unpredictable. The procedure is usually a two-stage procedure. When implants are placed 4–5 months after the transplantation there is still a risk of the graft loosening from the recipient crest.

In some cases vertical concavities with the diameter of the implants can be drilled into the buccal surface of the thin crest. The implants are then partly inserted into these concavities and secured apically in the cortex of the wider mandibular body. A particulate bone graft or particulate autogeneous bone mixed with hydroxyapatite could be packed between and buccal to the implants/crest and covered with a titanium membrane and left to heal for at least 6 months (Fig. 22.7b).

A third method is downfracture of the crest.³² After an incision 2–3 mm buccal to the top of the crest a vestibularly based buccal flap is raised and a horizontal cut is made along and almost through the crest, preferably with a thin fissure bur. The tip of the crest is then downfractured buccally to add to the width of

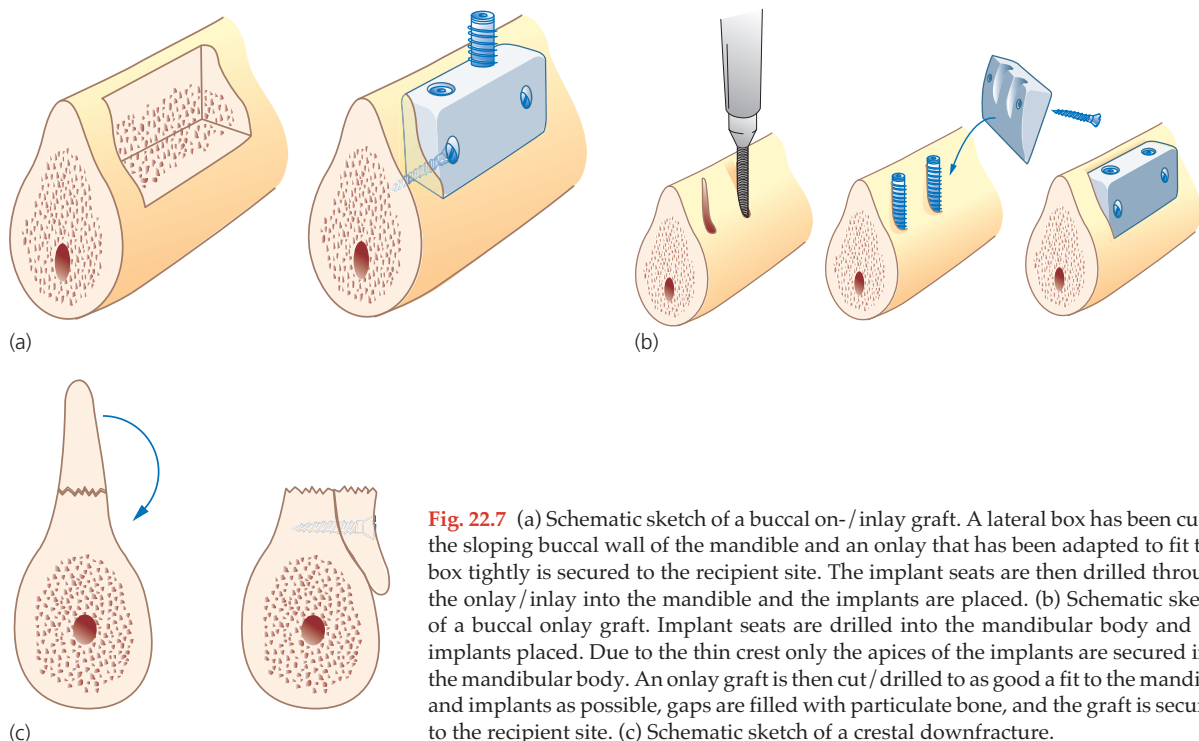


Fig. 22.7 (a) Schematic sketch of a buccal on-/inlay graft. A lateral box has been cut in the sloping buccal wall of the mandible and an onlay that has been adapted to fit this box tightly is secured to the recipient site. The implant seats are then drilled through the onlay/inlay into the mandible and the implants are placed. (b) Schematic sketch of a buccal onlay graft. Implant seats are drilled into the mandibular body and the implants placed. Due to the thin crest only the apices of the implants are secured into the mandibular body. An onlay graft is then cut/drilled to as good a fit to the mandible and implants as possible, gaps are filled with particulate bone, and the graft is secured to the recipient site. (c) Schematic sketch of a crestal downfracture.

base of the crest (Fig. 22.7c). The advantage of this method is that the blood supply to the “transplanted” bone is preserved by the soft tissue attachment. The drawbacks are that the height of the crest is reduced, the final width of the crest cannot be controlled, and attached gingiva is lost on top of the newly formed crest.

Distraction osteogenesis in the posterior mandible

Alveolar distraction was introduced by Chin and Toth in 1966.³³ This procedure has advantages compared to onlay grafting; the result is usually more predictable and there is no soft tissue displacement.^{34–37} The latter is especially important in the posterior parts of the mandible as the vestibule and the attached gingiva can be preserved. On the other hand distraction implies patient compliance during distraction and consolidation, more visits, and the added cost of the appliance. Control of the appliance and the vectors can be complicated,^{38–40} especially as the space for the appliance in the vestibule is limited in the posterior mandible. It follows that mechanical problems with the device as well as unplanned displacement and tipping of the transport segment are not uncommon.⁴¹ Distraction involves pressure on both the reference and transport segment. Fractures of the reference segment, the basal bone, have been reported.⁴² Pressure on the superior, transport, segment may induce not only suture dehiscence but progressive surface resorption with eventual collapse or fracture of this segment if it is not thick enough.^{38,40,43–46} This means that distraction is increasingly more difficult to perform as the distance between the alveolar crest and the canal is reduced. To prevent fractures a bone height above the canal of at least 5–6 mm is recommended.^{33,41,43} The procedure seems to be well tolerated by most patients⁴⁷ but a high incidence of complications including bleeding, transient nerve damage, unplanned displacement or fractures of bone segments, malfunctions or fractures of appliance may occur; incidences between 0 and 100% have been reported.^{42–45,48,49} This may indicate that thorough preoperative planning and gentle surgical technique is a prerequisite for success. Awareness of this

together with the relative complexity of the procedure may limit the usefulness of the procedure to rather few, carefully selected cases.

Surgical technique

A horizontal incision is made in the vestibule corresponding to the area of the alveolar crest to be distracted (Fig. 22.8). Care should be taken not to damage the branches of the mental nerve at the anterior end of the incision. Superiorly directed relief incisions are made at both ends. At the posterior end this may be replaced by an elongation of the horizontal incision. The flap is raised to expose the lateral part of the mandible. However, the top of the crest should be exposed as little as possible. While the index finger is placed against the lingual side of the mandible a horizontal osteotomy is made almost through the mandible not closer than 3 mm to the anticipated level of the neurovascular bundle. A medium-sized fissure bur is ideal for this purpose as it is readily felt by the index finger before the soft tissue is lacerated; a reciprocal saw is harder to control. Two vertical cuts are made through the crest at both ends of the horizontal cuts, slightly converging downwards. With the index finger still in place, the transport segment is then fractured from the rest of the mandible by means of a chisel. The distractor is then secured to the transport segment and the basal bone with mini-screws and activated to check that the transport segment may move freely and that the vectors are correct. Finally the distractor is inactivated and the flap resutured. After a latency period of 1 week the distractor may be activated for distraction by 0.5 up to 1.0 mm/day. An overcorrection of 1–2 mm is recommended. After a consolidation period of 4 months the implants are placed. The distractor should be in position until the implants are placed.

Penicillin V is administered: 2 g preoperatively and 2 × 2 g postoperatively until the suture lines are healed.

Nerve transposition

There is widespread confusion in the literature as to what this procedure should be called. The terms lat-

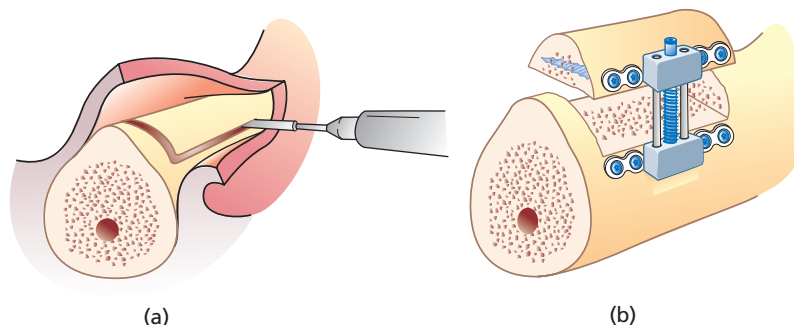


Fig. 22.8 Schematic sketch of the principles of distraction osteogenesis in the posterior mandible.

eralization, transposition, and repositioning are frequently used interchangeably. In this text, *nerve lateralization* includes procedures where the nerve is manipulated laterally posterior to the mental foramen. *Nerve transposition* is defined as a procedure where the neurovascular bundle is manipulated laterally after the incisor branch has been cut. This procedure was originally used to facilitate orthognathic surgery of the anterior mandible.⁵⁰ The area of the mental foramen is thus included. The expression *nerve repositioning* means "placed in the original position"; it is not applicable in this context and should be avoided.

The main advantage of nerve transposition and nerve lateralization is that both these methods permit installation of implants in cases where other methods would fail or be impossible to use. They may thus be regarded as "rescue operations". Both methods offer bicortical implant stability. The implant survival rate is high, around 93–95%.⁵¹ Marginal bone resorption does not differ from what is found after conventional implant placement^{51,52} and less than after distraction.⁴⁹ Once the surgeon is familiar with the procedure it is a quick procedure that can be made under local anesthesia. Nerve transposition may also be used to preserve neurosensory function in association with mandibular hemiectomy/reconstructions.

However, the procedure involves repeated trauma to the neurovascular bundle; the first trauma occurs when the incisor branch of the nerve is cut and a reactive swelling of the nerve follows. This swelling may induce a so-called compartment syndrome within the nerve trunk resulting in a "strangulation" of the vessels and hence lack of neurosensory function. The second trauma occurs as the nerve trunk is manipulated out of the mandibular canal.⁵¹ The alpha-fibers of the nerve are highly sensitive to traction. If the nerve is bent at any point up to approximately 5%, temporary neurosensory disturbances will occur. Normal sensation may be expected within 4–6 weeks although it may take considerably longer in some cases.^{51,53–55} However, if the traction is more than 5% the perineural vessels will rupture and permanent lack of neurosensory function or deficit function will result.^{53,56} Immediate postoperative loss of sensation or deficit nerve function can be expected in most cases but gradually normal neurosensory function is regained. Six to 12 months postoperatively only a few percent of the patients have persistent loss of sensation and some of these patients do not seem aware of this.^{51,56–58} In a study on 100 cases⁵¹ approximately 80% of the cases had no or deficient sensibility 1 week postoperatively, 25% 6 months postoperatively and 6% 18 months postoperatively. If corticosteroids are administered to reduce the peri- and postoperative swelling of the nerve, normal function seems to be regained a little earlier but the end result is approximately the same.⁵⁹ Although these figures may seem encouraging, patients seem to find it harder to accept nerve disturbances following nerve lateralization/

transposition than after, for example, sagittal split operations. It may be the implant patients are generally older and find it harder to adapt or there may be some quality factor in the nerve disturbance not registered by the method used. Some authors have advised against the use of this method as a routine procedure.^{60–63} Devitalization of the incisors after nerve transposition has not been reported, not even after bilateral procedures.

As the cancellous bone of the mandible is widely exposed during surgery and as the devitalized buccal cortex is reused in particulate form, the administration of antibiotics is imperative to avoid severe infections that have occasionally been reported.^{51,57} In extensively resorbed cases where the "buccal window" will cover most of the buccal surface there is a certain risk of mandibular fracture, especially if the seats are drilled deep into or through the cortex at the base of the mandible leaving only the lingual cortex to stabilize the mandible.^{64–66} The risk is most acute 1 week postoperatively when the decalcification phase of the remodeling coincides with the patient regaining full chewing force. However, it should be pointed out that fractures in conjunction with conventional implant placement in the posterior mandible have been reported.^{67–69}

Surgical technique

A subperiosteal flap is outlined by an incision along or slightly lingual to the top of the alveolar crest with a deep relieving incision into the vestibule anterior to the position of the mental foramen. To protect the anterior branches of the mental nerve, this should not be positioned too close to the mental foramen or made too vertical. Usually it is advantageous to add a posterior relieving incision. The flap is raised to expose the crest and buccal surface of the mandibular body, the mental foramen, and the base of the mandible. The subperiosteal dissection should continue into the mental canal to free the endosteum from the surrounding walls. A panoramic radiograph can usually provide information about the approximate level and course of the mandibular canal; the mental foramen indicates the highest point of the canal in the mandibular body. With a thin dissector between the roof and the nerve in the mental canal, a horizontal cut, parallel to the base of the mandible, is made from a point at least 6–8 mm anterior to the mental foramen and at the level of the roof of the mental canal, until a point some 8–10 mm posterior to the planned position of the most posterior implant seat. This horizontal cut should be made through the cortex and 1–2 mm into the cancellous bone. A second cut, as long as and parallel to the first, is made below the anticipated level of the mandibular canal. This cut involves two difficulties: access below the mental foramen and estimation of the level of the canal. To facilitate access to the area below the mental foramen a very shallow cut could be made through the periosteum/

endosteum that covers the nerve branches as they leave the foramen. This will expose the neurovascular bundle, usually the two main branches, and allow the flap to be retracted a little further. A correct estimation of the level of the floor of the canal is crucial. If the cut is not made into the cancellous bone but into the cortex of the mandibular base, the subsequent steps of the procedure will be more difficult and there is a definitive risk for mandibular fracture. If the cut is made too high, the nerve will be damaged. All cuts are made by means of a thin fissure bur. When in doubt the cut could be prepared by using a thin round bur to localize the correct position of the cut. Once the two horizontal cuts are made, these are united at both ends, in front of the foramen into the cancellous bone and at the posterior end only just through the cortex to avoid accidental cutting of the nerve (Fig. 22.9a). The thin elevator is again inserted into the mental canal and the roof of the canal is cut with a fissure bur, starting from a position adjacent to the elevator and working upwards in a V-form to remove the cortical roof of the mental canal. The buccal bone plate encircled by the cuts is then gently removed (Fig. 22.9b, c). The course of the mandibular neurovascular bundle in the mandible, the furcation at the mental foramen and the incisor branch are exposed (Fig. 22.9d, e). Usually small remnants of bone on top and below the bundle have to be removed with an elevator to free it completely. The incisor branch is cut some 7–8 mm anterior to the furcation and, starting from the mental foramen area, the neurovascular bundle is carefully lifted out of the mandible (Fig. 22.9f). It should be noted that the main trunk will start to retract and swell as a response to the trauma when the incisor branch is cut (Fig. 22.9f).

When the nerve is dissected out of the mandibular body care should be taken not to bend the nerve as this will induce neurosensory disturbances. Usually the nerve is more easily removed and the bleeding less, the longer the patient has been edentulous, as the small nerve fibers and vessels that earlier ran to the teeth are vestigial. While the nerve trunk is gently protected the implant seats are drilled, making only small impressions in the basal cortex, and the implants are inserted. They should be lightly stabilized against the cortex in the base of the mandible but not necessary perforating it, as this may increase the risk for mandibular fracture. It has been shown that the nerve trunk may be repositioned into the mandibular body protected from the implant surfaces by a resorbable membrane⁷⁰ or even without a membrane⁷¹ (Fig. 22.9g); however the author prefers to avoid direct contact between the implant and the nerve trunk in case an infection develops around an implant or the implant has to be removed for other reasons.

The bone plate that was removed earlier is cut into 2–3 mm pieces that are packed between and buccal to the implants while the neurovascular bundle is allowed to leave the mandibular body distal to the most posterior implant (Fig. 22.9h). Eventually a new

foramen will be formed in this area (Fig. 22.2). Cover screws are placed and the flap resutured. The patient is given penicillin V (2 g) and corticosteroids (4 mg) preoperatively. Later the same day the patient is given penicillin V tablets (2 g) and corticosteroids (3 × 4 mg). Penicillin V (2 g) is given for another 10 days postoperatively and corticosteroids are given, reducing the dose as follows: 4 × 3 mg the first, 4 × 2 mg the second and finally 4 × 1 mg the third postoperative day. Fig. 22.9i shows the postoperative radiographic result.

In cases of extreme resorption where the neurovascular bundle is close to or on top of the crest, the superior cortex is removed to reach the nerve which should be transpositioned posteriorly and into the lateral wall of the mandible. In these cases the crest should be allowed to heal before the implants are placed and the procedure should thus be performed as a two-stage procedure where the implants are inserted approximately 6 months after the transposition of the nerve.

Nerve lateralization

Nerve lateralization is one of the procedures indicated when implants are to be inserted posterior to the mental foramen (preferable from the first molar) and the height above the canal is inadequate. The method seems to offer both advantages and disadvantages compared to nerve transposition; as the area of the mental foramen is not involved, the procedure could be expected to be faster and easier to perform, and as the incisor branch of the nerve is not cut, one trauma less is inflicted on the nerve. On the other hand localized traction has to be applied to the nerve when the implants are placed and postoperatively it may come to rest against the threads of the implant which may constitute an additional long-term trauma. As the anterior cut through the cortex is placed in an area where the nerve runs close to or, in some cases, actually within the buccal cortex when nerve lateralization is performed, there is an increased risk of accidentally cutting the nerve in this area.

Due to better access, implant placement in the area of the second premolar seems to be facilitated by nerve transposition. Otherwise the results of the two methods seem comparable; neither implant survival nor postoperative neurosensory function seem to differ significantly.^{72,73} Hence, the choice of method seems to be up to the personal preferences of the surgeon.

Surgical technique

The flap design is identical to that used in nerve transposition. It is important to expose the mental foramen to help localize the level of the canal but the dissection is not continued into the mental canal. In the area where implant placement is planned two

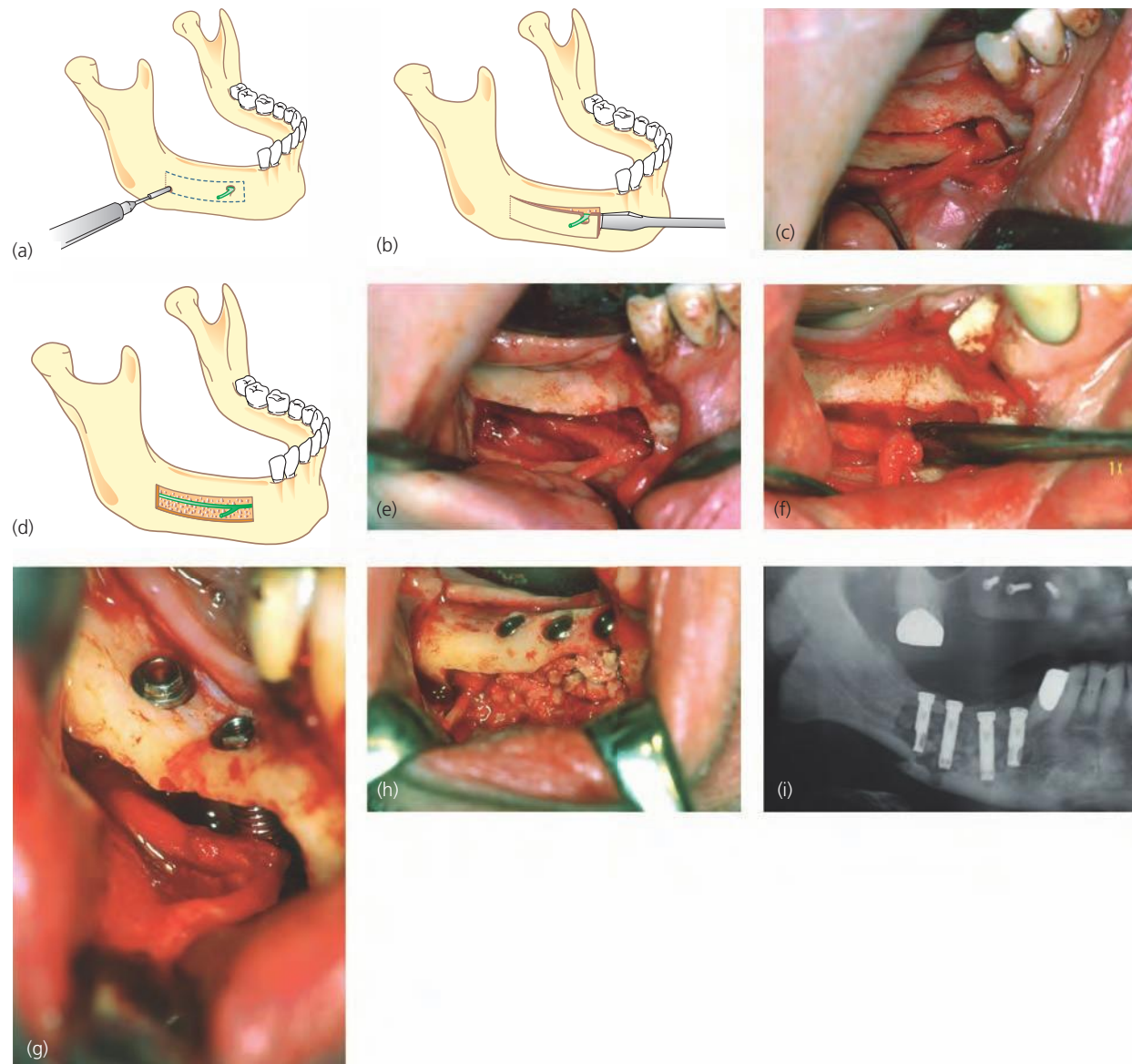


Fig. 22.9 (a) Schematic sketch of nerve transposition. The outline of the cortical window, later to be removed. The window includes the mental foramen. The superior, inferior, and anterior bone cuts are well into the cancellous bone; the posterior, however, is only almost through the cortex to avoid trauma to the neurovascular bundle. (b) The roof of the mental canal has been removed. The buccal window is gently being removed. (c) Clinical illustration of (b). (d) The buccal cortex has been removed. Schematic sketch. (e) Clinical view of (d). At the furcation the rather thick main trunk divides into a thinner mental branch and an even thinner incisor branch. Note that the nerve is situated more lingually in the posterior part of the mandible. (f) The incisor branch is transected and immediately starts to contract and swell as it is gently removed from the mandibular body. (g) The implants are placed. (h) The former buccal cortical window is cut into bone chips that are placed between and lateral to the implants. The neurovascular bundle now leaves the mandibular body posterior to the implants where eventually a "retromolar foramen" will be formed. (i) Radiographic view after implant placement.

horizontal cuts are drilled 1–2 mm into the cancellous bone, parallel to the base of the mandible, by means of a thin fissure bur, one about 2 mm superior to the anticipated level of the neurovascular bundle and the other below the mandibular canal. As in nerve transposition the level of the lower cut is the hardest to place correctly and any misjudgement involves the same risks. The horizontal cuts are then united by vertical cuts at both ends, at the posterior end only just through the cortex to avoid accidental cutting of the nerve (Fig. 22.10a). The anterior cut has to be made through the cortex which involves a certain risk of cutting the nerve if this is close to or within

the cortex. A more cautious cut that does not penetrate the cortex will make luxation of the buccal cortex and dissection of the nerve much more difficult, riskier and time consuming. To prevent accidental cutting of the nerve, preoperative tomograms are recommended.

When the buccal bone plate has been removed any small remnants of bone on top and below the nerve bundle are removed with an elevator to facilitate lateralization of the bundle. This has to be done more carefully than when transposition is performed as the medial part of the nerve bundle has to be accessible from a lateral approach. The neurovascular bundle is

then gently pulled 2–3 mm (or as required) out of the operation field. The author uses a home-modified instrument; a small dissector with the tip bent 120°. As has been pointed out above it is extremely important to apply only gentle traction to the nerve to reduce the risk of nerve damage. As in nerve transposition the nerve may start to swell as the vessels within the trunk rupture, but the swelling is usually less than when transposition is performed. The implant seats are drilled only a couple of millimeters into the basal cortex. The implants are placed and the neurovascular bundle is allowed to slip back into the mandible (Fig. 22.10b). Frequently the bundle contracts to lie against the treads of the implants. The long-term neurosensory impact of this is not known. To avoid a postoperative buccal concavity in the mandible the bone plate that was removed earlier can be cut into 2–3 mm pieces that are gently packed between and buccal to the implants and the neurovascular bundle. Any pressure should be avoided. Cover screws are placed and the flap resutured. The patient is given penicillin V 2 g preoperatively and 2×2 g postoperatively for ten days as well as corticosteroids 4 mg preoperatively and 3×4 mg postoperatively on the day of operation and 4×3 mg, 4×2 mg and 4×1 mg, respectively the following postoperative days.

Nerve repair

In principle, three types of damage may be inflicted on the mandibular nerve during implant surgery: superficial damage to the nerve sheath without damage to the nerve fascicles, damage to both the sheath and various numbers of fascicles, and complete interruption (transection) of the nerve bundle.

A solitary superficial injury to the nerve sheath is most likely to occur during drilling of an implant seat and during drilling the bone cuts for nerve distraction osteogenesis, transposition, and lateralization. This type of damage does not call for repair as normal function is usually re-established.⁷⁴

A combined injury to the nerve sheath and nerve fibers can be inflicted by deeper cuts during the procedures mentioned above, by pressure induced by inserting an implant too deep or by compartment syndrome brought about by swelling of the nerve due to external trauma, such as cutting of the incisor branch or rupture of multiple small dental vessels/nerve fibers. These injuries usually result in nerve entrapment by scar tissue that can itself worsen the neurosensory dysfunction and may make it permanent. It is thus important to establish the nature of the damage as soon as possible.

If the damage is caused by direct pressure from an implant inserted into the canal the implant should be rotated counter-clockwise until its tip is level with the roof of the canal. If the damage is caused by compression of the roof of the canal by an implant inserted further than has been drilled, the implant should also be rotated counter-clockwise to relieve the pressure on the nerve. However, sometimes an open procedure is needed to reduce the pressure on the nerve. Access to the area is described under the procedure for "nerve lateralization". In both cases action should preferably be taken within a week. Usually normal sensation will be regained within another week or two.

If some or all fascicles have been damaged due to swelling, the patient usually loses sensation in the lip/chin but normal sensation is usually regained in the region corresponding to the primarily undamaged fascicles. In a best-case scenario, depending on how much laceration of the fascicles has been caused by the trauma, sensation may return even in the areas served by the damaged fascicles. This usually happens within 3–6 months, up to a year. In some cases, however, an internal or external scar is formed. If such a scar has formed, sensation in the damaged fascicles will not return spontaneously. If an external scar is formed the nerve is gradually strangulated and sensation will be permanently lost in the whole sensory area of the nerve. In these cases, lysis should be performed to save the neurosensory function. Satisfactory results have been reported after external

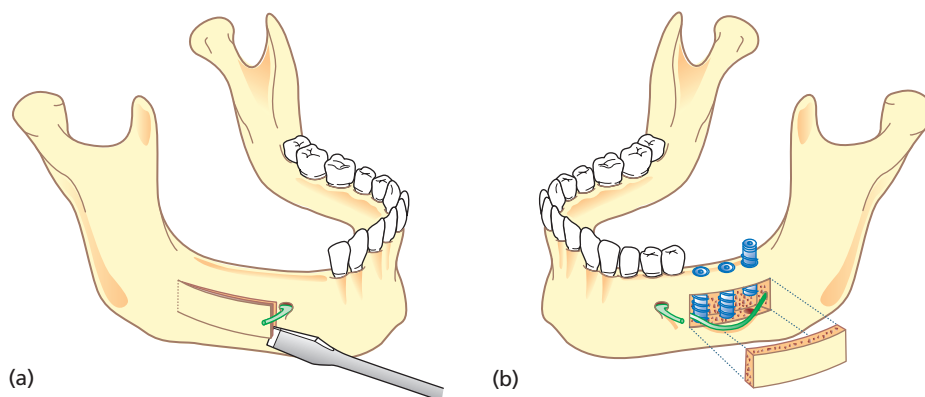


Fig. 22.10 (a) Schematic sketch of nerve lateralization. Outline of the buccal window. The mental foramen is not included in the window. (b) The cortical window has been removed, and the nerve trunk is gently pulled buccally as the implants are placed.

neurolysis as late as 12–24 months after the trauma.^{75,76} The procedure is best performed using 10× magnification. The damaged part of the nerve is exposed as described above, the bundle is gently pulled out of the canal and the scar tissue, which is usually easily identified, is excised by means of a pair of microscissors or a scalpel. Even internal scar tissue can be excised or the damaged part may be resected. Excision of an internal scar is a delicate task and is best left to an experienced microsurgeon. The author prefers to resect the damaged part, adapt the ends and suture the nerve.

The mandibular nerve bundle is elastic to a certain degree. Therefore, when the nerve is transected the nerve ends contract and a gap is formed between the ends. Only in exceptional cases will normal function be regained. The nerve should be repaired as soon as possible. The traumatized area is exposed as described above. If the cut is not clean the nerve ends should be trimmed into undamaged tissue and the ends are adapted. This, however, is usually not possible without tension due to the elasticity of the trunk and the necessary resection. An autogenous vein graft⁷⁷ or Gore-Tex tubing (Gore Company, Flagstaff, AZ, USA)^{78–81} may be sutured to the epineurium of the proximal and distal nerve trunks and used as a conduit for regenerative repair. However, this assumes that the gap is only a few millimeters wide,^{77,78} otherwise a nerve graft should be interposed. Again, this requires a specialist in microsurgery. The author prefers to perform a nerve transposition with transection of the incisor branch after which the ends can be adapted without tension. Ideally, each fascicle should be sutured to its counterpart. However, good clinical results may be obtained by removing the epineurium 3–4 mm from both cut ends, carefully aligning the ends without major consideration of adapting the individual fascicles and suturing the ends in this position. The sutures, as few as possible, are placed superficially in the perineurium.

One difficulty can be understood from the discussion above. In quite a few cases normal sensation will return after moderate damage to the mandibular nerve. Thus, as nerve repair is not a familiar procedure to most surgeons and not always successful there is a good case for a “wait and see” strategy. On the other hand delay involves an increased risk for permanent damage^{82–85} as nerve cells in the trigeminal ganglion are successively lost after transection of the nerve. It seems that nerve repair prevents further loss of nerve cells in the ganglion but those already lost at the time of repair are not re-established.⁸⁶ Unfortunately, the sparse clinical literature offers very limited evidenced-based knowledge on methods and timing of mandibular nerve repair.^{87,88} However, a useful rule of thumb may be that transections and deep cuts of the nerve should be repaired immediately by tension-free primary suturing, while it may be defensible to postpone nerve repair up to 6 months after superficial damage.^{75,76}

Ethical considerations

Before every single operation the surgeon should consider the balance between what the patient may gain and the risks involved. Implant surgery is an elective procedure; it does not save lives or prevent disablement. Hence, the surgical risk should be moderate. Unfortunately, it is not possible to give general recommendations on what level of risk is justifiable; that depends on each case and each patient. Some of the procedures discussed above are well proven and should be safe, others involve moderate risks and discomfort, while still others involve considerable risks for failure, pain, impairment, and may even prove fatal.

Although some of these procedures require considerable practice and skills, the real challenge for the surgeon is to judge each case from its own surgical as well as psychological merits to be able to offer the patient individual advice. The author has a conservative rule of thumb: “Would I recommend this procedure to someone in my family?” If the answer is no, why recommend it to a patient?

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Chapter 23

Autogenous Bone Harvesting Techniques

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This chapter is an overview of different techniques for harvesting autogenous bone graft. The needs for bone graft based upon anticipated volumetric and structural needs are discussed. The chapter describes intraoral harvesting sites, including mandibular symphysis, ramus and retromolar area, maxillary tuberosity, coronoid process, mandibular and maxillary tori, and zygomatic body. Extraoral harvesting sites include anterior and posterior iliac crest, costochondral area, tibial, fibular and ulnar bone graft. The surgical techniques in the different anatomic sites are described in detail and the potential risks for complications are also included in the chapter. The authors conclude that reconstruction of osseous defects in the maxillofacial region is one of the most challenging tasks to the reconstructive surgeon.

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lenging tasks to the reconstructive surgeon. The goals of bony reconstruction of the craniomaxillofacial skeleton are to restore:

1. Morphology and position of the reconstructed bone in relation to its opposing jaw and the other facial bones.
2. Adequate bone height and width.
3. Continuity of the maxilla, mandible, and the other facial bones.
4. Facial contour and support for overlying soft tissue.

Bony reconstruction of the craniomaxillofacial complex can be accomplished by a variety of materials and techniques. While many bone substitutes¹ are available (Fig. 23.1), autogenous bone grafts remain the “gold standard” for reconstruction.^{2,3} Autogenous bone has been demonstrated to be superior to allogeneic bone, xenogeneic bone, bone substitutes, and alloplasts in terms of the form, function, and adaptability.⁴ Moreover, autogenous bone is osteoinductive, osteoconductive, and immunologically safe.⁵



Fig. 23.1 Coral granules being placed into mandibular alveolar ridge after the removal of an ankylosed mandibular second deciduous molar in a growing pediatric patient. Such bone graft substitutes may be useful in alveolar ridge sparing or reconstruction procedures; however, the current gold standard for bony reconstruction in the craniomaxillofacial skeleton is autogenous bone.

Introduction

The need for bone

Reconstruction of osseous defects in the oral and maxillofacial region represents one of the most chal-

Recent developments in bone graft harvesting techniques have reduced patient morbidity and improved treatment outcomes.^{6,7} The majority of bone grafts in the maxillomandibular region are performed to reconstruct alveolar bone in order to provide complete functional rehabilitation with osseointegrated dental implants. Other indications for bone grafting may include, but are not limited to, orthognathic surgery, craniomaxillofacial surgery, restoration of traumatic defects, reconstruction of ablative defects left by tumor surgery, and temporomandibular joint reconstruction.

Several donor sites are available to the surgeon for autogenous bone harvesting.^{8,9} The most commonly used intraoral sites (Fig. 23.2) include the maxillary tuberosity and mandibular symphysis, ramus, and retromolar areas, while the most common extraoral sites are the ilium, costochondral area, calvarium, and tibia. Intraoral bone harvesting can usually be performed under local anesthesia alone or local anesthesia combined with nitrous oxide–oxygen sedation, intravenous sedation or oral premedication, as necessary. In contrast, extraoral harvest sites usually require general anesthesia and hospitalization of the patient. A thorough knowledge of the characteristics and limitations of intraoral and extraoral bone harvest sites is important for surgeons, but also for general dentists and other specialists, in order to provide accurate information to their patients and appropriately refer them for reconstructive surgery. The purpose of this chapter is to discuss the indications and techniques for autogenous bone harvest sites for oral and maxillofacial reconstruction as well as to point out the advantages and disadvantages of these various sites.

Choosing a harvest site based upon anticipated volumetric and structural needs

Human bones can be categorized as endochondral and intramembranous bones. Facial and calvarial

bones are of intramembranous origin while almost all other bones used for bone graft harvest (ilium, costochondral tissue, and tibia) are of endochondral embryonic origin. During intramembranous ossification, mesenchymal cells differentiate into osteoid-producing osteoblasts. Endochondral bone originates from a cartilage matrix which slowly calcifies to bone. There is both experimental and clinical evidence which suggests that intramembranous bone grafts undergo less rapid resorption than grafts of endochondral origin (ilium, costochondral tissue, and tibia).¹⁰ It has been proposed that intramembranous bone grafts revascularize more rapidly, which enhances early healing and allows for a more predictable maintenance of volume, than endochondral bone grafts.^{11–13} This phenomenon may be explained by the similar embryonic origin of both the donor and recipient site bone in the oral and maxillofacial region. However, at present, there is little definitive evidence in humans to demonstrate that this difference truly exists.

Bony reconstruction begins with assessing the bone to be reconstructed.³ The defect location, size, and relationship to adjacent structures are the main factors to consider in planning an esthetic and functional reconstruction.^{6,7,14} Thus the surgeon should first determine the quality and quantity of bone needed for reconstruction of the maxillofacial defect in order to choose the most appropriate donor site (Fig. 23.3). Clinical examination, and panoramic, occlusal, and periapical radiographs yield the basic information which will allow for the evaluation of bone volume and for the identification of any pathological lesion in the area. Additional imaging such as panoramic tomograms or computed tomography may also be used.

If a particulated graft is chosen then the surgeon must remember that the graft will serve as a nidus for osteogenesis but it will not have any structural integrity. If a cortical graft is chosen then it will have a slower resorption and integration into the recipient bed.

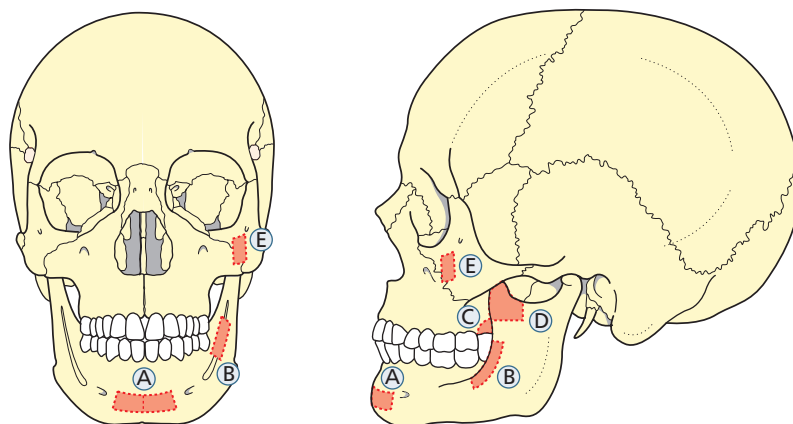


Fig. 23.2 Anterior and lateral views of skull diagrams showing the possible sites for autogenous intraoral bone graft harvesting. Site A: menton or chin mandibular harvesting site; Site B: mandibular ramus harvesting site; Site C: maxillary tuberosity harvesting site; Site D: mandibular coronoid process harvesting site; Site E: zygomatic harvesting site.

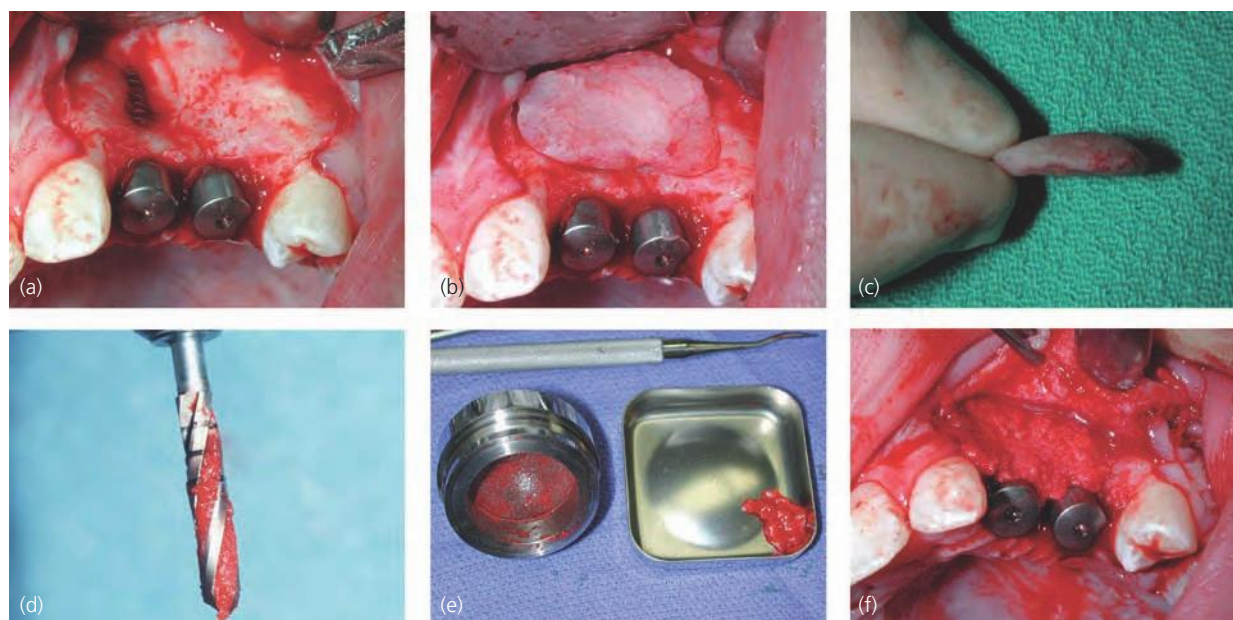


Fig. 23.3 (a) Defect in anterior maxilla left by the removal of a large odontogenic keratocyst. Note labial deficiency of bone with dehiscence revealing the exposed labial threads of the implant fixture placed at the left maxillary canine site. (b) A template is fashioned from bone wax to give the surgeon the dimensions of the required bone graft to satisfy the requirements of the area to be reconstructed. (c) Cross-sectional view of the bone wax template of the deficient alveolar ridge. (d) Autogenous bone graft material harvested from the flutes of the spiral drill used to prepare the implant sites. (e) Autogenous bone graft material harvested solely from the implant preparation sites using both the flutes of the spiral implant site preparation drills and a specially designed suction trap. (f) The particulate autogenous bone graft deposited over the dehiscence of bone in the area of the maxillary left canine to cover the exposed implant threads and to replace the deficient bone.

Cortical or cancellous graft?

Bone harvested from intraoral donor sites is mainly cortical in nature. In the symphyseal area, variable amounts of cancellous bone can also be harvested, but the volume may be limited.¹⁵ Cortical bone blocks from this region can be rigidly fixed in place using osteosynthesis screws or can be easily particulated with a bone mill. Rigidly fixed onlay grafts avoid the potential migration which can occur with particulated grafts. One problem with onlay block grafts is that they are prone to mucosal dehiscence if tension-free soft tissue closure is not achieved. In most cases, loss of the graft is due to wound dehiscence or infection during healing.¹⁶ Stable fixation of the graft along with small perforations of the cortical bone at the recipient site appear to improve graft union. This is most likely the result of an increase in the availability of osteogenic cells and the improved revascularization of the graft.^{17,18}

Particulated mandibular cortical bone grafts can be used as a paste in sinus lifts,¹⁹ in filling alveolar defects, in onlay grafting,²⁰ and in alveolar clefts.²¹ Particulated grafts from the symphysis to the maxillary sinus seem to maintain their volume well and are able to form new bone in 6 months.¹⁹ Bone chips collected during implant bed preparation or bone graft harvesting with a bone suction trap can be used, for example, to cover exposed implant threads or as an additional grafting material with another intraoral donor site.²²

Due to resorption of an onlay block graft, a slight overcorrection or overgrafting of the defect is generally recommended. Cancellous bone grafts resorb faster than cortical grafts. Therefore more overgrafting is needed if mainly cancellous bone is used. Cancellous bone grafts are remodeled faster than cortical grafts and this is probably due to the greater numbers of living cells found in cancellous bone. Also, revascularization is faster as blood vessels have difficulties in penetrating cortical grafts. The suggested healing time for bone grafts is usually 4–6 months, as extended healing periods without functional loading may enhance more bone resorption.^{23,24} An algorithm for graft harvesting is presented in Fig. 23.4.

Intraoral harvesting sites

The most significant advantages of the intraoral harvesting sites (Fig. 23.2) are that the harvesting can be done under local anesthesia as an outpatient procedure, the local donor sites have convenient surgical access, and the ischemic time of the bone graft is short. In addition, since both the donor and recipient sites are intraoral, there is no morbidity from a second surgical site such as a visible extraoral scar.^{10,21} However, the major disadvantage of intraoral harvesting is the limited amount of bone available and, as such, the small quantity may be insufficient for moderate to large defects.⁸ In addition, the harvested

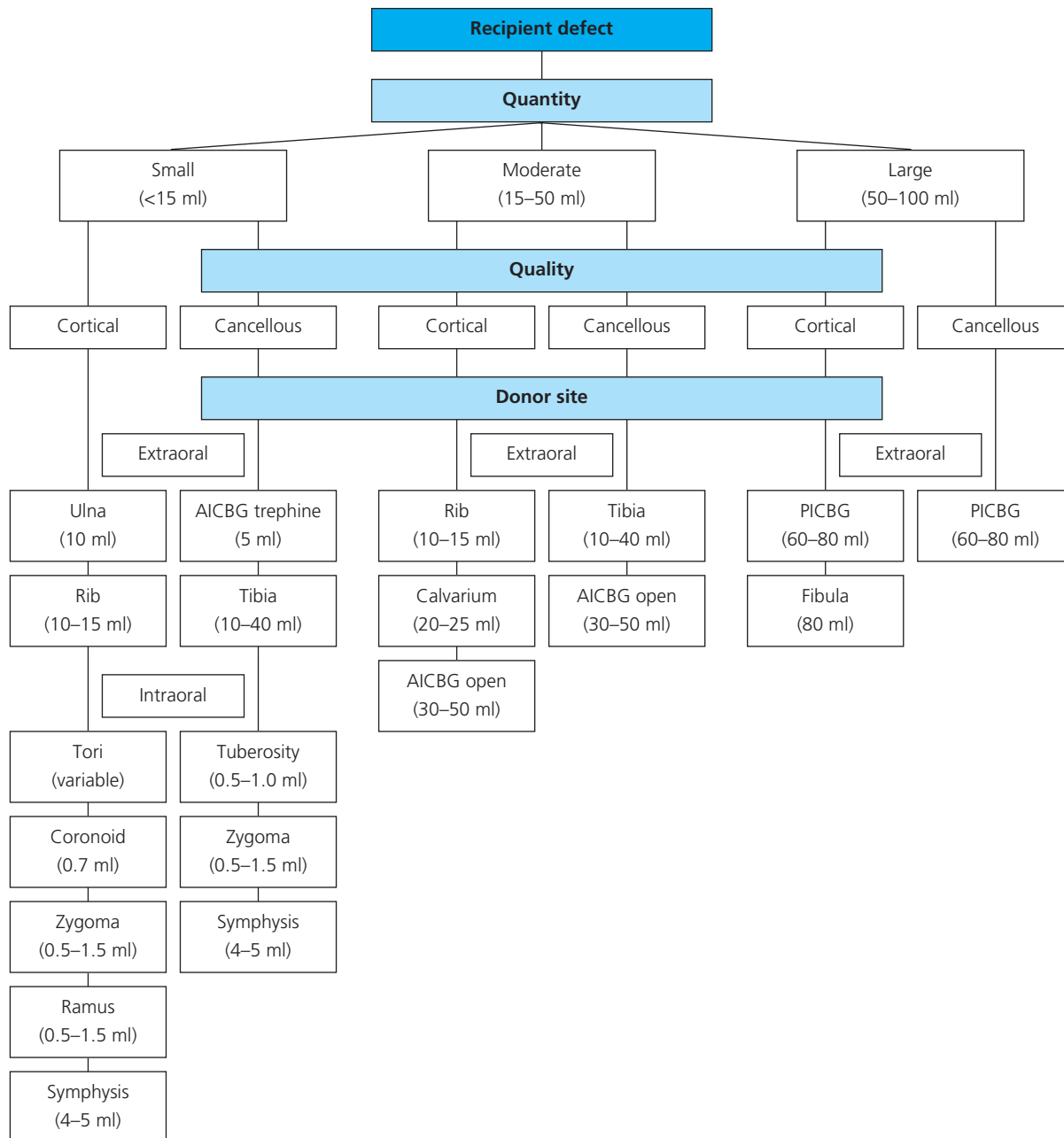


Fig. 23.4 An algorithm to help determine the most appropriate harvesting site for the reconstruction of a particular defect of bone in the craniomaxillofacial skeleton. First the quantity of bone required is considered. Then the quality of bone needed is determined, usually by structural requirements or the resistance to resorption. Finally a site is chosen which is determined by the quantitative and qualitative requirements of the defect to be reconstructed. This is balanced with the concept of the minimization of donor site morbidity, which is always kept in mind. (AICBG = anterior iliac crest bone graft; PICBG = posterior iliac crest bone graft.)

bone is almost completely cortical. Possible complications of intraoral harvesting include endodontic problems, neurosensory disturbances, infections, and wound dehiscence.²⁴ The various intraoral donor sites are discussed below.

Mandibular symphysis

Bone quality and quantity

Mandibular symphyseal bone has been used successfully in secondary alveolar cleft bone grafting,^{10,21} maxillary sinus grafting,¹⁹ grafting alveolar defects

before placement of dental implants,^{16,20} reconstruction of the orbital floor,²⁵ and with Le Fort I osteotomy.²⁶ The anterior mandible is the intraoral donor site producing the largest amount of bone (Fig. 23.5). The average volume of bone harvest available from the symphysis is in the range of 1.7–4.7 ml and the maximum average block size is approximately 21 × 10 × 7 mm.^{15,24} This amount of bone would facilitate a 7 mm increase in ridge width over a 21 mm mesio-distal distance. Particulating the symphysis graft with rongeurs or a bone mill will usually allow for a unilateral sinus lift procedure. Some cancellous bone can also be harvested from the symphysis but the

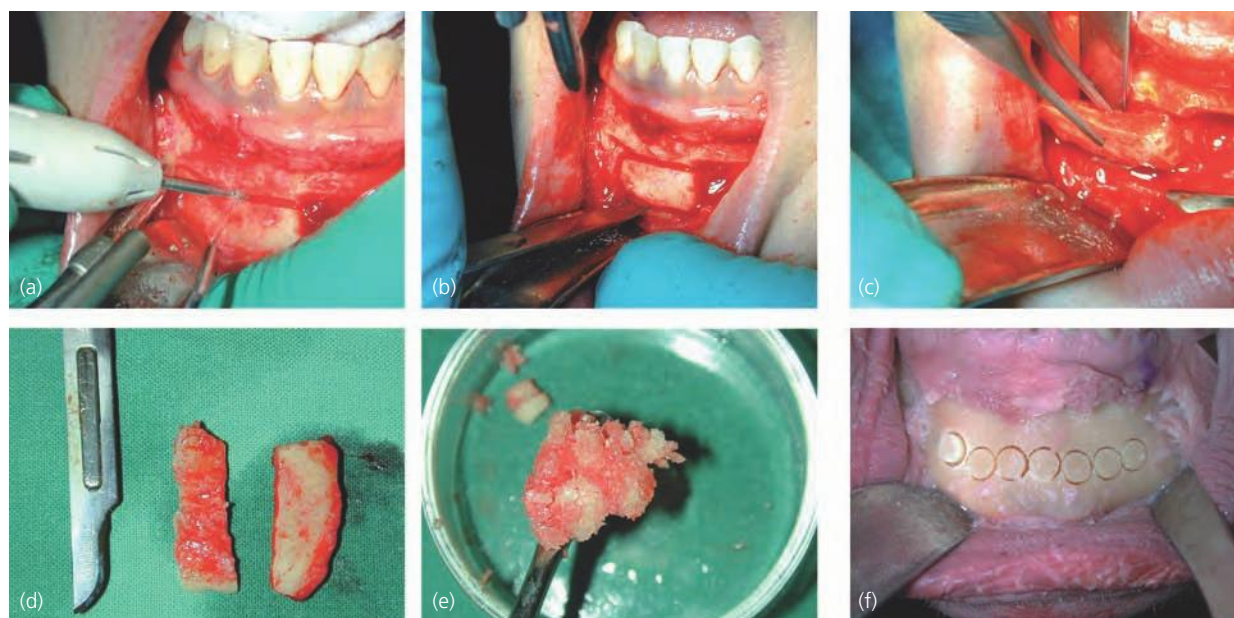


Fig. 23.5 (a) Horizontal osteotomy of anterior cortical layer in a chin graft harvesting procedure. (b) Once the superior, inferior, and lateral osteotomies of the anterior surface of the anterior mandible are completed an osteotome is used to remove the anterior cortex of the mandible. (c) The anterior cortical plate of the chin is being removed. (d) Two harvested cortical plates. The specimen on the left illustrates the nature of the inner aspect of the graft which contains cancellous bone. (e) More cancellous bone can be harvested from the deeper parts of the bony wound once the outer cortical plate of the mandible is removed. (f) A cadaveric specimen showing an alternative method of bone graft harvesting from the chin. A trephine can be used to harvest circular pieces of cortical bone. Once the circular pieces of cortical bone are removed this technique also gives access to the cancellous bone found deep to the outer cortical plate of the mandible. The volume of available cancellous bone in this area can be quite variable and unpredictable.

quantity is highly variable and at times only small amounts can be collected. If cancellous bone is preferred for a reconstruction, an alternative donor site should be considered.¹⁵

Technique

The operation can be performed under local anesthesia. Bilateral inferior alveolar and mental nerve blocks are given. Infiltration with vasoconstrictor-containing local anesthetic in the symphyseal soft tissue reduces bleeding during the operation.

After allowing adequate time for vasoconstrictor effects to take place, the incision is initiated in a layered approach through the labial mucosa and is continued through the mentalis muscles and periosteum. Exposure of the symphyseal bone is undertaken using periosteal elevators and the mental nerves are visualized bilaterally. The roots of the incisors and canines should be localized and bone cuts should be made at least 5 mm inferior to the root apices. The roots of canines can impede the operation and limit the size of graft. Similarly, the surgeon should stay at least 5 mm away from the inferior border of the symphysis and the mental foramina. Bone cuts can be made with a bur or reciprocating saw under copious saline irrigation. When the desired bone cuts have been completed, thin straight or curved osteotomes are then used to deliver the graft. The midline symphysis is usually left intact, and monocortical grafts can be harvested from the right and left sides simultaneously as necessary. It is possible to harvest a bicortical bone block

with this technique but it is not recommended because this may cause postoperative complications including significant pain, swelling, bleeding, endodontic problems, fracture of the mandible, sleep apnea, and loss of chin contour. As such, it is preferable to harvest a monocortical block and use curettes to scoop any necessary cancellous bone. Alternatively a trephine can be used to obtain the graft.

Hemostasis can be achieved using resorbable hemostatic agents or fibrin glue. Long-lasting local anesthetic, e.g. bupivacaine, can be applied to the area to achieve longer analgesia. The wound closure is done in two layers with a resorbable suture for the muscle layer and fast-resorbing suture for the mucosa. Flexible skin tape can be used on the chin for 3–5 days to reduce swelling and prevent wound dehiscence. Temporary chin paresthesia has been documented in at least 43% of patients.²⁷

Mandibular ramus and retromolar area

Bone quality and quantity

The lateral side of the mandibular ramus and retromolar areas are sources of mainly cortical bone suitable for onlay grafting.²⁸ The mandibular retromolar area is a relatively easy site for cortical bone harvesting (Fig. 23.6). Bone can be harvested from the buccal side of the mandible in the second and third molar area and distal to the molars.^{24,28,29} Mandibular ramus and retromolar areas are associated with fewer com-

plications than the symphyseal site.²⁴ The bone block from this area can be as large as 35 mm in length, 10 mm in height, and 4 mm in thickness and the average volume is 0.9 ml,²⁴ but the average size is usually about 16 mm in length, 9 mm in height, and 4.5 mm in thickness, and the volume varies between 0.5 and 1.5 ml.⁸ The block size depends upon the individual anatomy of the mandible and if larger blocks are harvested, the complications increase in incidence and severity.

Technique

The operation can be performed under local anesthesia. Inferior alveolar and buccal nerve blocks are given. After allowing adequate time for vasoconstrictor effects to take place, the incision can be placed in the gingival margin or in the buccal vestibule. A full-thickness mucoperiosteal flap is developed along the lateral aspect of the mandible, exposing the retromolar area and anterior lateral ramus. The size of the bone block needed is first measured and marked on the bone with a bur or a marking pen. The inferior alveolar nerve is near the buccal cortex,³⁰ and sawing or drilling should be performed with caution so as not to damage the neurovascular bundle. The osteotomy can be started anterior to the coronoid process at a point where adequate bone thickness is available. The osteotomy then continues along the anterior border of the ramus medial to the external oblique ridge. The anterior cut is placed in the mandibular body, in the molar region. The posterior vertical cut is made in the lateral aspect of ramus perpendicular to the external oblique osteotomy. The inferior osteotomy connecting the posterior and anterior vertical cuts is made with a bur in a straight handpiece (shallow cut to create a line of fracture). The bone block is released using a thin curved or spatula osteotome. Curettes

are used for cancellous bone harvesting. Sharp bony edges are smoothed with burs or bone files. Trephine burs for implant explantation can also be used to harvest bone cores. The retromolar area is also suitable for harvesting bone chips with a specially designed suction trap (CSMT, Mississauga, Ontario, Canada).^{8,22}

Bleeding is controlled and the wound is closed with a running resorbable suture for a sulcus incision or with interdental interrupted sutures if there was a gingival sulcular incision. Although rare, morbidity from this procedure may include mandible fracture, lingual or inferior alveolar nerve sensory disturbances, bleeding, and wound dehiscence.³¹

Maxillary tuberosity

Bone quality and quantity

Bone harvested from the maxillary tuberosity can be used to fill local alveolar defects before dental implantation.³² The volume of bone in the posterior maxilla is rather limited and the bone is mostly cancellous. It is possible to harvest small amounts of bone from the edentulous maxillary tuberosity, but if the second or third molars exist, this procedure is difficult (Fig. 23.7). This procedure is useful if additional bone is required to "extend" bone volumes in conjunction with other intraoral grafts. For example this may occur with maxillary sinus floor augmentation where it is quite simple to extend the incision and harvest more bone from the tuberosity area with drilling and a bone trap or by rongeurs.

Technique

The operation can be performed under local anesthesia. Local anesthetic with vasoconstrictor is infiltrated

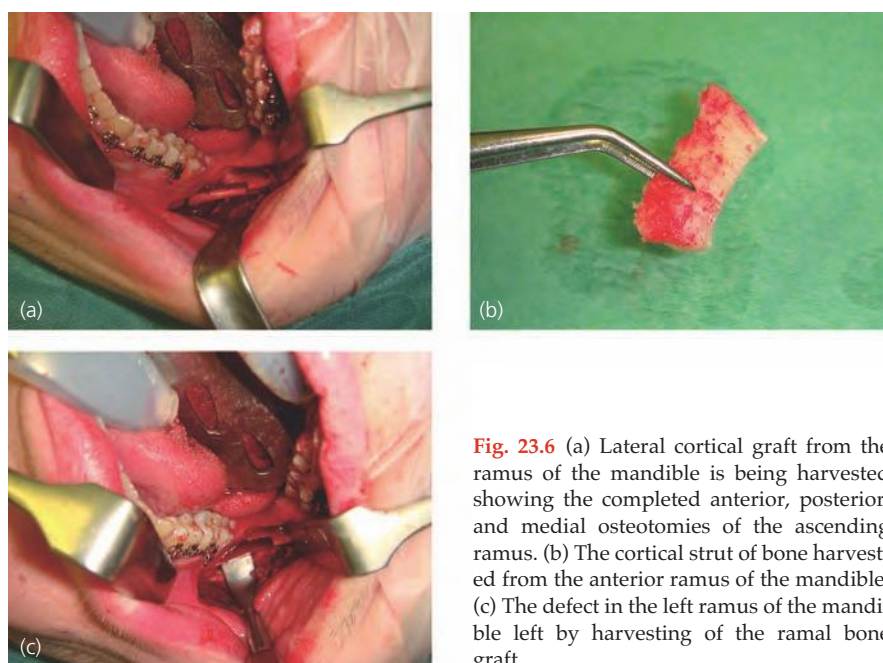


Fig. 23.6 (a) Lateral cortical graft from the ramus of the mandible is being harvested showing the completed anterior, posterior, and medial osteotomies of the ascending ramus. (b) The cortical strut of bone harvested from the anterior ramus of the mandible. (c) The defect in the left ramus of the mandible left by harvesting of the ramal bone graft.



Fig. 23.7 Cadavaric specimen showing a lateral view of the maxillary tuberosity.

to the buccal and palatal sides of the maxillary tuberosity. After allowing adequate time for vasoconstrictor effects to take place, a midcrestal incision is made and the posterior maxilla is exposed. The borders of the maxillary sinus should be localized and bone can be harvested posterior to the sinus with rongeurs, burs or chisels. During harvesting, special attention should be paid not to perforate the maxillary sinus. Bleeding is controlled and the wound closed with resorbable sutures.

Coronoid process

Bone quality and quantity

Bone from the coronoid process has been used for orbital floor reconstruction after a blow out fracture,³³ for nasal augmentation,³⁴ and for paranasal augmentation in conjunction with orthognathic surgery.³⁵ A maximum of 20.9×21.8 mm of coronoid bone is obtainable but it is very thin with a thickness ranging from 4.6–7 mm,³⁵ contains only cortical bone and is not optimal for grafting in implant surgery (Fig. 23.2).

Technique

The operation can be performed under local anesthesia but removal of the coronoid process can be a difficult operation and usually requires general anesthesia. Local infiltration and inferior alveolar and buccal nerve blocks are given. After allowing adequate time for vasoconstrictor effects to take place, an intraoral incision is made in the buccal vestibule. A full-thickness mucoperiosteal flap is developed along the lateral aspect of the mandible, exposing the retromolar area, anterior lateral ramus, and anterior margin of the coronoid process. Total stripping of the attachment of the temporal muscle can be performed with a periosteal elevator or electrocautery. After exposing the entire coronoid process, it may be osteotomized using a fissure bur or reciprocating saw from the sigmoid notch to the anterior ramus. It is then removed

with the use of a Kelly or Kocher clamp. Sharp bony edges are smoothed with burs or bone files. Bleeding is controlled and the wound is closed with a running resorbable suture for a sulcus incision.

Mandibular and maxillary tori

Bone quality and quantity

Mandibular and palatal tori can be used, if present, for alveolar augmentation with mainly cortical bone.

Technique

Considerable amounts of bone can be harvested from tori with a suction trap. If a torus is removed as one block, it can be particulated with a bone mill (Cytagenix, Montréal, Québec, Canada) (Fig. 23.8) or used as a block graft. Small amounts of bone are sometimes needed, for example to cover exposed implant threads. In this case it is possible to harvest bone from nearby edentulous areas with burs and a suction trap.

Zygomatic bone

Bone quality and quantity

Zygomatic bone can also be used as a donor site for alveolar bone reconstruction.^{36–39} It is possible to



Fig. 23.8 (a) Bone mill in its fully assembled state used for mincing cortical grafts into a more particulated workable form. (b) The inner aspect of the bone mill.

harvest about 0.5–1.5 ml of bone from the zygomatic area. The complications and morbidity with this technique have been very mild and the procedure can easily be done under local anesthesia. A bone graft from the zygomatic eminence and arch area has also been used to graft the maxillary step osteotomy and interdental osteotomy gaps during segmental Le Fort I osteotomies.⁴⁰

Technique

The operation can be performed under local anesthesia. Local anesthetic with vasoconstrictor is infiltrated to the buccal vestibule. After allowing adequate time for vasoconstrictor effects to take place, zygomatic bone is exposed through a vestibular incision and bone can be harvested by a trephine bur or round burs and a suction trap (CSMT, Mississauga, Canada) (Fig. 23.9).²² Bleeding is controlled and the wound is closed in layers with resorbable sutures.

Extraoral harvesting sites

Extraoral harvesting sites are recommended when the need for bone graft material is greater than the volume of bone that the intraoral harvesting sites can safely provide.⁹

Ilium

The ilium is the preferred donor site for bone grafting to the maxilla and mandible. It contains the greatest absolute cancellous bone volume and has the highest cancellous-to-cortical bone ratio. These grafts may be harvested from either the anterior or posterior areas of the ilium (Fig. 23.10). Donor site complications may include seroma, hematoma, nerve and vessel injuries, gait disturbances, iliac fractures, peritoneal perforation, infection, sacroiliac instability, and pain. Paresthesia or anesthesia of the skin following iliac crest bone graft harvesting has been reported in 8–38% of patients.^{41–46} Use of drains at the anterior or

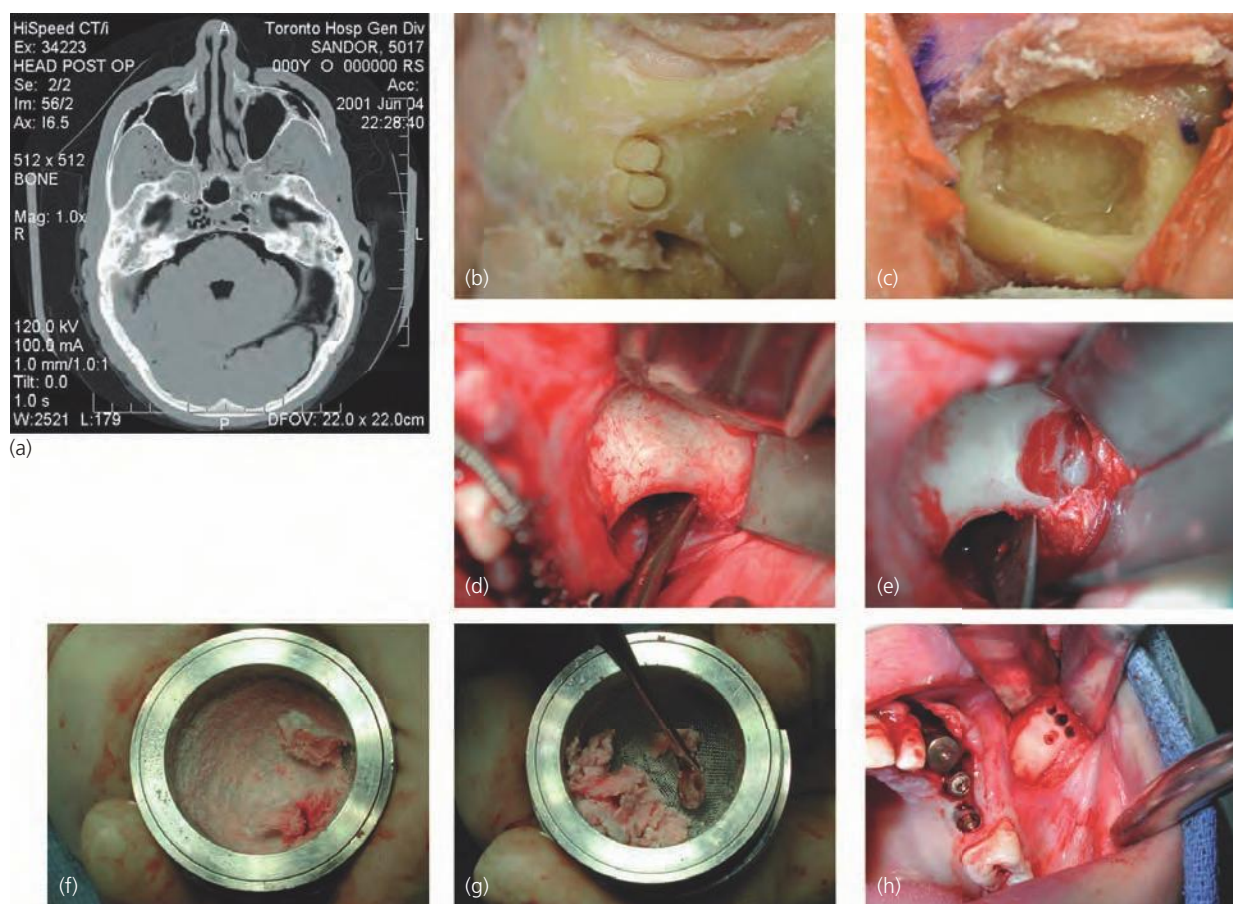


Fig. 23.9 (a) Axial view computed tomography (CT) scan showing defects in the anterior of the zygoma from where zygomatic bone can be harvested. (b) Cadaveric specimen showing trephine method of zygomatic bone graft harvesting. (c) Cadaveric specimen showing defect left by harvesting the zygomatic site with a large round bur to produce a particulated bone graft. (d) Clinical exposure of the zygomatic harvesting site using an intraoral incision in a living patient. (e) Harvesting site after preparation with a large round bur. (f) Particulate bone graft harvested from the zygomatic site collected by a specially designed bone trap. (g) Bone graft being gathered in preparation for transfer to the intraoral recipient site. (h) Zygomatic site seen just lateral and superior to implant placement site where bone graft material is needed. The zygomatic sites can be harvested by using spiral implant preparation burs and bone graft then removed by a suction trap. Note the proximity of the graft harvest site to the reconstructive site or recipient bed. Harvesting from nearby intraoral sites can help to reduce morbidity at the bone graft harvest site.

posterior donor sites are not mandatory and there is no difference in wound healing.⁴⁷

Anterior iliac crest – open

Bone quality and quantity

The anterior iliac crest has been used by oral and maxillofacial surgeons as a donor site for bone harvesting for decades.⁴¹ It is a dependable and versatile site (Fig. 23.11) allowing the harvesting of a predictable amount of cortical and cancellous bone for moderate-sized defects.⁴⁸ In some small individuals, however, the volume of bone harvested can be disappointing. In general, the anterior iliac crest should allow harvesting of 30–50 ml of bone.⁴⁹ The patients usually require postoperative hospitalization. The main limitation for the volume is that harvesting from medial and lateral plates should be avoided. If this is

done, the patient will have a high rate of morbidity after the operation. The risks and morbidity associated with this site include short- and long-term gait disturbances and lateral femoral cutaneous nerve paresthesia that can be quite troublesome to patients.^{41,50}

Technique

Anterior iliac crest bone graft harvest begins with site selection. Bone graft harvesting from the ipsilateral or contralateral side from the recipient site is mainly determined by the patient position relative to the anesthetic set-up and operating room team. After the nasoendotracheal tube has been passed to secure the patient's airway, the patient is prepared and draped in the customary manner for a two-team approach to this procedure. A separate field is used to avoid cross-contamination of donor and recipient sites, and as such the contralateral side is usually preferred.



Fig. 23.10 (a) Radiographic view of an anterior iliac crest harvest site. (b) Superior view of the bony skeleton of the anterior iliac crest.

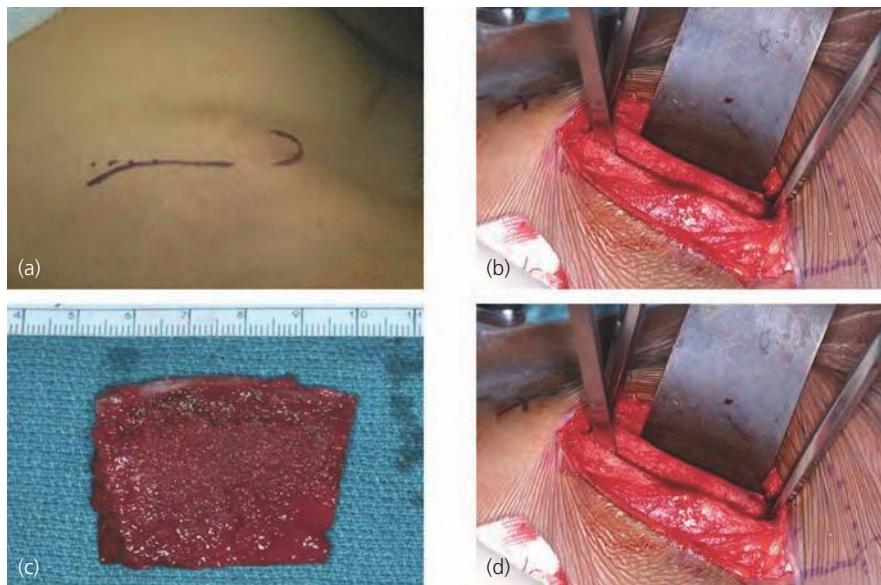


Fig. 23.11 (a) Outline of the skin surface anatomy over the anterior iliac crest. The backwards facing “C” represents the location of the anterior superior iliac spine, or the origin of the sartorius muscle. The harvesting technique should avoid this landmark as the force of the pull of the sartorius muscle could fracture away its insertion if the harvesting is too close. Harvesting too close to the anterior superior iliac spine may also result in bothersome paresthesia in the distribution of the lateral femoral cutaneous nerve on the anterior thigh. This is a complication that may be avoided by harvesting more superiorly. (b) The anterior aspect of the iliac crest is exposed. Osteotomies have been performed superiorly and inferiorly staying at least 1 cm above at the anterior superior iliac spine. The medial aspect of the iliac crest is preferred by the authors. (c) The harvested corticocancellous block of bone from the anterior iliac crest. (d) The defect left by harvesting the inner aspect of the iliac crest.

The planned incision is first marked on the left or right anterior iliac crest following the appropriate anatomic landmarks: the anterosuperior iliac spine and anterosuperior iliac tubercle are marked along with the anterior iliac crest. The nerve branches that are most at risk are the lateral cutaneous branch of the subcostal nerve (T12) and the lateral cutaneous branch of the iliohypogastric nerve (L1). The lateral femoral cutaneous nerve is located anterior and medial to the anterosuperior iliac tubercle; careful delineation of landmarks will minimize damage to this nerve. The skin overlying the iliac crest is gently pulled superiorly and medially to allow the incision to rest in a position inferior and lateral to the iliac crest. The resultant scar should be in a position where it is not rubbed or chafed afterwards by a belt or clothing.

The incision is made parallel to the crest of the iliac bone and a 3–4 cm incision is usually adequate to gain access to the iliac bone. A long-acting local anesthetic with vasoconstrictor is infiltrated subcutaneously as well as subperiosteally. After allowing sufficient time for vasoconstriction, and local anesthesia to take effect, an incision is made through skin and subcutaneous tissue with a scalpel. Dissection can then proceed with electrocautery through the fat and down through Camper's and Scarpa's fascia, and on to the periosteum.

Once through the periosteum, the subperiosteal dissection can proceed on to the medial or lateral surfaces of the ilium, depending on the approach used and the need for a mono- or bicortical graft. In the anteromedial approach the subperiosteal dissection continues on to the medial side of the bone, being careful to preserve the tensor fascia lata attachment to

the lateral ilium in order to minimize gait disturbances and pain. A medially placed retractor may be helpful to protect the iliacus muscle and peritoneal contents. In the anterolateral approach, the periosteum is reflected from the lateral ilium for easier access but at a cost of increased incidence of gait disturbance. Following identification of this region, the required size of the graft is outlined, and using saws, osteotomes, or a bur, osteotomies are performed under copious normal saline irrigation. After the block is freed, further underlying cancellous bone may be harvested using a series of bone gouges and curettes. Once sufficient quantity and quality of bone are harvested, all sharp bony edges are trimmed with a bone rasp. Bleeding points are then attended to, and the wound is irrigated with copious amounts of normal saline. The wound is closed in layers: periosteum, fascia, dermis, and skin. During the graft harvest, the recipient site can be prepared simultaneously to save time if two teams are available.

Anterior iliac crest – trephine

Bone quality and quantity

The era of minimally invasive surgery has led to the development of new technologies and surgical approaches that minimize postoperative morbidities. Cores of cancellous bone, with diameters of 4.0 mm and lengths up to 30 mm, can be removed from the iliac crest with a motorized trephine (Fig. 23.12). It has been reported that as many as seven cores can be removed giving 4 ml of bone, which is suitable for moderate-sized defects.⁵¹

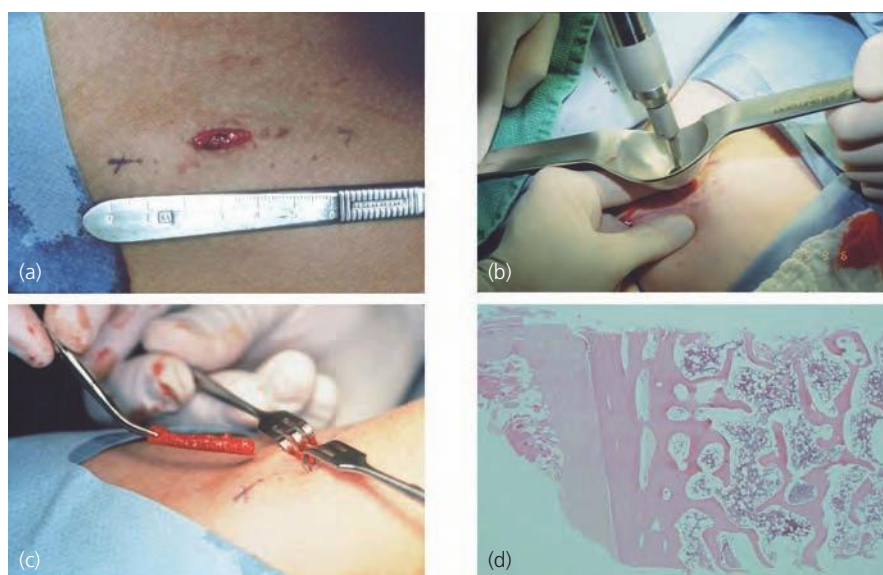


Fig. 23.12 (a) The exposure for an open approach to harvest bone from the anterior iliac crest can leave a scar 5 cm in length. Here the wound for motorized trephine harvesting shows that such harvesting is possible with a much smaller 1.5 cm long “stab” incision with reduced morbidity. (b) The motorized trephine is being guided by a propeller-shaped guide to help remove cores of bone from the anterior iliac crest. (c) A cancellous core of bone is being harvested from the anterior iliac crest. (d) Hematoxylin and eosin stained light photomicrograph of a harvested cancellous core showing all of the elements necessary for osteogenesis within the harvested graft material. There is an abundance of osteogenic and hematopoietic stem cells in the graft and a natural scaffold of cancellous bone.

Technique

The patient is prepared as above with the open approach but here the iliac crest can be approached through a stab incision as small as 1–1.5 cm for a motorized or hand-driven trephine, and stripping of muscle is not necessary.⁵¹ Pain, postoperative morbidity, and complications are less frequent than with an open approach² and the procedure can be performed as an outpatient⁶ or even out of the hospital in a surgical office.^{52,53}

Posterior iliac crest

Bone quality and quantity

If large quantities of bone are required, the posterior iliac crest provides a good source for most of the large maxillofacial defects,³⁸ excluding the cases in which microvascular grafts are needed. Both cortical and cancellous bone can be harvested, even though the majority of gain is cancellous (Fig. 23.13). Greater amounts of bone can be obtained from the



Fig. 23.13 (a) Patient in prone position for harvesting of bone from the posterior iliac crest. (b) A 12 × 3 cm corticocancellous block of bone harvested for major reconstruction as a non-vascularized graft from the posterior iliac crest. (c) Strips of cancellous bone can be further harvested from the posterior iliac crest donor site. (d) Harvesting of the posterior iliac crest can become a routine for the anesthetic staff, surgical staff, and the nursing staff. The patient is first anesthetized and intubated while in the supine position on a stretcher. (e) The patient is then “flipped” on to the OR table in the prone position with the face down. The anesthesia staff ensure that the airway is patent and that all tubes and monitoring devices are located satisfactorily before the patient is further prepared and draped. (f) The curvilinear incision is outlined once the harvest site is prepared and draped. Note the locations of the superior cluneal nerves (SCN) and the middle cluneal nerves (MCN) drawn on to the patient. (cont’d)

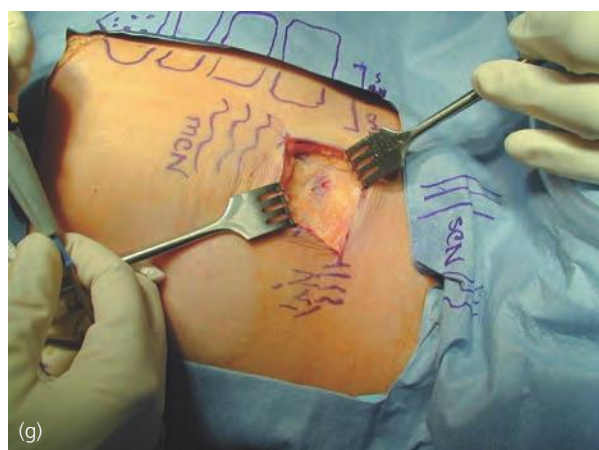


Fig. 23.13 (cont'd) (g) The incision is carried down through the superficial fascia and the posterior iliac crest is exposed. (h) Osteotomies can be made with a saw as shown or by using osteotomes and chisels. (i) The corticocancellous fragments are prepared for removal from the posterior iliac crest.

posterior ilium and the maximum obtainable bone approaches 90–100ml. Patients seem to tolerate harvesting from the posterior iliac crest better than from the anterior iliac crest site with less postoperative morbidity.^{41-45,54} This may be due to the fact that they convalesce lying with the full weight of their body on their wound, thereby causing less hematoma formation.⁵⁵

Technique

After the nasotracheal tube has been passed to secure the patient's airway, the patient is carefully turned from the supine position to the prone position in a slightly reversed-flexed position. All necessary areas of the face, pelvis, and thorax are supported to allow proper ventilation and the pressure areas are carefully padded to avoid any injury during the harvest of the bone from the posterior iliac crest.

After harvesting, the patient must be turned back to the supine position for the intraoral procedure. The anesthetist and surgeon must make sure that this is done safely. Because of repositioning, the bone graft harvesting and recipient site reconstruction cannot be done simultaneously which will lengthen the operation time.

The planned incision is first marked on the left or right posterior iliac crest following the appropriate anatomic landmarks: the spinous processes of the vertebra and the posterosuperior iliac spine and crest to avoid the cluneal nerves. A 5cm curvilinear incision is usually adequate to gain access to the iliac

bone. A long-acting local anesthetic with vasoconstrictor is infiltrated subcutaneously as well as subperiosteally. After allowing sufficient time for vasoconstriction, and local anesthesia to take effect, an incision is made through skin and subcutaneous tissue with a #10 scalpel blade. Sensory nerves at risk are the superior and middle cluneal nerves (L1 to S3). Using electrocautery dissection through the subcutaneous tissues, the posterosuperior crest is identified and the lumbodorsal fascia divided between the abdominal and gluteal muscles. A subperiosteal dissection proceeds, and the tissue is reflected laterally in the appropriate region to harvest the quantity of bone required for the procedure. Care is used to avoid the sacroiliac ligaments.

Following identification of this region, the required size of the graft is outlined, and using saws, osteotomes, or a bur, osteotomies are performed under copious normal saline irrigation. After the block is freed, further underlying cancellous bone may be harvested using a series of bone gouges and curettes. Once sufficient quantity and quality of bone have been harvested, all sharp bony edges are trimmed with a bone rasp. Bleeding points are then attended to, and the wound is then flushed with copious amounts of normal saline. The wound is then closed in layers: periosteum, lumbodorsal, dermis, and skin. At this point, the patient can then be carefully repositioned to the supine position on the operating table for the completion of the reconstructive part of the procedure.

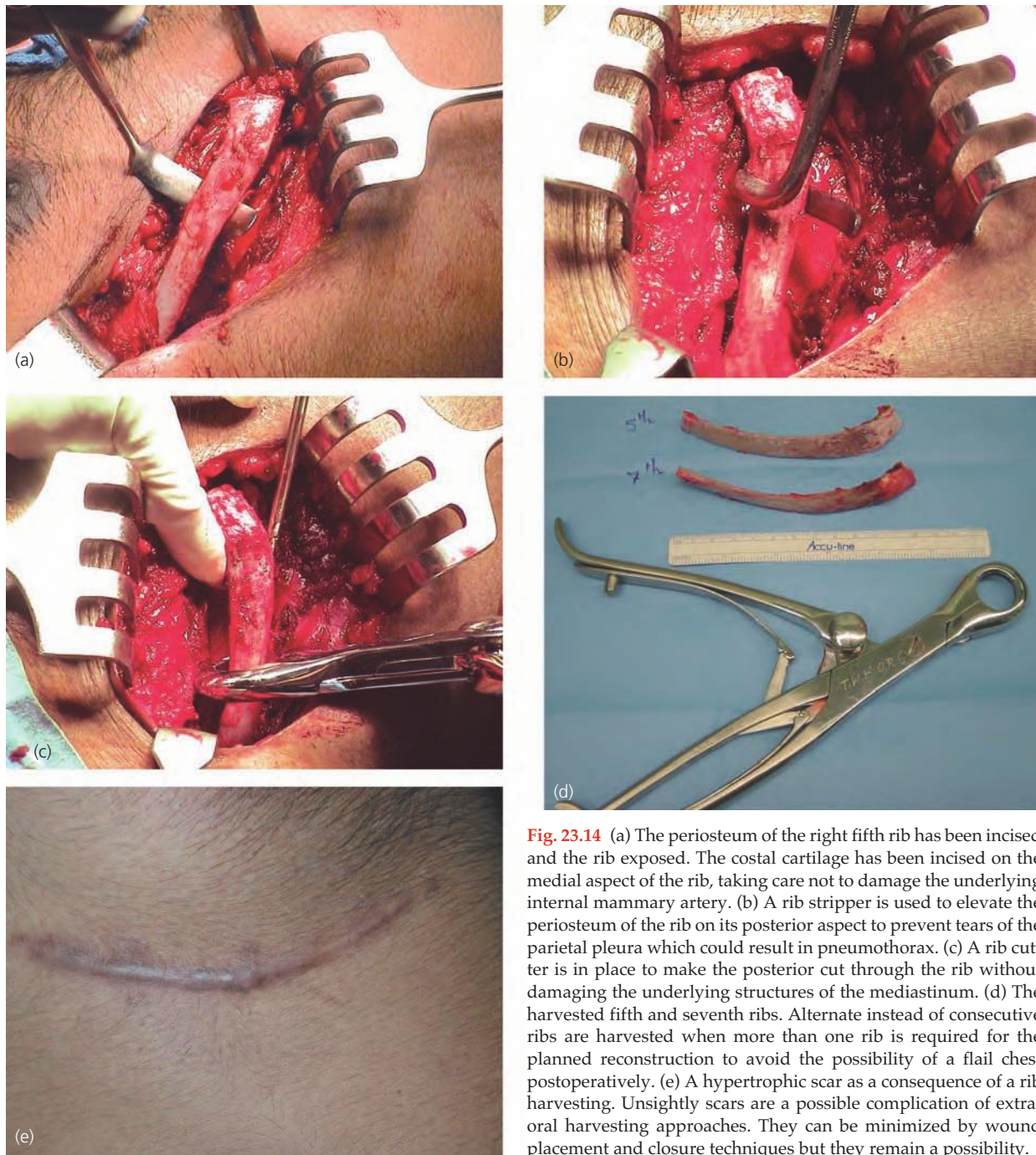


Fig. 23.14 (a) The periosteum of the right fifth rib has been incised and the rib exposed. The costal cartilage has been incised on the medial aspect of the rib, taking care not to damage the underlying internal mammary artery. (b) A rib stripper is used to elevate the periosteum of the rib on its posterior aspect to prevent tears of the parietal pleura which could result in pneumothorax. (c) A rib cutter is in place to make the posterior cut through the rib without damaging the underlying structures of the mediastinum. (d) The harvested fifth and seventh ribs. Alternate instead of consecutive ribs are harvested when more than one rib is required for the planned reconstruction to avoid the possibility of a flail chest postoperatively. (e) A hypertrophic scar as a consequence of a rib harvesting. Unsightly scars are a possible complication of extra-oral harvesting approaches. They can be minimized by wound placement and closure techniques but they remain a possibility.

Costochondral grafts

Bone quality and quantity

Costochondral grafts are beneficial since both cartilaginous and bony tissues may be harvested (Fig. 23.14). The chondral part may be useful as an articular surface for temporomandibular joint reconstruction and provides a potential growth center for growing patients.⁵⁶ The bone, however, is limited by the size, curvature, and strength of the rib. In young adults the cartilage may calcify early and be disappointing when costal cartilage is sought for temporomandibular joint reconstruction. For reconstructing the temporomandibular joint, the contralateral rib

usually has the more favorable contour and reconstruction can be performed with a graft from the fourth to eighth rib. Ribs from either side can be harvested, but most surgeons prefer to use the right side over the left side because of the position of the heart. Indications for costochondral grafts include reconstruction of the TMJ in congenital malformations such as in hemifacial microsomia, and in acquired cases such as TMJ osteoarthritis or condylar fractures.⁵⁷ The disadvantage of using rib as the donor is a visible scar on the chest. In women, the incision can be hidden in the submammary crease. In addition to pain, some postoperative pulmonary complications such as atelectasis and, rarely, pneumothorax have been reported.

Technique

After the nasoendotracheal tube has been passed to secure the patient's airway, the patient may be prepared and draped for a two-team approach for this procedure. A curvilinear incision is used that corresponds to the submammary crease and will usually overlie the sixth rib. The skin is incised sharply and sharp dissection with periosteal elevators follows in order to enter the plane overlying the ribs from the midaxillary line to the costochondral junction. A longitudinal incision is made over the bony portion of the rib, and a careful subperiosteal dissection is performed with periosteal elevators circumferentially around the rib. Great care is used during the dissection of the inferior and deep aspects of the rib, to avoid the neurovascular bundle, and on the medial aspect of the rib, to avoid lacerating the internal mammary artery. Either saws or rib cutters may be used to harvest the costochondral graft with a variable amount of cartilage attached to the end. The cut edge of the remaining rib in the donor site is rounded with bone rasps to avoid sharp edges. Normal saline is placed in the donor site, and the patient's lungs are inflated to assess for a possible pneumothorax which

may require pleural repair or a chest tube. The wound is closed in layers and a long-acting local anesthetic with epinephrine may be administered to the donor site.

Calvarium

Bone quality and quantity

The cranium is composed of two layers of thick cortical bone, the outer and inner diploë. The cancellous bone in between varies in thickness. Calvarial grafts from the areas of the parietal bone are the most useful and the thickness of the bone should be at least 6.0 mm to consider *in situ* harvesting. Cortical membranous grafts consisting of 4 × 4 cm pieces of tissue 5 mm thick can be harvested,⁵⁸ but there is little cancellous bone available. This graft site is ideal for reconstruction of zygomatic or orbital defects when using cortical grafts. Particulate grafts may also be harvested with the use of a suction trap (Fig. 23.15).^{22,59} Postoperative morbidity is surprisingly minimal as long as a drain is placed to prevent hematoma or seroma formation. Intracranial perforations are rare.

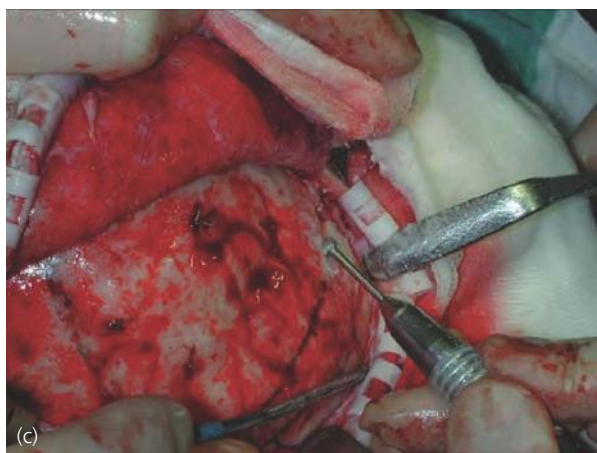
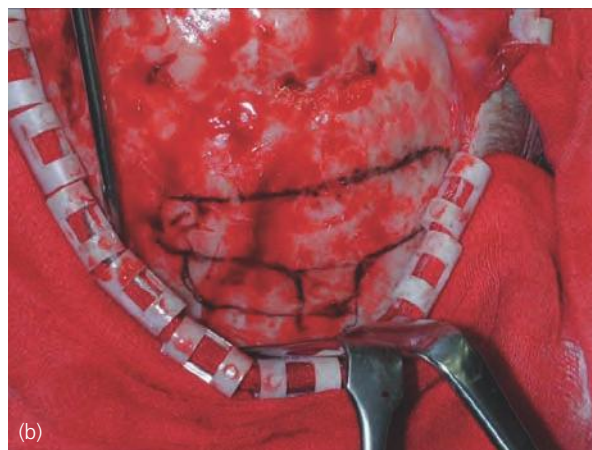


Fig. 23.15 (a) A coronal flap is used to expose the bones of the calvarium. (b) A pencil outline is made on the skull for a planned outer table calvarial harvesting to reconstruct a deficient zygomatico-maxillary complex. (c) A large round bur outlines the harvested bone. The outline of the harvest with a groove made by the large round bur facilitates the harvest by allowing space for the oblique placement of an osteotome to eventually help lift up the zygoma-shaped bone graft as a single piece. (d) The osteotome harvesting the outer calvarial plate or diploë. (*cont'd*)

Technique

Following the induction of general anesthesia, the patient is prepared and draped for a craniomaxillofacial procedure. A dilute, long-acting local anesthetic with vasoconstrictor is infiltrated along the pre-planned incision line. The planned scalp incision can be coronal or hemi-coronal and is made above the hairline parallel to the direction of the shafts of the hair follicles in order to minimize the risk of alopecia. After allowing adequate time for the vasoconstrictor to take its effect, a sharp dissection with a scalpel down to the subgaleal fascia is performed. Clips are

then applied to the edge of the wound to control bleeding from the scalp. The dissection of the scalp flap should proceed in the subgaleal plane, and then the pericranium of the calvaria should be incised sharply. The planned area of the graft harvest is marked out with a bur, staying at least 2 cm from the sagittal suture to avoid overlying the sagittal sinus. A large round bur is used to make initial cuts through the outer cortex of the calvaria. A suction trap is continuously used to harvest particulate bone even when a cortical graft is harvested. The cuts are beveled to allow insertion of a curved osteotome in a plane parallel to the outer surface and at the diploic level.

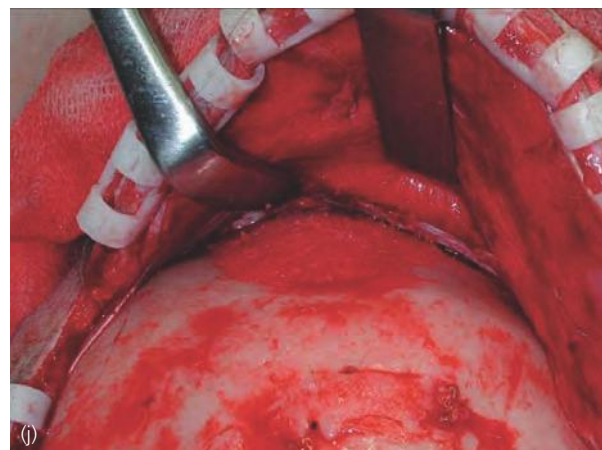
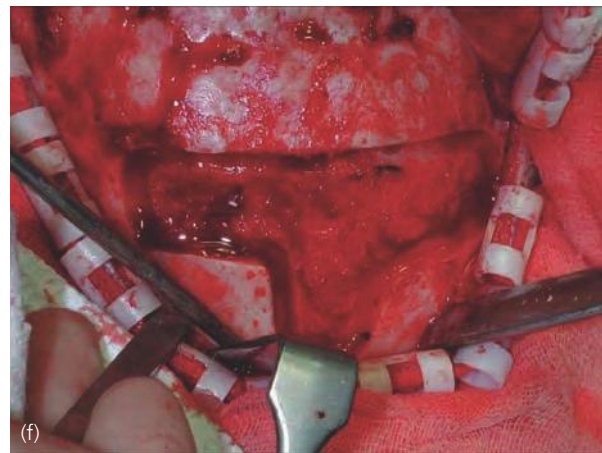


Fig. 23.15 (cont'd) (e) The mobilized graft is then removed in one piece. (f) The resulting defect of the outer calvarial table. (g) A specially designed bone trap with valves to prevent both desiccation of the graft and minimize bone graft contamination (CSMT, Mississauga, Ontario, Canada). (h) The "blown-apart" components of the bone trap. (i) The suction trap was used while the large round bur was drilling through the outer calvarial diploë of the skull. The particulate bone graft which the trap collected would otherwise have gone to waste. This material can be used in further reconstruction of the the defect left by harvesting the outer table of the skull. (j) The particulate graft from the outer layer of the skull was used to reconstruct a frontal bone defect.

Once the graft has been harvested the donor bed is checked to assure integrity of the inner cortex, and a piece of gelatin foam is placed over the site. The periosteum is reapproximated and the scalp closed in layers, with the galea being reapproximated. The skin can be closed with either staples or sutures.

Proximal tibia

Bone quality and quantity

The proximal tibia is another important source of autogenous bone. A great deal of cancellous bone can be harvested even though the quality of bone in elderly patients is less predictable due to fatty deposition in the bone marrow (Fig. 23.16). Cancellous bone from tibia can be compressed and used in sinus lift-

ing and in alveolar augmentations. Equal amounts of bone are available from either lateral or medial approaches and up to 42ml of cancellous bone may be harvested.⁶⁰⁻⁶² For more extensive volumes of grafts, bilateral graft harvesting may be performed but may result in impaired early ambulation. Morbidity may include temporary gait disturbances, scarring, and fracture of the tibial plateau.^{63,64}

Technique

The proximal tibia has become popular as a bone graft donor site due to low morbidity and the fact that the operation can be performed in an outpatient setting under sedation and local anesthesia. The graft is usually harvested with the patient in the supine position. Harvesting is made close to Gerdy's tubercle,

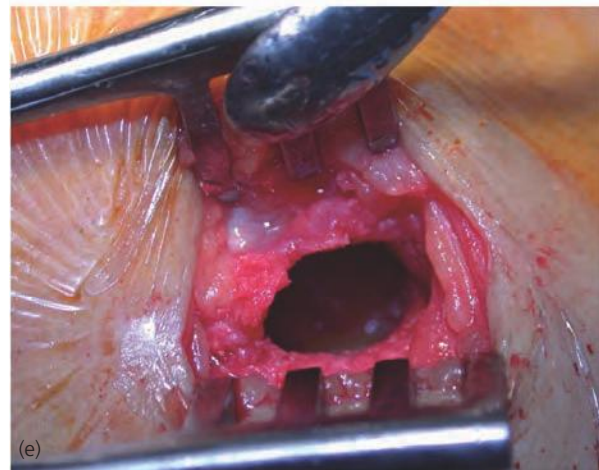
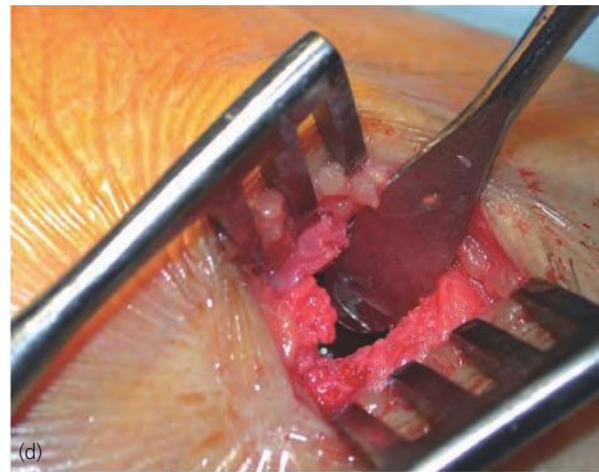


Fig. 23.16 (a) A lateral view of the flexed knee showing the iliotibial band insertion. Gerdy's tubercle is located just inferior to this insertion. F represents the fibular head, immediately in front of the common peroneal nerve which must be avoided during this harvesting technique. T pinpoints the attachment of the ligamentum patellae into the tibial tubercle. (b) The incision below the iliotibial band is carried through the superficial fascia until the periosteum over Gerdy's tubercle is incised. (c) A circular osteotomy is performed through the lateral cortex of the tibia to permit access to the cancellous bone within the tibial metaphysis. (d) Generous quantities of cancellous bone are removed from within the tibia. (e) The empty tibia harvest site and wound just before closure.

which is clinically easy to find, about 4cm inferior and lateral from the knee's patellar bone.^{60,61,65} The planned 3cm longitudinal and slightly angled incision is marked on the skin overlying Gerdy's tubercle and a long-acting local anesthetic with vasoconstrictor is infiltrated subcutaneously as well as subperiosteally. After allowing sufficient time for vasoconstriction, and local anesthesia to take effect, an incision is made through skin and subcutaneous tissue with a scalpel. Sharp suprapariosteal dissection overlying and inferior to Gerdy's tubercle proceeds. This dissection should be medial to the tibialis anterior muscle and lateral to the patellar ligament. A cortical window is made with burs, saws, or osteotomes measuring 1 × 1 cm. A medial approach to the tibia may also be used to avoid the iliotibial tract insertion.^{62,66} In this approach, the landmarks are a vertical line drawn through the patella and tibial tuberosity, and the other perpendicular to the first, through the tibial tuberosity. It is recommended that an oblique skin incision be made centered over a point 15mm superior to the horizontal line and 15mm medial to the vertical line.⁶² Subperiosteal dissection continues along this incision and a bone window is made to harvest the cancellous bone with curettes. Alternatively, a small stab incision and a trephine may be used for smaller amounts of bone. The wound is then closed in layers. After harvesting, a tensor bandage must be applied to the knee and ambulation is encouraged immediately.

Proximal ulna

Bone quality and quantity

The upper extremity of the ulna has several sites of potential bone graft harvesting.⁶⁷ The posterior surface of the ulna just distal to the olecranon is one of them (Fig. 23.17). Cortical strips can be harvested, for nasal reconstructions, and the bone suitable for harvest in the adult is approximately 5 cm long and 1 cm deep and wide.⁶⁸ Cancellous bone volume is relatively unpredictable at this site but the morbidity is rather minimal. A tourniquet can be used to minimize blood loss. Postoperative pain at the donor site is usually much less than that experienced at an iliac crest donor site.

Technique

The operation can be performed in an outpatient setting under sedation and local anesthesia. The olecranon process is the bony protuberance of the proximal ulna palpable dorsally under the elbow skin. It is continuous as the subcutaneous border of the posterior ulnar bone, bounded by the insertion of the triceps and anconeus muscles, and the origin of the flexor carpi ulnaris muscle of the forearm. The non-dominant arm is usually chosen for harvesting the graft and a pneumatic tourniquet is applied to the arm. A

long-acting local anesthetic with vasoconstrictor is infiltrated subcutaneously as well as subperiosteally. After allowing sufficient time for vasoconstriction, and local anesthesia to take effect, a transverse incision is performed with a scalpel. A longitudinal incision is made in the periosteum and a periosteal elevator is used to strip the periosteum and the adjacent muscles. An oscillating saw or fissure bur may be used to cut through the cortex of the proximal ulna and the cortical graft is liberated with a curved osteotome. The cancellous bone can be harvested at the desired thickness with the use of osteotomes and curettes. Bleeding points are then controlled and the periosteum is closed with resorbable sutures. The incision is closed in two layers and a bandage is applied to the elbow after which the tourniquet is removed.

Vascularized fibula

Bone quality and quantity

A vascularized graft is an ideal choice in large defects or when the bone graft recipient bed has questionable vascularity. Patients with decreased vascularity, such as those who have received radiation to the head and neck area, may be at risk for future osteoradionecrosis. Patients with hypovascularity such as those with Raynaud's disease should be treated using a vascularized bone graft. The fibula is one of the most versatile sites for maxillofacial reconstruction requiring a vascularized bone graft (Fig. 23.18).

One major disadvantage of this flap is that the harvested fibular bone graft is tubular in shape and therefore not always the most ideal shape or form for grafting in the maxillofacial area. It has a dense outer cortex and a marrow space which is sparsely occupied by trabeculae when compared to the mandible.⁴⁸ Soft tissue may also be harvested together with the fibula and may be useful if there is a lack of soft tissue coverage for the graft at the recipient bed. The procedure is also very time consuming and the donor site morbidity may be significant: scarring and ankle instability are possible complications.

Technique

After the nasotracheal tube has been passed to secure the patient's airway, the patient is prepared and draped in the customary manner for a two-team approach to this procedure. The patient's hip and the knee are slightly flexed, and a pneumatic tourniquet is placed in the proximal aspect of the leg. A separate field is used to avoid cross-contamination of donor and recipient sites.

A line connecting the lateral malleolus to the fibular head is drawn. The dissection is carried down to the crural fascia that is subsequently incised. A 2–3 mm muscle cuff surrounding the bone is maintained. The dissection continues through the anterior border of the peroneal muscles. The extensor digitorum

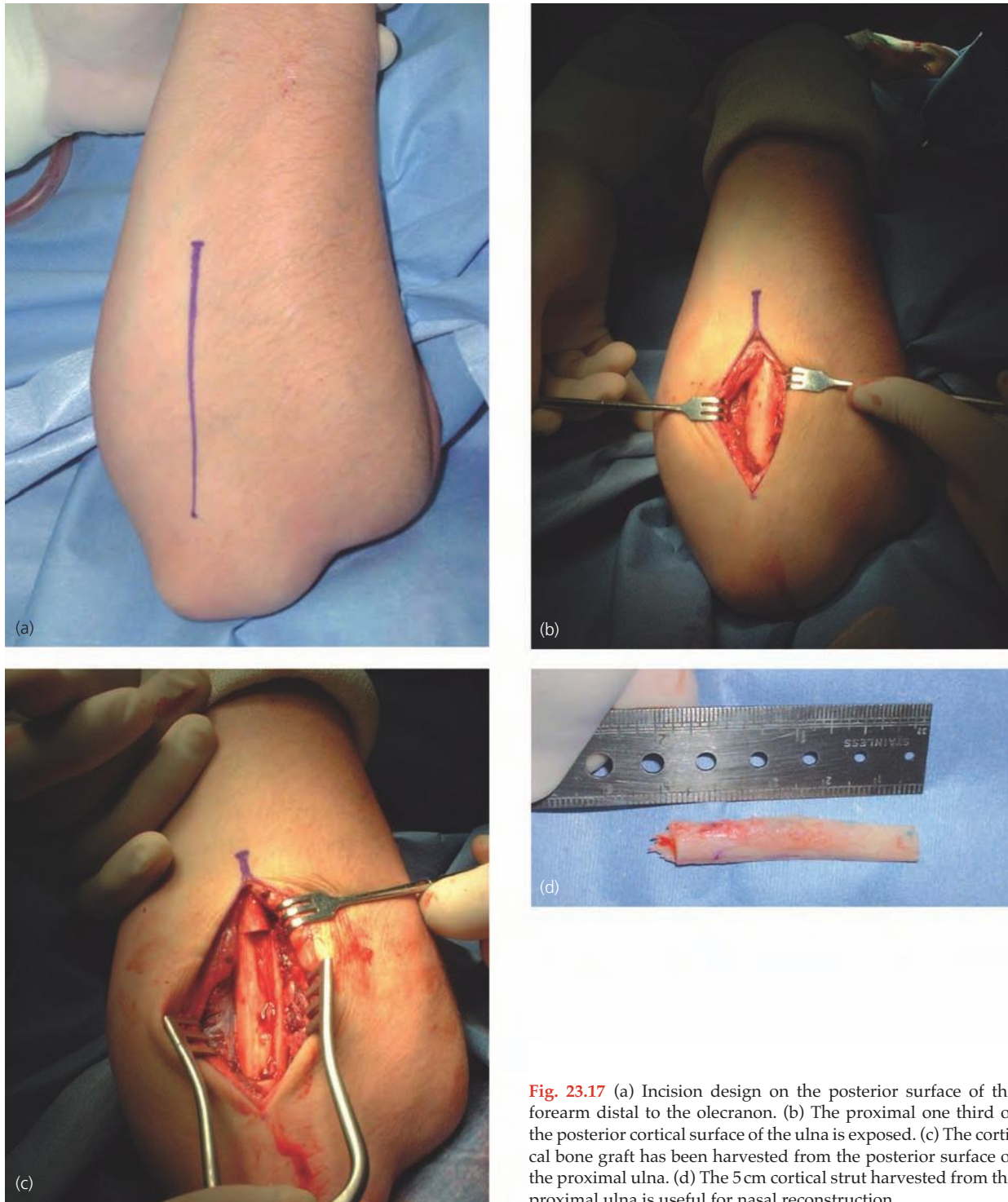


Fig. 23.17 (a) Incision design on the posterior surface of the forearm distal to the olecranon. (b) The proximal one third of the posterior cortical surface of the ulna is exposed. (c) The cortical bone graft has been harvested from the posterior surface of the proximal ulna. (d) The 5 cm cortical strut harvested from the proximal ulna is useful for nasal reconstruction.

longus and the extensor hallucis longus are elevated anteriorly, exposing the interosseous septum between the fibula and the tibia. Next, two horizontal incisions are performed in the proximal and distal aspects of the fibula where the osteotomy is planned. The osteotomies to shape the fibula should be performed while the fibula is still pedicled to the proximal vessels in order to minimize the ischemic time. The vessels in the recipient site are prepared before ligation of the proximal aspect of the peroneal artery. The osteotomies are done in the donor site leg with a saw while a malleable retractor protects the medial aspect.

The distal aspects of the peroneal vessels are ligated and the vascular pedicle is carefully dissected superiorly to identify the branching of the peroneal artery from the posterior tibial artery. The graft is harvested together with the branches of the peroneal artery and its accompanying veins or venae committantes.⁶⁹ The donor site can be closed primarily or managed by the placement of a split-thickness skin graft. At the recipient bed, the graft is secured into position and the vessels are anastomosed to the superior thyroid, lingual, facial or superficial temporal arteries and branches of the internal jugular vein.

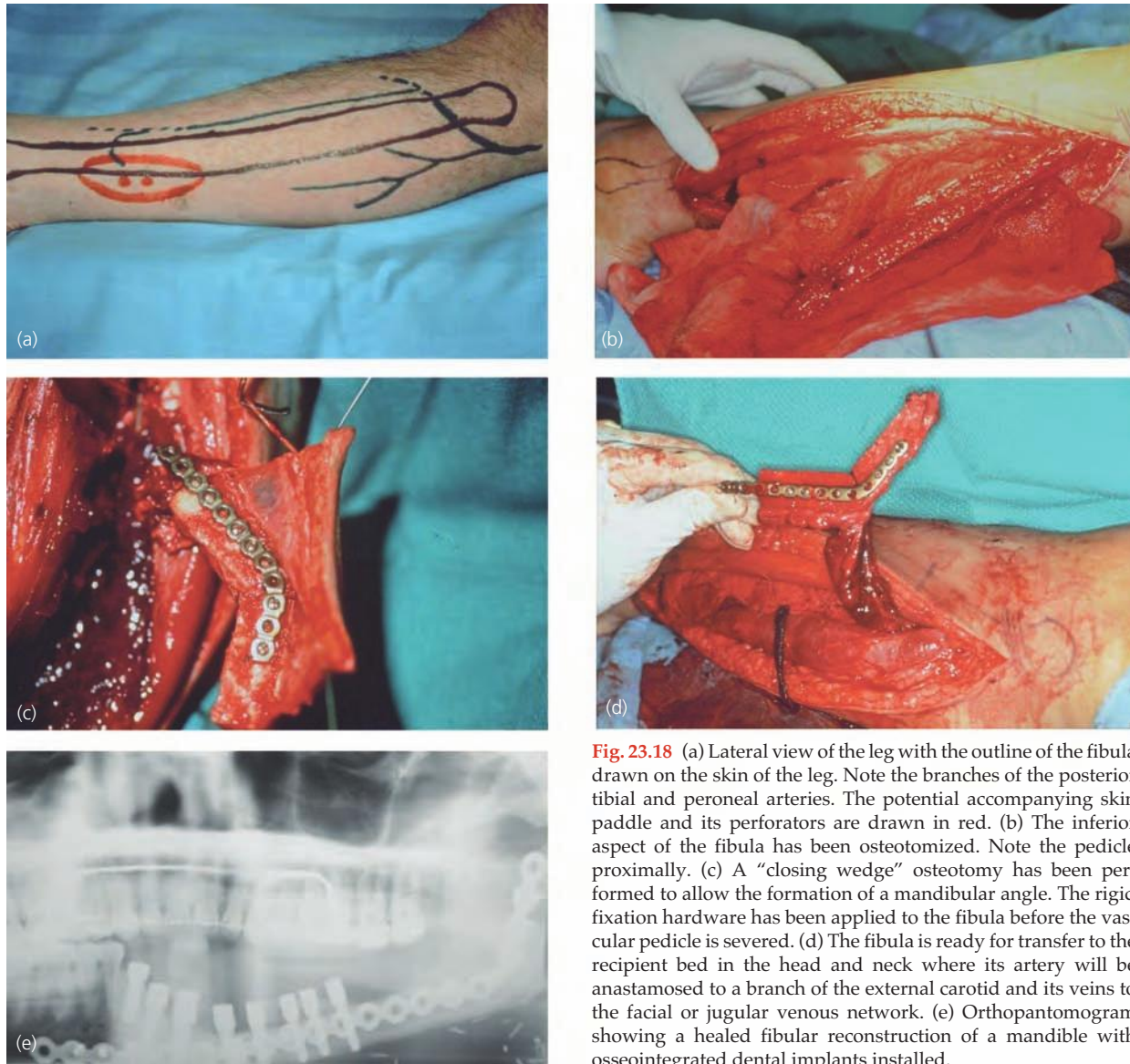


Fig. 23.18 (a) Lateral view of the leg with the outline of the fibula drawn on the skin of the leg. Note the branches of the posterior tibial and peroneal arteries. The potential accompanying skin paddle and its perforators are drawn in red. (b) The inferior aspect of the fibula has been osteotomized. Note the pedicle proximally. (c) A "closing wedge" osteotomy has been performed to allow the formation of a mandibular angle. The rigid fixation hardware has been applied to the fibula before the vascular pedicle is severed. (d) The fibula is ready for transfer to the recipient bed in the head and neck where its artery will be anastomosed to a branch of the external carotid and its veins to the facial or jugular venous network. (e) Orthopantomogram showing a healed fibular reconstruction of a mandible with osseointegrated dental implants installed.

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Chapter 24

Treatment of Bone-deficient Ridges in Implant Rehabilitation

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The chapter contains an overview of bone graft treatment in implant surgical rehabilitation. Aspects of bone graft behavior in the clinical situation, treatment of the bone graft, and discussion about particulated bone or bone block are included in the chapter. Use of the bone graft as an inlay graft or onlay graft is another important issue. Surgical techniques associated with bone grafting are also part of the chapter together with comments on the surgical split crest technique. The use of platelet-rich plasma (PRP) in connection with bone graft is discussed. Finally possible complications associated with different bone-grafting techniques are considered.

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Introduction

Surgical techniques today enable us to reconstruct bone-deficient regions equally well in the maxilla and the mandible. Bone grafting techniques, using autogenous bone from the iliac crest, mandibular symphyseal region, mandibular angle or cranium, can provide material for reconstruction purposes. Onlay grafting is especially indicated for defects, thin ridges and as a complement to inlay grafting. Inlay grafting, in combination with orthognathic surgery, has a predictable outcome.

Background

Functional rehabilitation with dental implants has been in practical use for over 40 years with extremely good results.¹ Early long-term studies over 15 years, in rehabilitation of edentulous mandibles, show success rates of over 90%. Edentulous, resorbed mandibles were the most problematic situations and these were also where fixed implant bridge solutions were most indicated.² The long-term results have been remarkably good, which today means that it is possible to have immediate loading in most edentulous mandibles.¹ The maxillary region is more problematic, especially with regard to the anatomic situation, where the maxillary sinus and nasal cavity occupy large areas. The cortical structure of the bone tissue in the maxilla is not the same as in the mandible, due to factors such as muscle influence on the mandible. The fact that the mandible is the mobile part of the chewing system explains some of the differences between the two jaws. The edentulous maxilla may require much more augmentation than the edentulous mandible, in order to be suitable for implant rehabilitation.

Bone deficiency was considered a contraindication for implant surgery and rehabilitation. In the mandible, a reduced amount of bone could still be enough for implant success because of its cortical bone structure, but in the maxilla, inadequate bone volume was considered to make implant stability and success impossible.

Over time biomaterials of different kinds have been more extensively used with a satisfactory clinical outcome, although the healing time is at least double that for autogenous bone grafts. Other surgical techniques to solve bone-deficient situations, such as zygoma implants, are also available for this kind of patient. There is a spectrum of surgical techniques available today which enable us to solve all kinds of

bone-deficient situations, equally satisfactorily in the maxilla and the mandible.

Aspects of bone graft behavior

Autogenous bone graft material is the gold standard for reconstruction of bony defects in the facial skeleton, such as tumor resections, congenital defects or atrophy of the jaws.^{3,4} Fresh, immediately harvested bone graft is by far the most reliable.⁵⁻⁸ The bone graft may then be replaced by new bone by osteoconduction or by osteoinduction through surviving osteogenic cells.⁹⁻¹³ The microvascular system in the bone graft may also make minor anastomosis with the surrounding vital tissue and revascularize the graft material.¹⁴⁻¹⁶ The predictability of the graft volume has not always been certain. Postoperative resorption may radically reduce the volume by up to 50–70% of the graft when it is in a passive state. Several factors may be of importance in maintaining as much bone graft volume as possible. Recent literature reviews indicate that the embryonic origin is important for the graft survival. Membranous bone should be superior to endochondral bone with less resorption over time.¹⁷⁻¹⁹ The revascularization of the graft is extremely important for starting new bone formation. Another important factor in preserving bone graft volume is rigid fixation of the graft, probably because this creates an appositional phase earlier, and thereby promotes improved osteoconduction.²⁰⁻³⁰

The recipient bed for the bone graft may also be of importance for graft survival and volumetric changes. The influence of the bone graft recipient site on the survival of onlay bone grafts has been studied in several publications. The degree of contact between bone graft and recipient site as well as the orientation of the bone graft may have a significant effect on remodeling of vascularized bone grafts.^{31,32} Cortical preparations of the recipient bone marrow site allow exposure of osteogenic stem cells to the graft and may improve bone healing.^{33,34} Several publications support the theory that perforated bone is superior to unperforated. The perforations may increase initial healing of the bone graft, but there is no evidence that they help to maintain bone graft volume. Osteopromotive membranes have been used to improve bone graft survival and stability; these allow free diffusion of tissue fluid and prevent cellular ingrowth from adjacent connective tissue.

Membranes in combination with hyperbaric oxygen, growth hormones or bone morphogenetic proteins have been shown to increase the rate of bone formation. Bicortical bone grafts retain bone volume better than unicortical grafts. A major improvement in graft incorporation was observed following exposure of underlying recipient marrow through cortical perforations.^{35,36} The effects of marrow exposure by cortical perforations of the recipient bed resulted in a migration of the recipient bone marrow into the graft

as well as decreased loss of graft volume. A combination of cortical perforations of a bicortical graft and the host bed gave increased graft stability and corticalization of the marrow.³⁷ When the graft was covered by an osteopromotive polytetrafluoroethylene (e-PTFE) membrane, improved graft integration and greater size preservation were observed even after membrane removal.³⁵

However, full volumetric maintenance of the graft was not accomplished in any of these studies. A combined treatment of high concentration of recombinant human bone morphogenetic protein-2 (rh BMP-2) and membrane lead to a rapid and complete graft integration and pronounced bone formation resulting in maintained or even enhanced graft size. However, the use of high dose rh BMP-2 without a membrane resulted in loss of graft size, extensive bone resorption, and only small amounts of new bone formation.^{38,39}

Block or particulated bone

Bone block grafts are structurally stable and initially resistant to resorption. The autogenous bone block will be almost totally necrotic after the grafting procedure and needs revascularization to survive. Incorporation of the graft is initiated by an inflammatory reaction during the first weeks, which then turns into a granulation phase, followed by osteoclastic activity. The new bone in and around the bone block is formed by creeping substitution and development of lamellar bone. This process proceeds gradually and has not reached normal physiological strength until 1 year. Normal biological properties are not present for at least 2 years, and part of the block will still contain residual necrotic original bone. Mechanical strength will be reduced during the first year.

Particulated bone graft can be both cortical and cancellous. Particulation can be done with a bone mill or the bone can be cut into pieces with bone scissors. Cancellous particulated bone has a larger surface area exposed to the surrounding tissue and vascular ingrowth may proceed faster than in bone block grafts. Compared to cortical graft it regains biological properties faster but it is also more exposed to resorption activities. The cancellous bone graft gradually increases in mechanical strength and it takes 1–2 years to reach normal mechanical strength, much like the bone block graft.

Onlay grafting

Different methods for reconstruction of the atrophic maxilla can be used, depending on the actual situation. An atrophic alveolar ridge, with sufficient height but which is too thin, may be suitable for onlay grafting, where bone graft from either the hip or the mandible can be used for augmentation. Onlay grafting aims to widen the crest and make it possible for

implant insertion. It is very important not to reduce the vertical height of the crest, since vertical augmentation may be very difficult to achieve.

The first attempts to treat severely resorbed maxillae were made in the 1980s with large horseshoe-shaped bone blocks grafted to the maxillary ridge (Fig. 24.1). Technical difficulties with the technique made it unpredictable and there was a relatively high morbidity rate.⁴⁰⁻⁴⁸

The buccal onlay grafting technique is useful in the posterior mandible as well as in the maxillary alveolar process. To achieve as good a result as possible it is advisable to make small perforations in the buccal cortical bone before attaching the graft with plate screws. If there is a discrepancy between the graft and the host bed it is also advisable to put bone graft material in the space to optimize the contact between bone graft and crest. The bone graft may be harvested from the mandibular angle, chin region, iliac crest or cranial sites. The most important issues in this procedure are to achieve rigid fixation of the bone graft and to close the covering soft tissue in such a way as to prevent microorganisms from the oral cavity entering the grafted area. The flap covering the bone graft should have a relaxed position and not be

forced into a position by use of a strong suture. If this is not achieved, the graft may be totally destroyed due to wound dehiscence and compromised vascular blood supply.

A large onlay graft was the initial method of choice for reconstruction of the resorbed edentulous maxilla. The bone graft was obtained from the iliac bone beneath the iliac crest not involving the arch of the crest (Fig. 24.1a, b).⁴⁹ The horseshoe-shaped bone graft was modeled to fit on to the exposed upper jaw crest (Fig. 24.1b). The remaining, exposed crest of the upper jaw was ground to fit the graft better and the cortex was perforated with a small round drill to form a better vascular bed for the grafted bone. After modeling of the bone graft to fit on to the crest, implants could be inserted into the graft and retained in the residual crestal bone in a one-stage procedure; alternatively the graft could be attached to the crest with plate screws in a two-stage procedure, thus allowing the graft to become more vascularized before implant insertion. The choice of a one- or two-stage concept depends, of course, on the amount of available bone volume. More residual bone allows for a better and safer implant stability and is therefore more suited for one-stage procedures. Large onlay

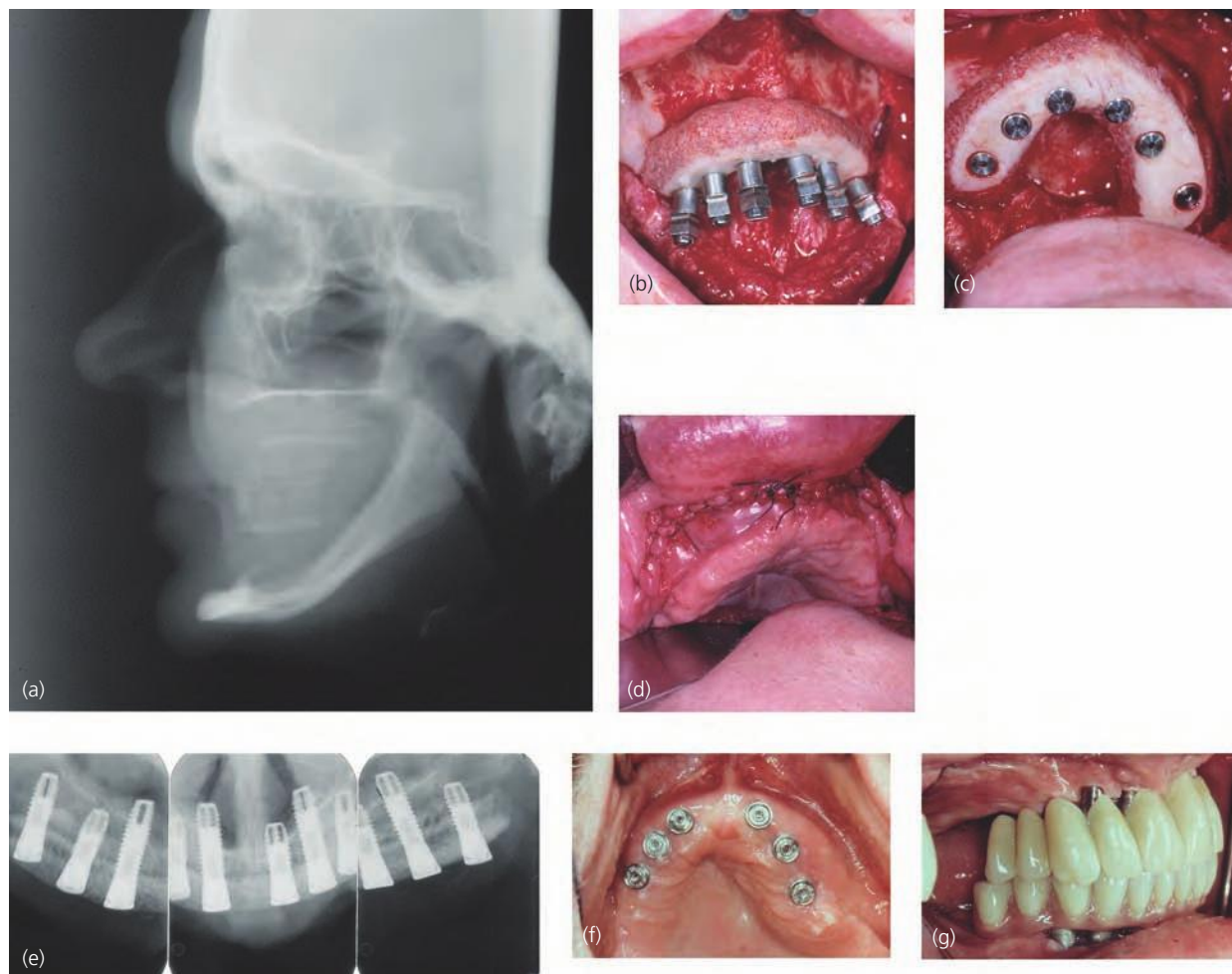


Fig. 24.1 (a) Lateral radiograph of edentulous maxilla and mandible. (b) Onlay grafting with block graft from the iliac bone and simultaneous implant insertion. (c) Implant and bone graft in place. (d) Soft tissue closure. (e) Radiograph of implant placement. (f) Abutment connection 6 months later. (g) Implant-supported bridge construction.

block grafts have been shown to achieve a good result in long-term follow-up with one-stage implant surgery and also with a two-stage surgical technique.^{43,44,46,48,50}

Long-term clinical and radiographic follow-up studies have shown very good success and survival rates with onlay grafts with only minor drawbacks.

With all autogenous bone grafts, healing follows the same principles, according to Wolf's law. The bone graft has to be functionally stimulated in order not to be resorbed. So a bone graft without functional stimulation by chewing through implants will resorb more or less completely. This functional stimulation is very well achieved by the implants. One should always allow for some remodeling resorption where excess bone graft material will disappear.

Bone graft will always adapt to functional load and stimulation. Thus in the horseshoe-shaped iliac bone grafts with excess volume outside the implants,

functional remodeling, especially in a buccal–lingual direction was clearly evident. In patients with a retrognathic position at the upper jaw this resorption of 2–3 mm was negative since the retrognathic look of the maxilla was still noticeable even after implant surgery.⁴⁵

A technical problem of onlay grafting for small and larger surgical procedures is that the additional bone material has to be covered by a vital, vascularized flap, which must not be stretched or damaged. Careful closure of the incision without communication with the oral cavity is another important factor for a successful result. Big bone blocks for treatment of edentulous maxillae are especially vulnerable since there is so much foreign material to be incorporated within the tissue. Therefore it is of utmost importance to have as thick a flap as possible, with good vascularization to form a tension-free cover over the bone graft material (Fig. 24.1d).

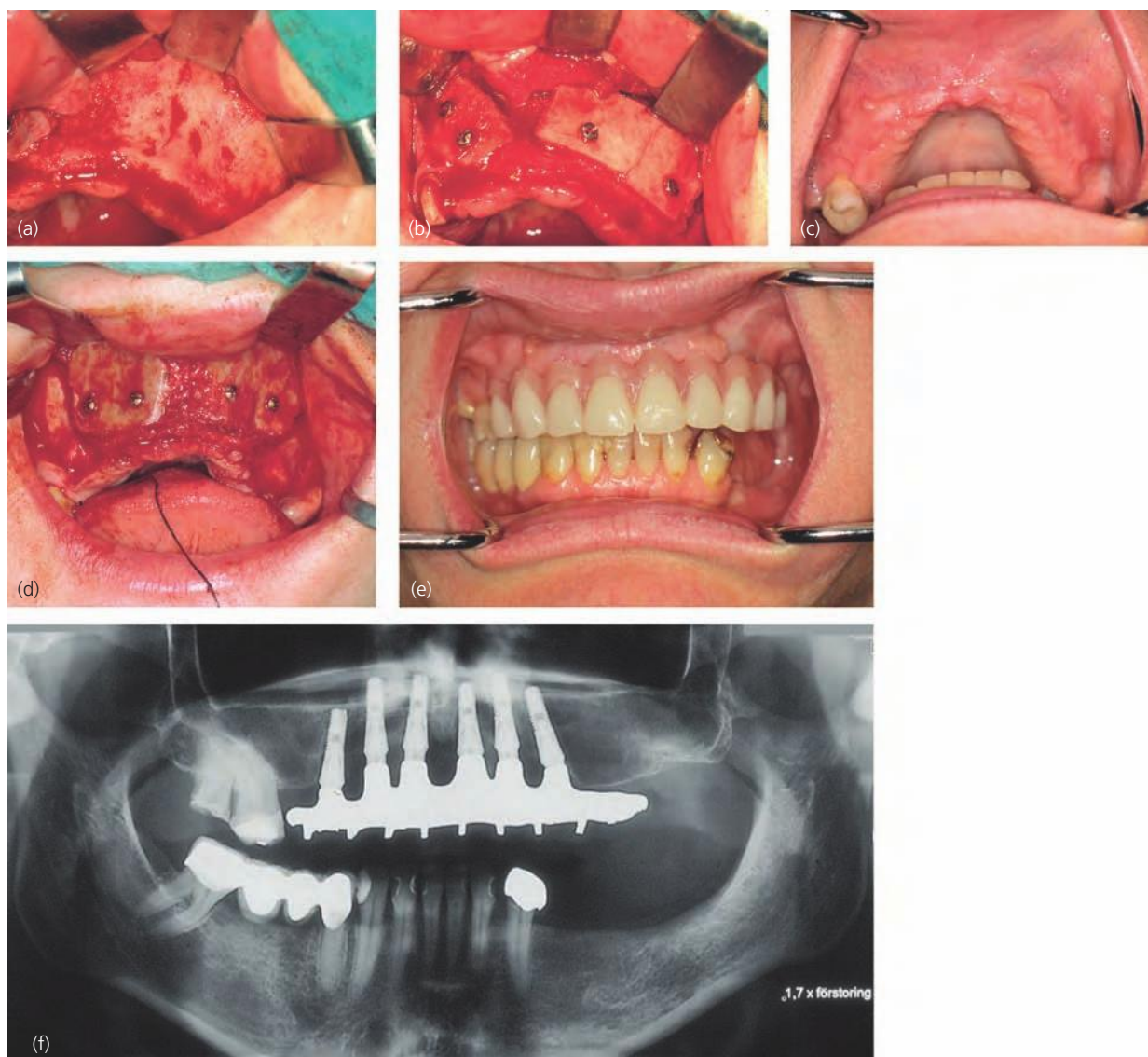


Fig. 24.2 (a) Clinical view of thin but high alveolar crest in the anterior region of the maxilla. (b) Onlay grafts positioned to increase the width of the alveolar crest. (c) Soft tissue healing. (d) Bone graft healing. (e) Implant-supported bridge in place. (f) Panoramic radiograph after bridge construction.



Fig. 24.3 (a) Single tooth loss in the anterior maxilla. Thin alveolar crest. (b) Onlay graft in place to increase bone volume of the crest. (c) Implant insertion in the reconstructed crest. (d) Implant-supported crown in place.

The onlay bone graft can be attached to the residual crest with either the implants (one-stage procedure) or in a two-stage procedure with either plate screws or osteosutures. The choice of which technique to use depends on the clinical situation (Figs 24.2, 24.3).

Inlay grafting

In cases with an atrophic maxilla, bone grafting can be performed using orthognathic surgical procedures. Maxillary osteotomy Le Fort I with down-fracturing of the maxilla is an excellent method for augmenting the alveolar process. After mobilization of the maxilla it can be manually repositioned with the finger or carefully with Rowe and Killey's disimpaction forceps. The sinus membrane and the sinus recesses are carefully extirpated. The nasal mucosa should be intact or repaired to avoid contamination from the mouth or from the nose. Bone graft material is modeled to fit into the sinus recesses and nasal floor and secured with osteosutures. Normally five wire loops are sufficient to attach the bone graft of combined cortical plate and cancellous bone to the sinus recess and nasal floor. The maxilla is then held in its planned position with plates and screws. Anterior repositioning up to 10–15 mm is possible in most cases. Inferior repositioning can also be done to adjust the relationship between lip and upper jaw crest.

The Le Fort I technique for bone graft augmentation of the edentulous maxillas was originally introduced by Sailer who simultaneously performed grafting and implant installation.⁵¹ In the present model the bone graft is allowed to heal for 4–5 months before implants are installed. Implants can then be positioned as required for the prosthetic solution (Fig. 24.4). Slightly different approaches can be used for this procedure but the outcome with the treatment is usually very good with success and survival rates of 90–95%.^{52,54–60}

Preparation of the grafting site to optimize the healing process can be done by perforating the cortex of the recipient site with a small round bur. The cortical surface can also be roughened to stimulate bone formation. When pure cortical bone graft is placed against a cortical ridge the healing time should normally be extended 1 or 2 months to avoid the risk of inadequate healing between the two cortical surfaces.

The advantages with this method for treatment of deficient bone volume in the maxillary alveolar process are the predictable result and the possibility of correcting sagittal discrepancies between the jaws to some extent to improve the prosthetic possibilities. The problem with soft tissue closure which may arise in onlay grafting is not a problem in inlay grafting.^{57,58,61,62} The choice between one- or two-stage procedure as well as early loading is up to the surgeon.^{63,64} The author's own experience suggests a

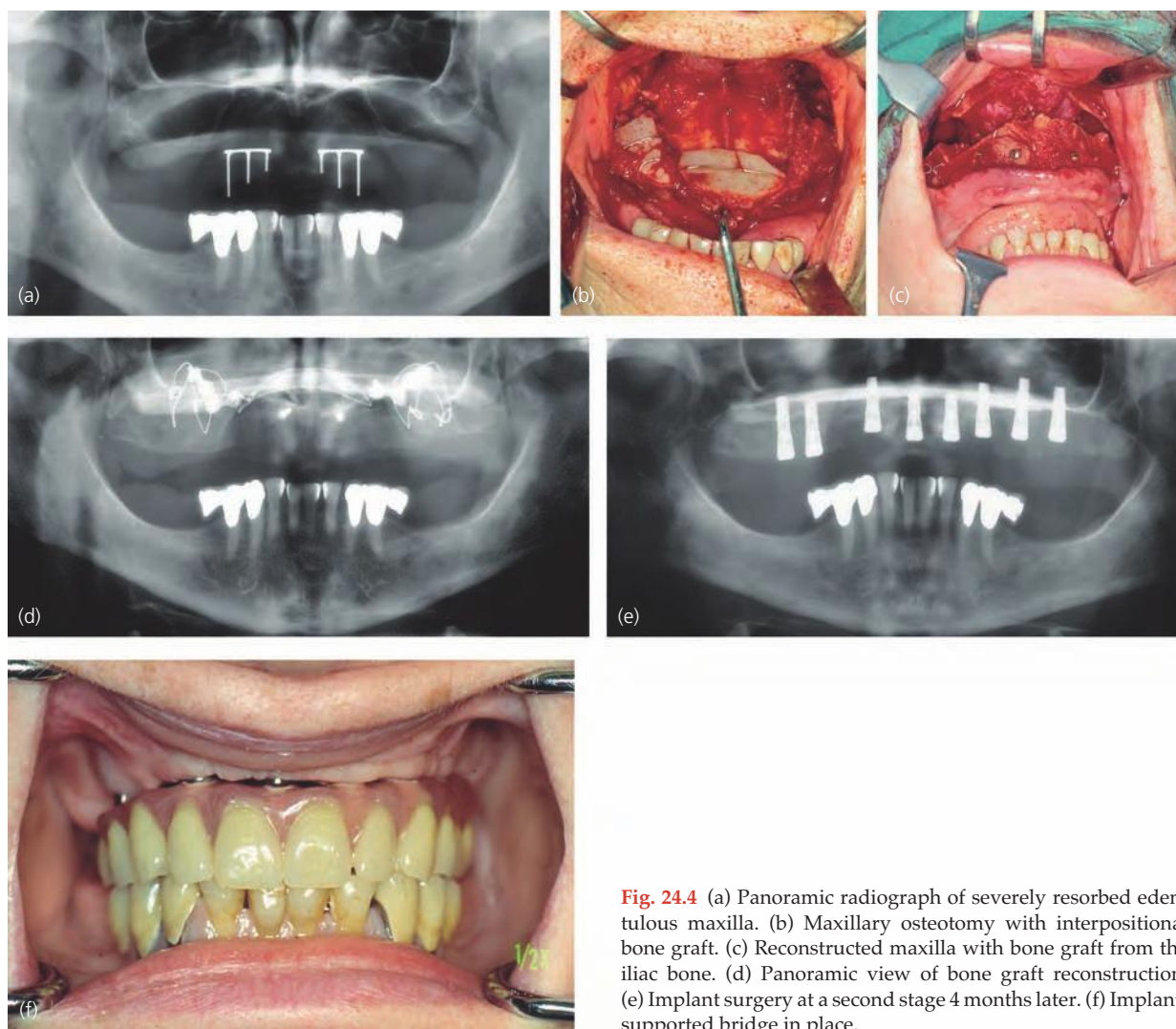


Fig. 24.4 (a) Panoramic radiograph of severely resorbed edentulous maxilla. (b) Maxillary osteotomy with interpositional bone graft. (c) Reconstructed maxilla with bone graft from the iliac bone. (d) Panoramic view of bone graft reconstruction. (e) Implant surgery at a second stage 4 months later. (f) Implant-supported bridge in place.

two-stage procedure with bone graft healing for 4–5 months and implant healing for at least 3–4 months (Fig. 24.2).

Bone graft reconstruction of the jaws before implant placement

Surgical incision and flap technique are of utmost importance for obtaining a positive result for a grafting procedure. If possible the incision line should not be placed over the bone graft, but preferably in the vestibule to avoid contamination of the graft from the incision line. The relaxation incisions should also be placed away from the grafted area and should allow closure of the incision without tension or gaps. The flap should not be stretched at the time of wound closure, as this results in immediate constriction of the vascular supply and subsequent bone graft exposure.

The flap should be as thick as possible: a larger vascular net in the flap gives a better chance of revascularization of the bone graft. Preparation of the graft site is also important for healing and for the rate of bone graft integration. If the recipient site is cortical it

can be an advantage to make some small holes in the surface with a round bur. The contact area between bone graft and recipient site should be as good as possible to facilitate bony union. Smoothing the contours of a bone graft block with a drill to avoid sharp edges or spicules is necessary to avoid perforation of the mucoperiosteal flap.

The bone graft block should be fixed with plate screws of varying length, however overlong screws should be avoided, as these may perforate bone and mucosa on the opposite side. Osteosutures may also be used as retention devices. When particulated bone graft is used a resorbable or non-resorbable membrane may be inserted to keep the bone graft material in position. When using both resorbable and non-resorbable membranes, it is extremely important to have a water-tight flap closure over the region in order to avoid contamination from the oral cavity into the graft region. A non-resorbable membrane is removed at the time of implant placement. Resorbable membranes do resorb within 3–4 months, i.e. about the same length of time as for bone graft integration. If particulated bone is being used, the mucoperiosteal flap can serve as a membrane; the flap is raised no more than is absolutely necessary so there is a pouch

to fill with particulated bone or biomaterial. In these cases relaxation incisions are not required.

The healing time for onlay grafts and inlay grafts is between 3 and 6 months. Dense cortical bone graft material takes longer for bone integration. If the surgical approach into the grafted area is made too early there is a risk that the bone graft will come out and the whole procedure will have to be done again.

With particulated bone graft there is a risk that resorption will remove parts of the graft if one waits too long. Ideal times for cortical bone grafts are between 4 and 6 months and for spongy particulated bone grafts between 3 and 4 months. Healing time for biomaterials like Bio-Oss[®] granules are at least 6–9 months. If the Bio-Oss[®] grafted site is approached too soon, there will only be loose Bio-Oss[®] particles and the procedure will have to be performed all over again. When using Bio-Oss[®] or similar biomaterial, a biological membrane like Bio-Gide[®] or similar can be used to collect and concentrate the granules; the flap is then carefully repositioned and closed to form a water-tight seal, otherwise inflammatory reactions may occur and jeopardize both the graft material and part of the original crest.

Grafting procedures as a whole demand surgical skill and experience both in obtaining the graft material from different regions (see Chapter 23) and in preparing the graft site and handling of the bone graft material. If these procedures are not performed well, the risk for failure is obvious.

Avoiding crestal incisions in bone grafting procedures is not always possible but when there is an opportunity to do a vestibular incision and raise the flap over the crest, enabling total closure over the bone graft block, it is a clear advantage. However, this technique is not always possible or practical and then a mucoperiosteal flap must be used, which is lengthened by periosteal cutting at the base of the flap to ensure a tension-free flap when closed.

Use of platelet-rich plasma

The use of platelet-rich plasma (PRP) in connection with bone grafting was introduced by Robert Marx in 1948.⁶⁵ He used a concentrate of platelets when doing cancer reconstruction cases. The theory of getting a concentrate of platelets into the wound area is to concentrate bone morphogenetic proteins, bone morphogenetic enzymes, and immunoglobulins as close as possible to the bone graft material in order to enhance and speed up the bone regeneration and bone integration processes.

Later studies with both animals and humans have not been able to confirm the effect on bone regeneration. However, a markedly noticeable effect on soft tissue postoperatively has been observed. Concentration of enzymes and immunoglobulins may reduce the initial inflammatory surgical response and therefore result in a less pronounced inflamma-

tory postsurgical reaction. Human grafting studies by Thor and co-workers do, however, suggest that there may be a slight adjunctive effect of PRP on bone healing and bone regeneration, especially in conjunction with use of implants which have a moderately roughened implant surface.^{50,66–69} Further studies will be needed to verify the benefit of PRP in bone grafting cases.

The reason for the limited effect on bone graft healing could be that PRP releases enzymes and bone morphogenetic proteins with a very brief action time span, while bone regeneration is a slow process with a time span of weeks and months. Once it is possible to have slow-releasing adjuvants or surfaces on implants to allow for slow release of enzymatic substances, there may be a dramatic change in the effect of PRP and related substances. Until then it is advisable to reduce surgical time both in the bone graft harvesting and positioning and modeling of the bone graft, in order to maintain the graft's vitality. In a cortical bone graft block there will be a gradually "creeping substitution" of capillaries to vitalize and build up the new bone from inside and outside.

Staging of grafting procedures

Bone grafting by use of autogenous bone graft material can be done in one- or two-stage procedures. A one-stage procedure means that the implants are installed at the same time as the grafting procedure. A two-stage procedure means that one waits for healing of the bone graft before placing the implants.⁷⁰ In a one-stage procedure the outcome of the implants depends on revascularization and vitalization of the bone graft before osseointegration can occur.⁷¹ Experimental studies have shown that there is a slow phase in bone-implant contact in the bone graft area compared to in normal non-grafted bone tissue. The implants will depend on the residual bone tissue for primary stability and osseointegration before the bone graft gets enough microcirculation and living cell population for the osseointegration process. If the primary stability in the residual bone is too poor or the naturally occurring remodeling process due to the surgical trauma impairs the primary stability, there is a big risk of failure of the implant. The matter of immediate or early loading of the implants also depends on the initial stability and on the loading forces. Immediate or early loading can provide stimulation for the osseointegration process but may also be a disaster if the balance between stability and loading is negative.

Split crest surgery

An option already discussed for reconstruction of the alveolar crest which is too thin for implant placement is the buccal onlay graft, which will enlarge and

thicken the crest thus enabling implant placement. However, if the anatomy is suitable for splitting of the crest, i.e. two cortical layers and an intermediate marrow space, it may be possible to split the crest in the middle and place bone graft in between the two plates. If the alveolar crest is composed of only cortical bone there is a big risk of fracturing and destroying the crest. The incision should be made on the alveolar crest top, with minimal exposure of the bony surfaces on either the palatal side or the buccal side, so that the circulation to the two bony plates is not jeopardized. An osteotomy can be carried out with a chisel, oscillating saw or Lindemann drill to get the right direction. The two bony pieces are then separated with a chisel and the buccal bone segment with attached soft tissue fractured out of the base of the alveolar process. Particulated bone or Bio-Oss® can now be placed adjacent to the implant and the soft tissue closed. The procedure may be most successful in cases where there is a possibility of placing thinner implants without splitting the crest. However, in those cases where there is an indication for split crest surgery, the method has proven to be successful. If the crest is split in pieces a more complicated grafting procedure has to be done.

Complications

Complications may arise due to inadequate coverage of the graft or flaps which are stretched too much, with compromised vascular circulation. Exposure of the graft may result in loss of part or all of the graft. Smoking may seriously affect graft healing since capillary sprouting is reduced by nicotine.

Wound dehiscence problems may be the most frequent complication, since healing contraction may disrupt the wound. That is why it is so important to have a flap which covers the surgical area without tension. Stretching of the flap to close the wound may impair healing due to strangulation of the small vessels and capillaries. Wound dehiscence in the early stage of healing is mostly located in the incision line; however, in later stages after several weeks of healing, dehiscence may appear where the vascular supply is inadequate to supply and revascularize the bone graft material.

In early dehiscences there is a big risk of losing the bone graft totally and contamination from the oral cavity will make a reclosure almost impossible since the bone graft has already been contaminated. When the dehiscence occurs at a later stage, when a major part of the bone graft has been incorporated by revascularization and regeneration of bone, the problem is more limited to the actual area of exposure. The part of the bone graft which has been exposed and devitalized can gradually be removed and secondary healing will close the dehiscences over vital bone tissue.

Inlay grafting is performed with extremely thin maxillae, so there is always a risk for fracturing of the

palate or the sinus walls. It is possible to repair fractures by use of bone graft but it may be difficult to advance the maxilla once it has been fractured. Sinus infection can occur but is not a common complication.

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Chapter 25

Implant Rehabilitation in the Posterior Maxilla Using Autogenous Bone Material

Karl-Erik Kahnberg

The chapter contains descriptions of techniques for bone augmentation in the posterior part of the maxilla by use of autogenous bone graft material. The morphologic and anatomic aspects of the maxillary sinus area are described in the first part of the chapter. Bone graft harvesting from different regions depending on the volume needed is briefly commented upon. The surgical techniques for sinus lift procedures with autogenous bone graft material in partially edentulous cases as well as single tooth cases are described. Alternative surgical solutions, like the impaction technique, are also included in the chapter. Finally possible complications that may happen in the sinus lift procedure are covered.

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Introduction

For a long time the sinus lift procedure has been the solution for implant rehabilitation in the posterior maxilla with a limited amount of bone below the sinus cavity. Sinus lift involves opening a bone window to get access to the sinus cavity and gentle lifting of the sinus membrane, thereby creating a space for graft material. There are several surgical modifications and methods for the procedure but the aim of all of them is to increase the amount of bone for implant integration.

It is extremely important to keep the sinus membrane intact when it is elevated in connection with bone graft inlays. Since the drainage from the maxillary sinus is effected only through the osteum, a small canal leading out in the nose below the second nasal

concha, there is a huge risk that infections established in the maxillary sinus cavity with minimal drainage will be very severe. As soon as bony fragments or salivary products gain access into the sinus cavity there is a risk for sinusitis development. A massive sinus infection will drastically reduce the chance of inlay grafts surviving and thus prevent anchoring of implants.

If the available amount of bone volume below the sinus cavity approximates 10–15 mm there may be no need for a sinus lift procedure. It is therefore important to have determined the bone volume preoperatively by tomographic measurements or computed tomography (CT) evaluation.

Background

The technique and idea of reconstruction and augmentation of the alveolar crest within the sinus cavity was originally introduced by Hill Tatum in the 1970s.¹ His idea of the window technique has since then been approved and used worldwide.^{2–18} Invasive surgery into the maxillary sinus cavity has not always been successful. Perforations of the sinus membrane in connection with extraction could result in sinus fistula and sinus infection. Dislocation of a root into the sinus cavity would lead to a massive sinusitis unless treated. There is potential for inflammatory reactions in the maxillary sinus since the cavity is a closed space with very limited drainage capacity: the osteum is the only site of drainage and it is small. These circumstances inevitably make the sinus lift procedure sensitive, since it is of utmost importance to keep the sinus membrane intact to avoid any complication of graft or biomaterial going into the sinus cavity causing inflammation. The space to be used for implant rehabilitation is normally just a small part of the total volume of the maxillary sinus. With careful surgical technique and avoiding membrane perforations, the

results have been encouraging as reported in a massive literature.^{19–29}

The modern type of implantology focused initially on rehabilitation in the edentulous mandible. Removable dentures were most problematic in the lower jaw, where there were difficulties in achieving good retention. When implants were developed the initial area of use was the lower jaw. The results of the screw-type implants of the Brånemark type have shown excellent results in the lower jaw for more than 40 years. However, in the upper jaw it is often more problematic because there is more spongy and less cortical bone. Furthermore the hollow structure of the maxilla, with maxillary sinus and nasal cavity, reduces the bony base for implant surgery. As long as the alveolar processes maintain a decent volume both in height and width there is a good opportunity to install implants.^{30–40} However, if the residual bone volume beneath the maxillary sinus and nasal cavity is too small to provide anchorage for dental implants the alternative is to augment the residual bone volume either by onlay grafting or inlay grafting. Inlay grafting using the maxillary sinus recess or the nasal floor below the sinus membrane and the nasal mucosa, respectively, is a possibility for ridge augmentation. The techniques have been named sinus lifting and nasal lifting procedures, respectively. The maxillary sinus and nasal cavity occupy a large space in the midface and expansion of the sinus cavity down into the alveolar process often occurs in conjunction with tooth loss. The most logical augmentation for implants is the inferior part of maxillary sinus and sometimes also the nasal cavity by inlay grafting. With this type of augmentation the vertical height of the alveolar process will not be affected which may be a slight disadvantage, but the bone graft will be surrounded by bony walls of the sinus recess.

Current understanding is that the sinus membrane is not osteogenetic, so just lifting the mucosa does not produce more bone, otherwise the sinus cavity would be filled with bone. It is utterly important to keep the sinus membrane intact when it is elevated in connection with bone graft inlays to avoid sinus infection.^{41,42}

Morphologic aspects

The maxillary sinus in the newborn infant is only a small invagination in the middle concha. With time it expands into corpus maxillae and with some variations fills out the maxillary bone. The border zones are superiorly towards the orbita, medially to the nasal cavity, posteriorly towards the pterygopalatine fossa and infraorbitalis, inferiorly towards the alveolar process and laterally to the zygomatic process. The maxillary sinus drains through the ostium which is situated in the middle concha region. The ostium is located at the top of the maxillary sinus; sometimes there may be one or two accessory openings. The

deepest point of the maxillary sinus is normally located in the molar tooth area. Root tips of molar teeth and sometimes also premolars may project up in the sinus cavity covered only by very thin bone and by the sinus membrane, also called the Schneiderian membrane. The sinus membrane is in contact with the breathing air and also acts as a biological membrane with immunological properties like the nasal mucosa, but to a lesser degree. The membrane is multilayered, with cylindrical ciliated epithelium on the surface and an underlying basal membrane similar to periosteum. Mucus cells in the epithelium produce liquid that keeps the membrane moist and protects the ciliated epithelium. Seromucous glands with mainly serous secretion can be found in the sinus membrane. The transport capacity of the ciliated cells is limited to minor particles like dust but not larger ones like bone graft particles and root fragments. Damage to the ciliated epithelium results in partial or total stop in secretion and removal of minor particles.

The maxillary sinus in the adult can vary considerably in size and there is a continuous extension with increasing age. Expansion of the maxillary sinus into the alveolar process is especially observed in connection with extraction of molar teeth and also premolar teeth. Edentulism creates a situation where finally only a paper thin bony wall exists between the oral cavity and the sinus cavity. The maxillary sinus cavity is usually one big solitary cavity but it may also sometimes be divided into smaller chambers separated by septa. The origin of these septa may be a consequence of the sinus going deeper in the molar region and thereby forming septa towards the anterior part. The maxilla has a dense vascular network. The maxillary sinus is supplied by branches from the maxillary artery via the infraorbital artery and the greater palatal artery and the facial artery. Venous flow is via the sphenopalatine vein, the pterygoid plexus, and the facial vein. There seems to be a connection between reduced vascularity and atrophy of the alveolar process. Loss of the maxillary teeth and increasing age result in marked reduction in the vascularization of the alveolar process.

Surgical technique

The lateral window technique is by far the most frequently used surgical procedure (Fig. 25.1). The lateral window gives access to the sinus cavity and enables positioning of graft material. The buccal windows are made by a round bur, diamond bur or piezoelectric surgery for gentle removal of bone over the sinus membrane. All possible precautions should be taken to avoid perforation of the sinus membrane. The smooth touch technique is recommended in approaching the sinus membrane. The window may be infracted, in which case the superior part of the bone incision is only part way through the buccal bone. Alternative

options are to loosen the window but still have it attached to the sinus membrane, or remove the window completely and use it as a free graft (Figs 25.2–25.4). The sinus membrane is carefully elevated with a blunt instrument from the bottom of the sinus cavity. It can be difficult to elevate, especially if extractions have been performed recently, causing a tight connection with the sinus membrane (Figs 25.3, 25.4).

Another critical point is in the superior corner of the window where the infraction technique is used. The infraction may give rise to sharp contours which may easily cause perforation of the sinus membrane. If the perforation is very small a resorbable membrane (Bio-Gide®) can be used to cover the perforation. If the membrane is manipulated or wrinkled too much, trying to suture it, to cover the perforation, there is a clear risk that the perforation will be enlarged and destroy any chance of sinus lifting augmentation. Complications like sinusitis may occur if there are loose particles within the sinus cavity.

Bone graft material can be obtained from the iliac bone, cranial bone, tibial bone or mandibular bone. Small amounts of bone material can also be obtained by use of the bone collectors like Bone Trap® or safe scraper (Fig. 25.5). The amount of bone needed depends on the type of procedure. Bilateral sinus lifting for rehabilitation of the premolar and molar

regions demands a large amount of bone graft material, whereas for unilateral sinus bone graft, material from the mandibular angle or chin is enough. Sinus lift for single tooth replacement needs even less bone graft material.

The bone graft material from the mandible is mainly cortical, as is cranial bone graft, whereas bone graft from the iliac bone and tibial bone consists of cancellous bone material. Cortical bone graft is more resistant to resorption but normally a mixture of cortical and cancellous bone graft is used.

The bone graft material, either in cortical block or particulated bone, is placed below the sinus membrane in the inferior chamber of the sinus cavity. Depending on the clinical situation the graft material can be immobilized with wires, plate screws, or it can just be packed below the sinus membrane. It is important to ensure that the bone material is stable and not moving around. A piece of cortical bone can be used as a roof for the particulated bone which is collected in the sinus inferior recess. The bone can easily be immobilized with wire osteosutures or plate screws. The surgeon may choose to use a resorbable membrane to cover the bone graft. In a two-stage procedure the bone graft is normally left to heal for at least 4 months. Shorter healing time will risk failure of the bone graft; if the healing time is too long, resorption of the graft will begin. The question of whether to do

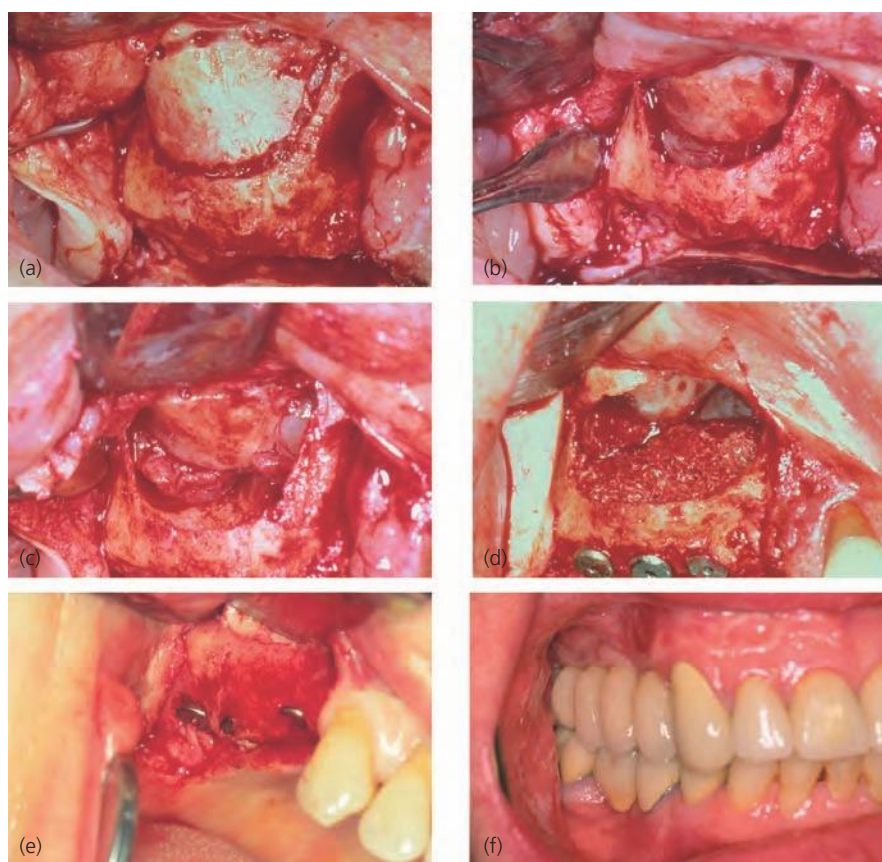


Fig. 25.1 (a) Sinus lift by use of the “window technique”. A lateral bone “window” is created using a round bur. (b) Infraction of the buccal window. (c) Sinus membrane is lifted and a space is created below the window and the sinus membrane. (d) Bone graft is positioned in the lower part of the sinus cavity and kept in place by the implants (one-stage surgery). (e) Bone healing after 6 months. (f) Implant-supported bridge in place.

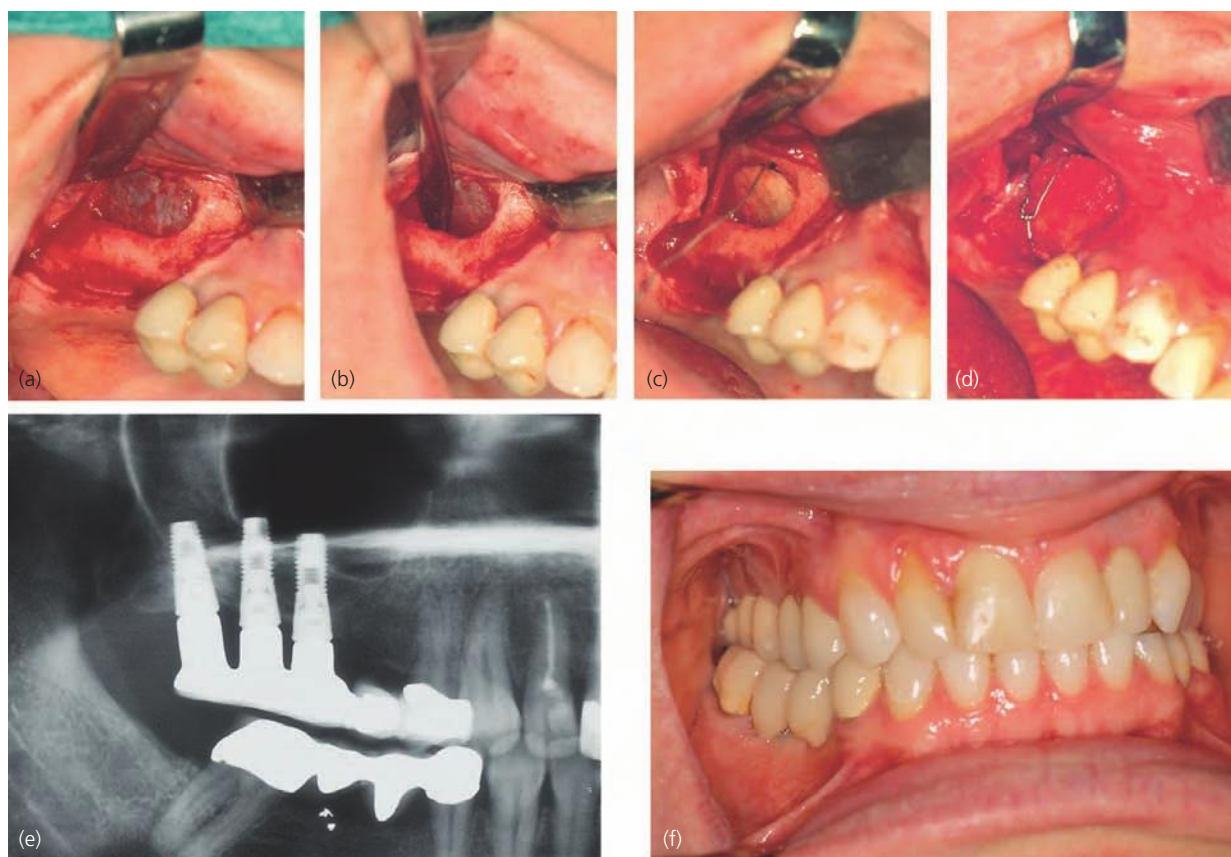


Fig. 25.2 (a) Sinus lift by use of a two-stage technique. Bone window is removed and sinus membrane is lifted. (b) Space for the bone graft is created in the basal part of the sinus cavity. (c) Cortical bone graft is positioned as a roof in the basal part of the sinus cavity. (d) Particulated bone graft is placed below the cortical “roof” and secured with wires. (e) Radiograph of implants in the sinus graft. (f) Implant-supported bridge.

the bone grafting and implant placement in one or two stages is still open to discussion and needs more clinical scientific follow-up and analysis.

It is extremely important to have enough bone volume below the sinus cavity when doing one-stage surgery, attaching the bone graft with implants. Bone graft and implants have to be stabilized rigidly and even resist some reduction of marginal bone level and the grafted bone in the postoperative remodeling phase. If the alveolar bone below the sinus cavity is too thin (<5–6 mm) there is a high risk that the implants, together with bone graft material (blocks or particulated), become mobile with subsequent failure of the implants and the bone graft. However, in cases with more bone volume available in the alveolar process the one-stage technique can be suitable. Primary stability should be enough for one-stage surgery when there is 5–10 mm of residual bone volume in the alveolar process.

There are many opinions on immediate or early loading of the implants placed in grafted bone. If the residual bone volume in the alveolar process below the maxillary sinus is qualitatively and quantitatively sufficient to stabilize the implants and give them an initial primary stability ($R_f \approx 60$) it is possible to use early loading. The maxillary bones, however, are not often of good enough quality for immediate loading. Immediate or early loading can have a stimulating effect on bone healing and osseointegration, depend-

ing on the extent of loading and the primary stability of the implants. However, even with resonance frequency measurements (RFM), which give the surgeon an idea of primary stability, it is difficult to know whether immediate loading will be appropriate in individual cases.

Sinus lifting procedures using an impaction technique were originally presented by Summers.⁴³ The purpose of this technique is to initiate a split of the alveolar crest between the inner cortical part towards the sinus cavity and the outer cortical part (towards the crest).^{44–46} The residual bone in the alveolar crest should be at least 6–7 mm and contain both cortical bone and marrow to be suitable for the impaction procedure. The intention with Summer’s impaction technique is to fracture the cortical bone underlying the sinus cavity in such a way that the sinus floor can be elevated without rupturing the sinus membrane.⁴⁴

The procedure begins with flap elevation then preparation of implant sites with a round bur and spiral bur, but only through the cortical bone and slightly into the spongy bone (2–3 mm). The subsequent surgery is done with osteotomes which normally have the same diameter as the implant surgery drills. By gradually enlarging the implant site with osteotomes up to the desired length and width the implants can be installed without perforating the membrane. Usually the best result is obtained with

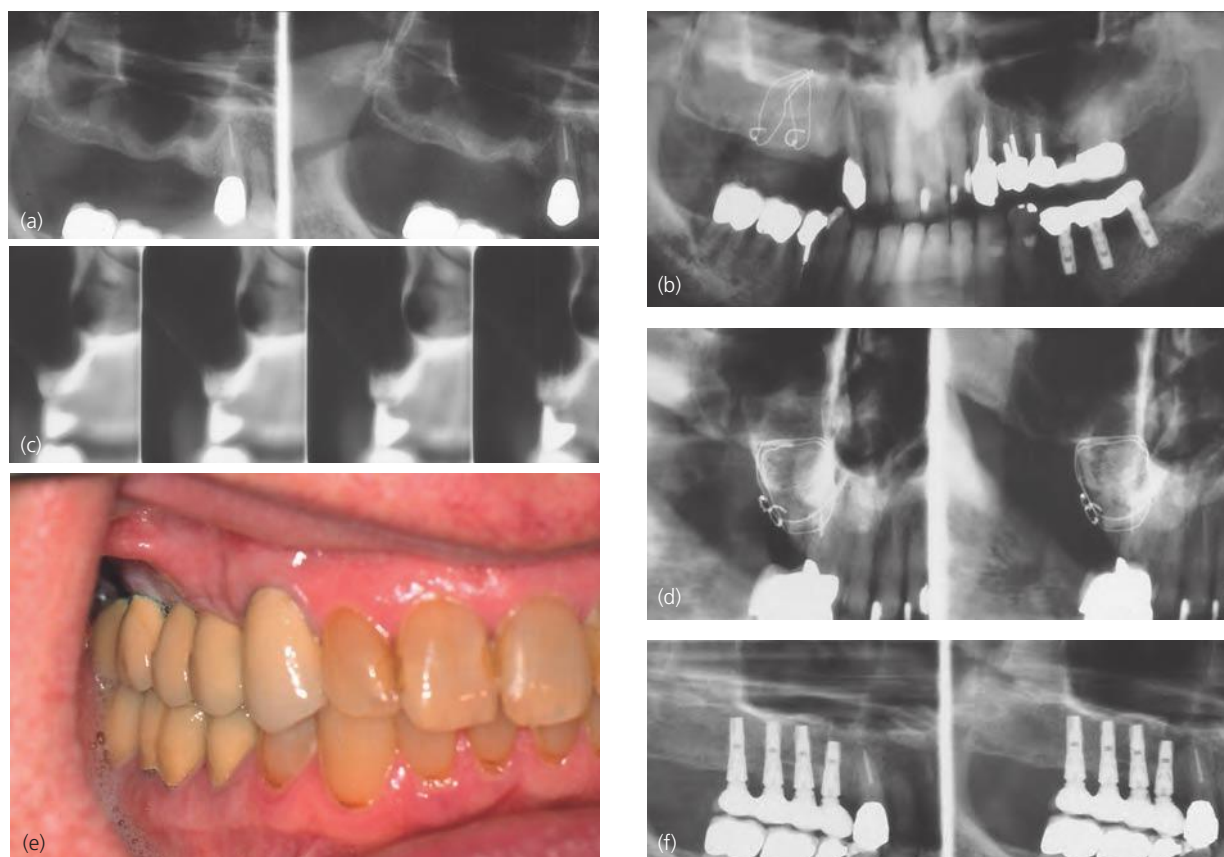


Fig. 25.3 (a) A case with a large sinus cavity and minimal residual bone volume. (b) The same case after sinus lift and bone grafting. (c) Tomograph in close section of the posterior maxilla before grafting. (d) Tomograph after sinus lift and bone grafting. (e) Clinical view of implant-supported bridge in place. (f) Radiograph after implant rehabilitation.

two to three implant sites in a row to be able to induce the sinus floor fracture. With only one implant site it is more difficult to achieve the same sinus floor elevation. The effect with only one impaction site may be very limited and the risk of sinus membrane perforation increases. In indicated cases, sinus impaction is a less demanding procedure for the patient, but still with predictable results.⁴⁴⁻⁴⁶ The choice of filling the space below the elevated sinus floor with bone graft or biomaterial is up to the surgeon. However, it is important to check for eventual perforations in the sinus membrane. Healing time after impaction and implant installation should be a little more extended than for conventional implant surgery.

In cases of single tooth replacement in the posterior maxilla where the available bone volume is too small to allow for conventional implants, a modified kind of sinus lift can be made. Where the sinus cavity has expanded down into the alveolar process after tooth extraction there is an ideal situation for a local sinus lift (Fig. 25.5).⁴⁰ A crestal incision is made in the edentulous area and connected with diverging relaxation incisions in the vestibule. A circular area over the sinus cavity bone is removed to the sinus membrane and the bone material collected with a bone collector during the preparation. The sinus membrane is carefully lifted and the implant installed through the crestal bone by standardized technique. The implant is allowed to penetrate into the sinus cavity below the

sinus membrane about two to three times the residual bone volume. The sinus membrane is thus held up by the implant which acts as a tent pole for the membrane. The bone material is placed around the exposed part of the implant but no attempt is made to completely fill the space around the implant. Recent results indicate a predictable outcome of the method.

The most frequent complication associated with sinus lifting procedures is perforation of the sinus membrane with maxillary sinus infections. Drainage from the sinus cavity is very limited with only the osteum as a natural canal, so there is a high risk for sinusitis after perforation with graft material moving around in the sinus cavity. Sinusitis must be treated with antibiotics and, often, the sinus must be reopened and cleaned, maybe removing part of the graft material. Dislocation of an implant into the sinus cavity may also occur which demands exploration and extirpation. Complications like lack of osseointegration, as in conventional implant surgery, may also occur. The complications in sinus impaction surgery are also connected with perforation of the sinus membrane and dislocation of graft material.

Literature review

There has been a large number of publications during the last decade regarding the outcome of sinus lift

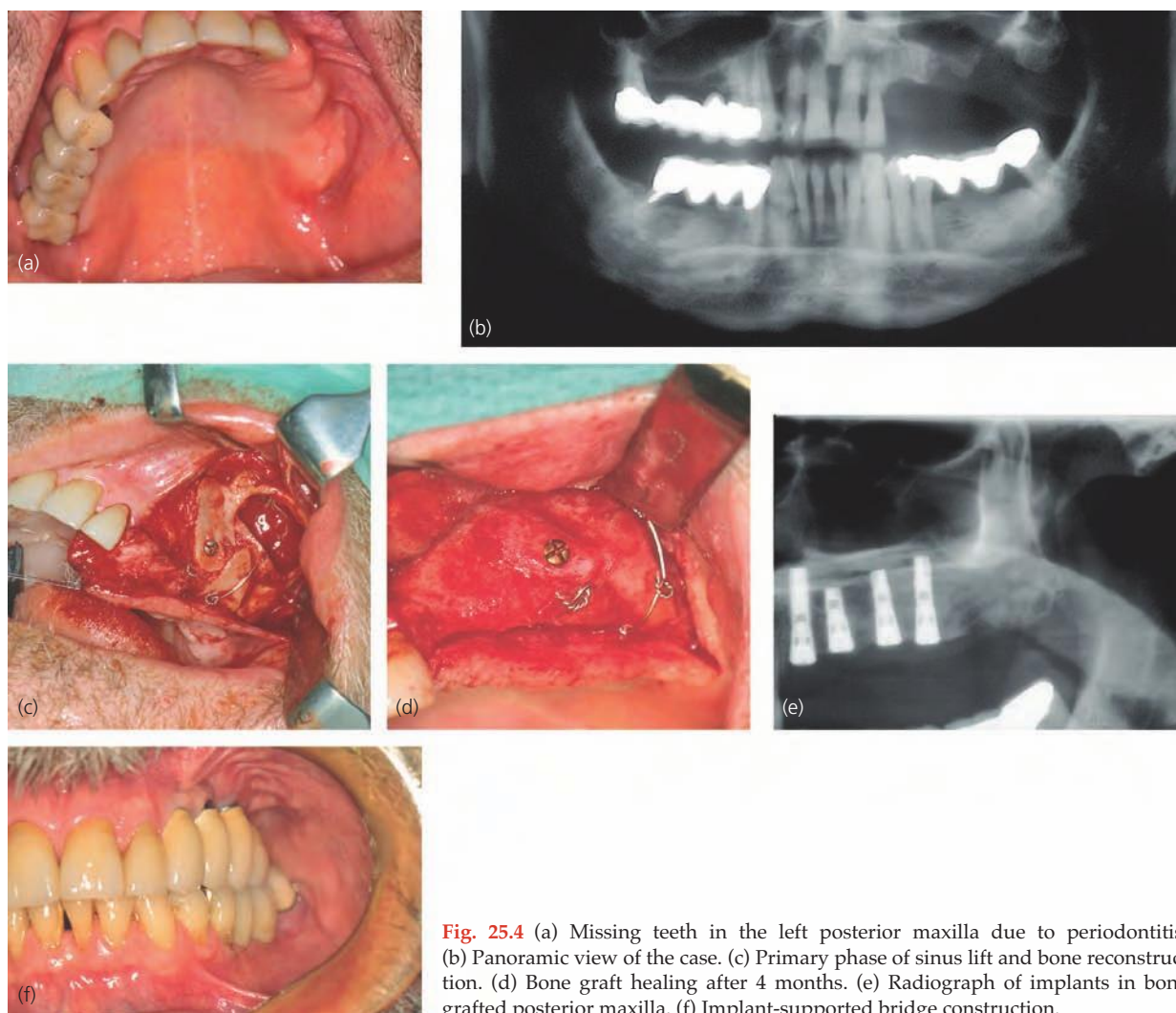


Fig. 25.4 (a) Missing teeth in the left posterior maxilla due to periodontitis. (b) Panoramic view of the case. (c) Primary phase of sinus lift and bone reconstruction. (d) Bone graft healing after 4 months. (e) Radiograph of implants in bone grafted posterior maxilla. (f) Implant-supported bridge construction.

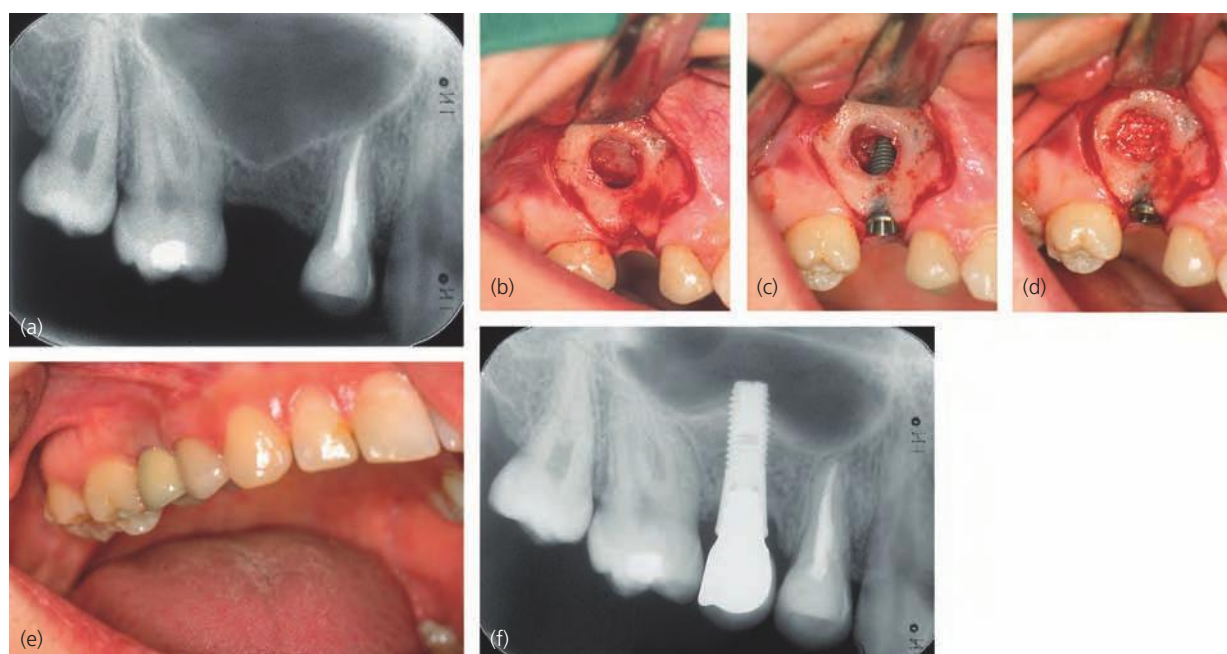


Fig. 25.5 (a) Second premolar missing in the posterior maxilla. Sinus cavity extending down in the alveolar process. (b) Local sinus lift by use of a circular bone window. (c) Implant insertion into the sinus cavity using the implant as a tent pole for the sinus membrane. (d) Particulate bone from Bone Trap[®] placed around the implant. (e) Crown restoration in place. (f) Two-year postoperative control showing a satisfactory bone filling around the implant.

procedures. Both retrospective and prospective type studies have been made regarding the outcome and results from varying periods of follow-up. One-stage surgical protocol, two-stage surgical protocol, and immediate loading results have been published.

The presurgical conditions may not always have been clearly defined which makes it a bit difficult to evaluate the impact of the bone grafting in the osseointegration process.

The reports in the literature about complications with sinus membrane perforations and subsequent treatment and treatment outcomes have been relatively few. However, the quality and thickness of the sinus membrane may vary quite a lot and the very healthy membrane can be extremely thin and consequently very vulnerable to a surgical approach. Repair of the membrane by suturing is not possible with the normal healthy ultra thin membrane but could eventually be made with a chronically affected thicker sinus membrane. The use of resorbable membranes like Bio-Gide® is a possible solution to cover small perforations in the membrane since the collagen material sticks to the membrane and may be an effective seal. Modifications of the conventional sinus lift procedure using particulated bone graft, cortical bone blocks or different kinds of biomaterials have also been reported in the literature.

Experimental studies both in animals and humans trying to lift the sinus membrane with the implants (implants acting as tent poles) and just collecting blood coagulum or blood with additional platelet-rich plasma have given some bone augmentation.⁴⁷⁻⁵¹ Long-term studies of the outcome by use of these methods are still not available.

There are numerous publications on sinus lift procedures either one or two-stage techniques, with immediate or delayed loading and with different kinds of grafting materials. Most studies present with a success and survival rate of between 90 and 97% with few exceptions. The method is well documented and generally has a predictable outcome. Sometimes the residual bone volume could have been enough for conventional implants, especially the short implants recently described. Long-term follow-up of the different solutions for implant rehabilitation in the posterior maxilla will evaluate which method is the most reliable and also indicate when and where we should be using the different techniques. Clinical and experimental studies will provide recommendations in due course.

Complications associated with sinus lift procedures

The sinus lift procedure is not without problems. Due to the anatomical situation with a large hollow space

with a minimal drainage via the osteum, infections may very well arise without proper drainage. Complications may happen during the operation with perforation of the sinus membrane during careful dissection and lifting. Small perforations can usually be repaired by use of a collagen membrane like Bio-Gide®, however larger defects more than 5 mm can be problematic in some cases and make the procedure impossible. Suturing of the sinus membrane has been suggested, but it is absolutely impossible with a healthy membrane which is thin like a soap bubble; it could sometimes be possible where there is a chronically inflamed and thickened membrane. When a bone graft block is used some authors claim that a perforation is not a risk factor for sinusitis, but that particulated bone graft is more prone to initiate inflammation if bone particles are moving around the sinus cavity.

Obstruction of the osteum prevents normal drainage of the maxillary sinus and should be avoided.

If a sinus infection occurs postoperatively it is of utmost importance that it is treated early in order to avoid a massive inflammatory reaction with obliteration of the sinus cavity and suppuration. Usually graft material or biomaterial particles have entered the large sinus cavity via a perforation. Access for evaluation, and drainage of the sinus cavity, are limited. It may even sometimes be necessary to make a naso-antral passage below the inferior concha in the nasal floor.

Bleeding in the sinus cavity is not desirable, since the blood clot may be a source for infection. The postoperative complications that may occur include wound dehiscence due to swelling and mechanical forces on the suture line. Signs of infection may appear during the first two postoperative weeks but can also occur at a later stage during the healing period.

Actions which can influence the intrasinus air pressure, such as sneezing, blowing the nose, and even drinking through a straw, should be avoided as much as possible as they may create a negative intrasinus pressure which can jeopardize the membrane healing process.

Implant failures may appear at an early stage within the healing period but usually the lack of osseointegration is noticed at the abutment connection. Temporary denture wearing should be avoided during the first two postoperative weeks in order to let the soft tissue incision heal before prosthetic loading.

As with other procedures involving use of bone graft material, smoking is a negative factor for the healing process due to nicotine's interference with the capillary growth during the microvascularization of the bone graft. Heavy smokers should be advised to stop smoking for at least 6 or even up to 9 months prior to the operation.^{51,52}

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Chapter 26

Biomaterials for Bone Replacement in Implant Surgery

Carlo Maiorana

The chapter is a comprehensive overview of biomaterials which can be used as grafting material in the jaws. Different criteria for a biomaterial to be used in the human body are listed. The biomaterials most used in osseointegration are then described in detail. The different surgical situations where biomaterials can be used are presented, including postextraction sites, horizontal defects, vertical defects, sinus elevation and reconstruction in major, advanced osseointegration. Finally there is a section on the possible role of growth factors and bone morphogenetic proteins (BMP) in the integration of biomaterials.

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an alteration of the intermaxillary relationship which is not compatible with prosthetically guided implantology. As for other areas, bone augmentation procedures can be performed prior to placement of the implant, concurrent with implant placement, or subsequent to it.

Up to 10 years ago, autogenous bone was considered to be the gold standard in the reconstruction of atrophic areas of the jaws, due to its osteoconductive, osteoinductive and regenerative properties; it should still be chosen as the most suitable material in severe atrophies (class V and VI according to Cawood and Howell¹). In the treatment of smaller defects (class III and IV), bone substitutes of synthetic and xenogenic origin have been playing an important role in implant surgery. All those materials, generally called “biomaterials”, are able to favor the adherence of cells and tissue regeneration thanks to a variable degree of osteoconductive activity; after being tested for several years through randomized prospective or retrospective studies, they can be considered as a reliable way to rebuild bone.

Something fundamental is to remember that the difficulty in rebuilding a bone defect is more related to its extent than its depth. In other words, a very deep but localized defect is easier to treat than a superficial but extensive one. Careful preoperative analysis of the site to be regenerated is important instead of deciding at the time of surgery.

Introduction

When bone reconstruction of the posterior atrophic maxilla is needed, different surgical procedures can be used. Following the loss of teeth, alveolar ridge resorption leads to a combined vertical and horizontal reduction of the bony support, at the same time increasing maxillary sinus pneumatization. Such a condition makes implant placement impossible, either because of insufficient vertical bone volume or

Biomaterials

The term biomaterial generally indicates any substance used to create a medical device destined for diagnosis, prevention, control, mitigation or therapy of a human disease, on condition that it persists in the body for at least 30 days after implantation. First of all, cytotoxicity, genotoxicity, and hemocompatibility of a biomaterial have to be evaluated. After that,

attention has to be paid to its macrostructure and microstructure, by evaluating the isotropy. Finally, its mechanical, physical, and chemical properties should be taken into consideration. Which characteristics should a biomaterial have to be considered for implantation in the human body? They can be summarized as follows:

- non-carcinogenic;
- non-antigenic;
- hydrophilic;
- radiopaque;
- easy handling;
- versatile (usable in several clinical fields).

Biomaterials can be obtained from the patient (autogenous), from beings belonging to the same species (homologous), from beings belonging to different species (xenogenic) or from minerals (alloplasts). Apart from autogenous bone, which has osteoconductive, osteoinductive, and osteoproliferative properties, and homologous bone, whose properties are mainly osteoconductive and slightly inductive, all the biomaterials used for bone regeneration are only osteoconductive scaffolds. Bone substitutes were created in order to promote bone regeneration, avoiding the necessity of harvesting bone from the patient. The first materials on the market were represented by ceramic hydroxyapatite of different macrostructures (coral, bioglass, ceramic hydroxyapatite) and the osteoconductive potential, and the resorbability, were not excellent with regard to the implant field. A few years later, demineralized freeze dried bone allograft (DFDBA) from human donors was introduced in the US; osteoinductive properties were claimed for this, because the demineralization process was able to expose bone morphogenetic proteins (BMP). In addition, some publications confirmed an osteoconductive property for this material.²⁻⁴ Unfortunately, the properties of DFDBA were not confirmed by later histologic and clinical studies in sinus elevation and guided bone regeneration (GBR) procedures.

At the same time, xenogenic anorganic bone was obtained from cattle,⁵⁻⁷ followed by similar materials from equine or porcine sources. These mineral scaffolds, resulting from a treatment to eliminate any trace of organic material, promote colonization of bone tissue via osteoconduction. They are slowly replaced by newly formed bone; both the quality and the quantity of lamellar bone is well documented and, at the moment, they are considered a first-choice material in bony defect repair in implantology, with the exception of class V and VI atrophies (Cawood and Howell¹), where the use of autogenous bone, alone or in association with xenogenic materials, is mandatory to rebuild the bony architecture prior to implant placement.

Biomaterials currently used in osseointegration

Anorganic bovine bone

This is an osteoconductive and slow-resorbing material composed of an anorganic mineral matrix deprived of the organic scaffold in order to leave intercrystalline microtunnels and microcapillaries between the bovine apatite crystals. The high osteoconductivity is due to the natural microstructure of the material, which demonstrates a large inner surface area and a system of intracrystalline spaces and microtunnels available for ingrowth of blood vessels and osteoblast migration. Long-term stability has been proved by many clinical studies. There must be no direct contact of the material with the implant surface for good implant osseointegration. The only early contact is between the clot and the matrix particles, and the angiogenesis and osteoblasts deposition occur from there. Integration is due to replacement of the bone substitute with newly formed bone. Histomorphometric analysis demonstrated that anorganic bovine bone increases the mineral portion in regenerated areas as compared to host bone areas.⁸⁻¹² Some of the material remains in the bone tissue and is slowly embedded in lamellar bone, resulting in denser bone, and this could explain the high survival rate of implants placed in areas augmented with it.

Calcium phosphate

Depending on the Ca/P ratio, the presence of water and impurities, this material crystallizes into two different shapes:

- calcium hydroxyphosphate or hydroxyapatite (HA);
- beta calciumtriphosphate (beta TCP).

Synthetic HA has the same chemical composition as human HA, but a slightly different structure. Its rate of metabolism by the human body depends on its structure, chemical composition, and interface surface area.

Beta TCP is also resorbable and is slowly converted to HA inside the human body; it supports early bone apposition (woven bone), although beta TCP's degradation products may provoke an inflammatory response that impairs and reverses bone apposition in the defect site. Histologic evaluations showed a solid host-bone connection at 9 months,¹³ but newly formed bone is confined to the periphery of the graft, the center being partially filled with connective tissue. This material can only be said to be basically resorbable, and no conversion process to trabecular, functional spongiosa occurs, since remodeling cannot take place. A recent report on sinus elevation showed

no difference between a test site treated with beta TCP and a control site treated with autogenous bone in terms of quantity and rate of ossification in a significant sample of patients.¹⁴⁻¹⁶

Calcium sulfate

This material has been used as a synthetic grafting material. Once the sulfate is embedded with water, an exothermic reaction results in crystalization and hardening of the granules. The host bone forms in concentric layers around the resorbing sulfate, which is different to the situation with other ceramics. Dense calcium sulfate does not seem to stimulate early bone apposition, but bone repair is more advanced at 12 weeks as compared with those treated with beta TCP, despite beta TCP's support for direct bone apposition at 1 week. Calcium sulfate appears to provide a more stable osteoconductive scaffold^{16,17} and seems to be a good material in sinus elevation.¹⁸ The osteoconductivity of calcium sulfate is documented, but the clinical experience remains limited.¹⁹

Calcium carbonate

This material is derived from calcified coral polyp skeletons with an aragonite crystal structure and mineral trace elements. Histologic examinations showed absence of direct bony ingrowth of the carbonate in the bone, while connective tissue appeared to encapsulate the implant, isolating it from the autogenous bone and preventing osteoconduction. Bone remodeling looks incomplete due to an insufficient amount of newly formed bone.²⁰⁻²²

Bioactive glass

This is a resorbable amorphous material composed of silicon dioxide, calcium oxide, and sodium oxide. The osteoconductive property is documented,^{23,24} but the most reliable clinical studies in sinus elevation were carried out using a mix of bioactive glass and autogenous bone in single-arm studies or comparing a mix with a control test represented by autogenous bone alone. Studies evaluating bone repair using bioglass alone are necessary to verify the properties of such a material.

Demineralized freeze-dried bone allograft

This material, from human donor bone, has been extensively used in the treatment of periodontal and periapical osseous defects. The material provides a source of type I collagen, which is the only organic component of bone. The processed bone results in lyophilized particles demineralized with hydrochlo-

ric acid. The particles are then recovered by centrifugation, frozen and freeze-dried again. Many reports confirm its ability to induce new bone formation²⁵ thanks to the exposure of BMP, whose inductive properties are well known. Despite this property, DFDBA's osteoinductive action has never been demonstrated and it seems that the regenerated bone is insufficient either in quality or in quantity to allow predictable implant placement, particularly following sinus elevation procedures.^{26,27} Other clinical studies confirm the reliability of DFDBA in sinus elevations, but always in conjunction with other materials.²⁸ Similar results are reported relating to the use of DFDBA in association with expanded polytetrafluoroethylene (e-PTFE) membranes.²⁹

Surgical techniques

The choice of procedure for reconstructing the posterior areas of the maxilla depends both on the depth and the extent of the defect. Starting from the postextraction defects up to the most severe atrophies, the classification according to Cawood and Howell¹ clarifies, in a simple way, what kind of surgical technique is the most suitable for each specific bony defect. In accordance with that classification, the defects can be listed as follows:

- postextraction sites;
- horizontal defects (including dehiscences and fenestrations around implants);
- vertical defects;
- combined (vertical and horizontal defects);
- sinus elevation.

Postextraction sites

An alveolar bone loss of 23% in the first 6 months after tooth extraction and of 11% during the following 5 years was demonstrated by Carlsson.³⁰ Alveolar bone loss not only reduces the amount of bone available for adequate support of the prosthetic load, but can also adversely affect the implant position, the peri-implant hard and soft tissue anatomy, and, consequently, the final esthetic and functional outcome. The immediate insertion of an implant in a postextraction site would therefore preserve a greater amount of alveolar bone and also reduce the treatment time. Whenever an immediate implant placement cannot be carried out due to the impossibility of obtaining primary stability for the fixture, physiological resorption due to the remodeling processes must be avoided. This goal can be easily achieved by filling the alveolus with bone substitutes and a free gingival graft, and implant placement surgery can be carried out about 6 months later.³¹

Where there is an infection in the extraction area, immediate implant insertion should be avoided and the surgery postponed until 40–60 days after the extraction.

Immediate technique

When the gap between implant and alveolus is less than 1 mm, tissue regeneration is entrusted to the clot. For larger gaps, bone substitutes can be used to fill the defect and the area is covered with a collagen membrane and a free gingival graft, which improves the quantity and the quality of the soft tissues around the implant at the time of connection of the healing abutment (Fig. 26.1). The soft tissue graft is obviously useless in cases of immediate temporary crown placement.

Delayed technique

In this situation, 6–8 weeks are needed to rebuild the gingival tissue after extraction and implant placement is performed by means of a standard procedure.

Horizontal defects

These defects occur where there is adequate height but insufficient width of the ridge. They include implant dehiscences and fenestrations and can be

treated with GBR procedures associated with autogenous bone chips and bone substitutes as well as ridge expansion techniques (at implant placement time) or autogenous bone blocks (prior to implant placement).

Dehiscences and fenestrations

Dehiscences are exposures of the implant at the level of its head, occurring when the thickness of the ridge is insufficient in the most coronal area. Fenestrations are exposures of the middle third or the apical portion of the implant with or without involvement of the implant head.

A bone regeneration technique using autogenous chips, bone substitutes, and resorbable or non-resorbable membranes is a possible treatment. Most dehiscences and fenestrations can easily be treated with a resorbable membrane acting as a stabilizer of the underlying material (granules or chips). When the exposed surface of the implant exceeds 1 mm outside the bone envelope and a large volume of bone has to be regenerated, non-resorbable e-PTFE membranes are indicated. In both cases, membranes have to be stabilized with pins or mini-screws and periosteal releasing incisions of the buccal flap are mandatory to avoid tension at suturing. Vertical mattress and single sutures are required in all the techniques.

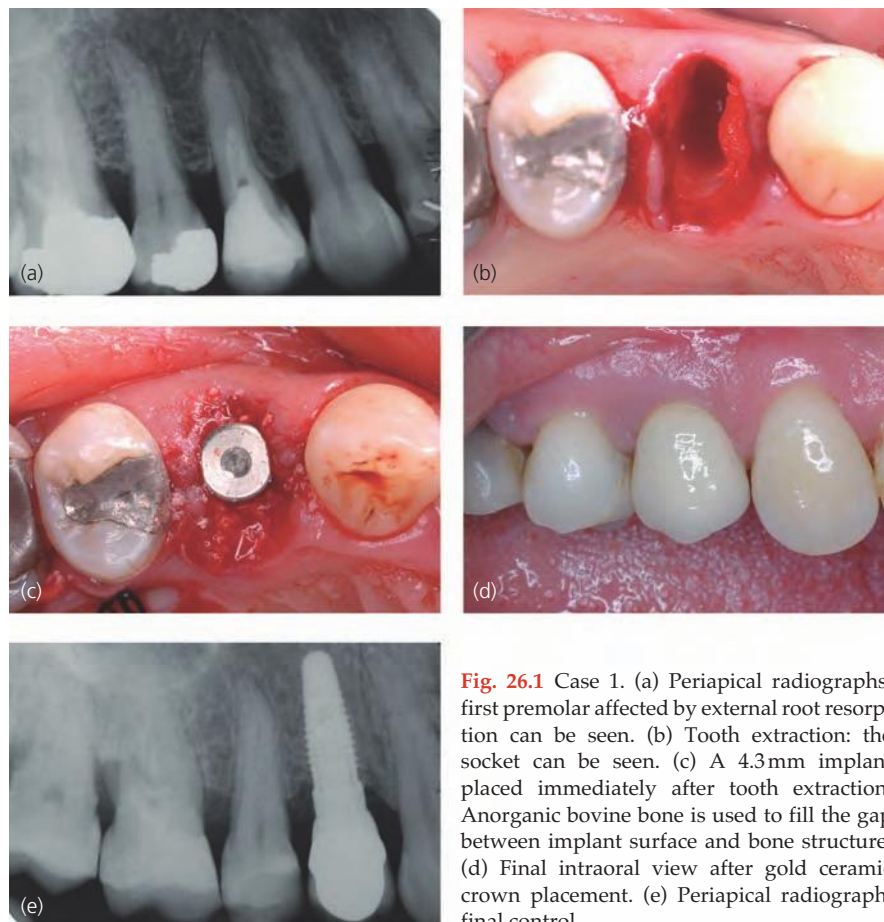


Fig. 26.1 Case 1. (a) Periapical radiographs: first premolar affected by external root resorption can be seen. (b) Tooth extraction: the socket can be seen. (c) A 4.3mm implant placed immediately after tooth extraction. Anorganic bovine bone is used to fill the gap between implant surface and bone structure. (d) Final intraoral view after gold ceramic crown placement. (e) Periapical radiograph: final control.

Autogenous intraoral or extraoral blocks

Harvesting bone blocks from inside the mouth (chin, mandibular body or ramus) is indicated in the correction of a class IV atrophic ridge, particularly in the case of an extended narrow ridge. When a larger amount of bone is necessary (such as a resorbed maxillary ridge in edentulous patients) blocks can be harvested from the hip or the calvaria. In all these situations, attention must be paid to the block's resorption during the 4 months' healing. An average resorption of 20–30% can be observed^{32,33} at implant placement time, in blocks harvested from the chin or the hip, due to their cancellous component; and 15% resorption occurs in blocks harvested from the mandibular body and the ramus, which are basically made of cortical bone. An original technique to reduce the resorption of autogenous blocks was presented in 2005,³⁴ involving a layer of anorganic bovine bone placed on the top of the block and covered by a collagen membrane. Using this procedure it is possible to maintain the original size of the block; it can be successfully used for either intraoral or extraoral grafts (Figs 26.2, 26.3).³⁵

Vertical defects

If the bone height is insufficient to guarantee long-term implant stability or the prosthetic rehabilitation would result in too long crowns, vertical ridge augmentation is mandatory. Vertical augmentation can be achieved by means of corticocancellous blocks harvested from the chin or the ramus or, alternatively, with cancellous bone chips, bone substitutes, and a titanium-reinforced e-PTFE membrane.^{36,37}

Vertical GBR with membrane

This procedure is very predictable, but it has to be carried out strictly according to the surgical protocol, in order to limit the risks of the membrane exposure.

The most common flap design comprises a full-thickness mid-crestal incision within the keratinized mucosa of the edentulous ridge, extended mesially and distally to at least one adjacent tooth. Vertical releasing cuts are performed at the mesial and distal line angles of the incision. A proper preparation of the recipient site is crucial for new bone formation. The buccal and palatal flaps are reflected and gently managed to avoid any perforation of the flap. Stainless steel mini-screws are used as "tent poles" to prevent collapse of the membrane and to predetermine either the width or the height of the future alveolar ridge. The mini-screws are placed and left to protrude out from the bone level to the expected height. The cortical plate is then drilled with a round bur to expose the cancellous bone and to provoke some bleeding. The titanium structure of the e-PTFE membrane is bent with pliers to adapt it to the ridge anatomy and it is trimmed with scissors to extend at least 4–5 mm

beyond the margins of the defect. Once placed over the surgical recipient site, the membrane is secured to the lingual/palatal aspect of the bone crest with fixation mini-screws or pins. The cancellous autogenous chips mixed with the bone substitute are placed to reconstruct the defect and the buccal portion of the membrane is adapted to the vestibular bone plate, and also secured with screws or pins. A releasing horizontal incision of the periosteum is now performed to give elasticity to the flap and obtain tension-free adaptation at closure. The two margins of the flap can be considered sufficiently released when they overlap by at least 7–10 mm. Closure is done with horizontal mattress sutures first and with interrupted sutures later. The membrane is usually removed 6 months after surgery, at implant placement time. See Fig. 26.4.

Sinus elevation

When atrophy of the posterior maxilla reduces the amount of bone suitable for implant placement and a bone augmentation procedure is required, first of all one needs to evaluate if the sinus really has migrated from apical to coronal towards the margin of the alveolar ridge or if the sinus is in its previous position and the vertical height loss is due to vertical resorption. In the first case, a sinus elevation procedure is indicated, while in the second situation vertical ridge augmentation should be performed without any sinus involvement.

Once the necessity of performing a sinus elevation has been decided upon, the second step is the choice between a one- or a two-stage procedure. According to the recent literature,³⁸ a residual alveolar ridge of 4 mm is the absolute minimum height for bone augmentation and simultaneous implant placement and a two-stage technique must be done where there is a residual height of 0–4 mm.

The surgical procedure is the well known Boyne and James technique³⁹ and autogenous bone or bone substitutes have been used for a long time to fill up the subantral cavity. At present, long-term prospective and retrospective studies confirm that bone substitutes are able to regenerate new bone without harvesting autogenous bone and that the implant survival rate in augmented sinuses with biomaterials is significantly higher than that obtained using autogenous bone chips.^{40–42} In Cawood and Howell¹ class VI defects only autogenous bone is recommended, since the severe atrophy needs all the power of an osteoproliferative material.⁴³ This means that at least 80% of sinus elevation procedures can be done by using biomaterials alone.

What kind of bone substitute should be chosen to get the best result? All the bone substitutes currently used in the sinus elevation procedures, either xenogenic or alloplasts, offer osteoconductive properties only. The decision should be taken after considering the human hydroxyapatite structure: the more a

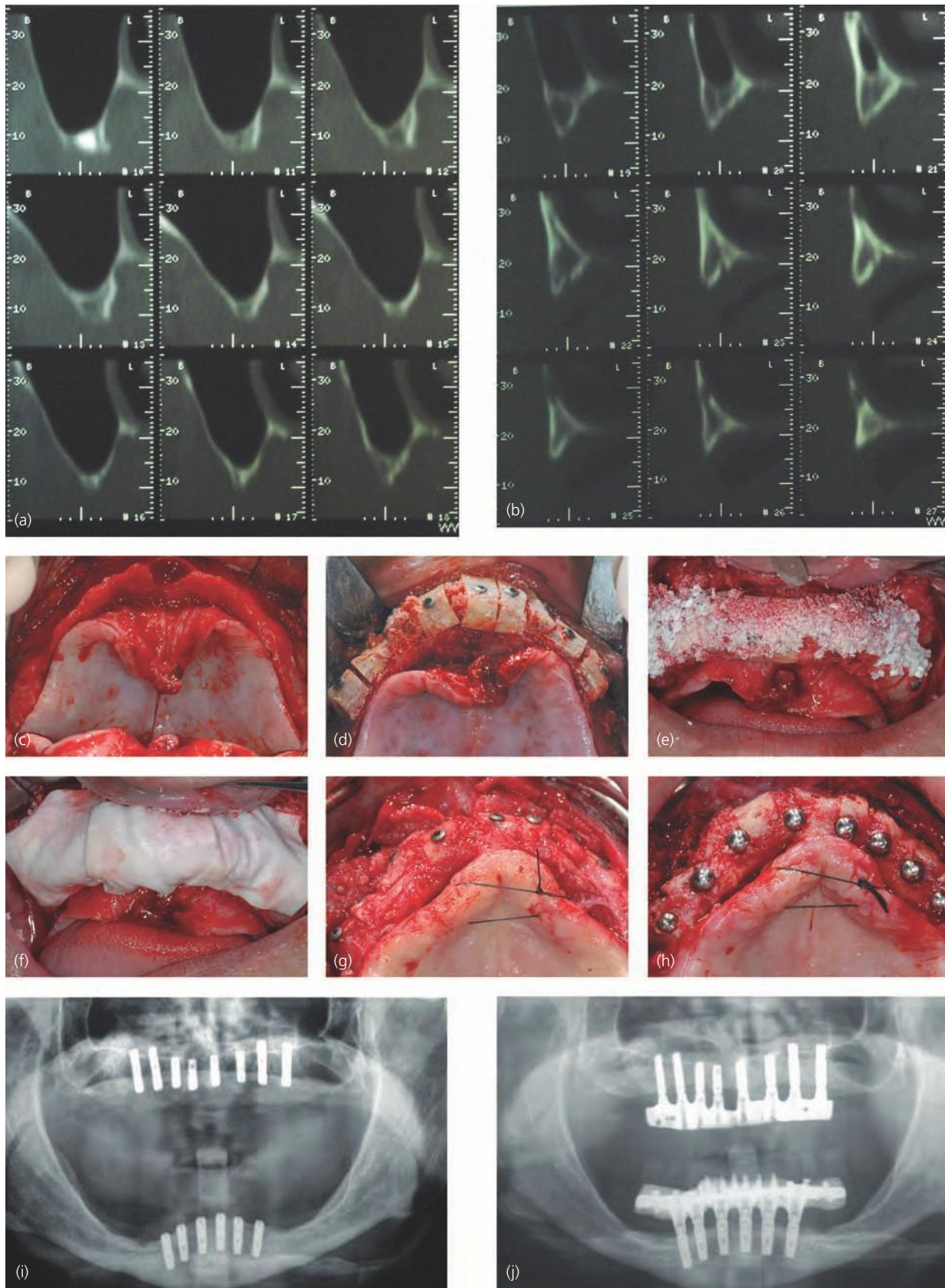


Fig. 26.2 Case 2. (a, b) CT scan view: sinus pneumatization and horizontal bone atrophy can be appreciated. (c) Intraoral view after bilateral sinus lift procedure: before grafting procedure. (d) Autogenous bone block harvested from the hip stabilized to the recipient site by means of cortical screws. (e) Anorganic bovine bone coverage to reduce bone block resorption. (f) Resorbable collagen membrane used to stabilize the anorganic bovine bone. (g) Intraoral view: the new ridge width can be appreciated after removal of cortical screws and before implant placement. (h) Eight implants placed in the newly formed bone. (i) Postoperative panoramic radiograph. (j) 1 year follow-up panoramic radiograph.

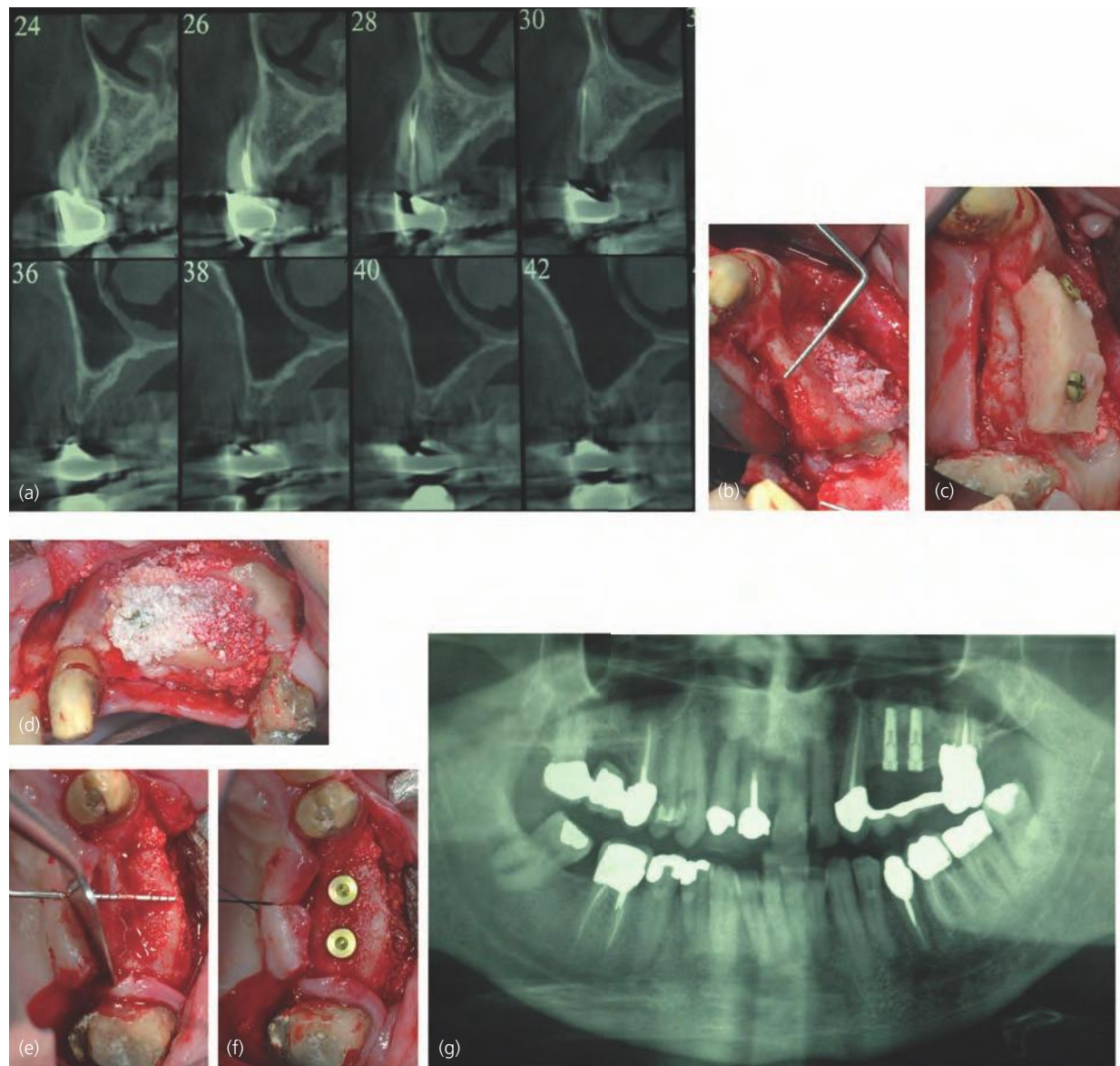


Fig. 26.3 Case 3. (a) CT scan view: sinus pneumatization and horizontal bone atrophy can be appreciated. (b) Intraoral view after sinus lift procedure: a 2 mm wide atrophic ridge can be noticed. (c) Corticocancellous bone block harvested from the chin stabilized by means of cortical screws. (d) Anorganic bovine bone coverage. (e) Intraoral view after 3 months' healing: the newly formed bone can be appreciated. (f) Two implants placed in the augmented edentulous ridge. (g) Postoperative panoramic radiograph.

granule of a biomaterial is similar to a human hydroxyapatite crystal, the more it is possible to get an osteoconductive effect. Another parameter to be taken into consideration is the biomaterial's resorption time. A material which is resorbed too quickly does not allow the osteoblasts and the new vessels to promote formation of woven bone. A material which is resorbed too slowly, by delaying its total substitution with newly formed bone, inhibits bone-implant contact which is essential for osseointegration. A resorption time of 6–10 months can be considered reasonably ideal. In the author's experience, anorganic bovine bone has given excellent results in over 15 years of sinus elevation surgeries, giving a new bone quality close to a class 2 native bone⁴⁴ and a very

good osteoconductive property, as verified from many histomorphometric studies.^{45–47} Nevertheless, the author has been using other bone substitutes, such as beta TCP, calcium sulfate, and DFDBA, whose clinical efficacy has been demonstrated in some studies, although with a lower predictability in terms of bone quality and implant survival rate.

When the height of the residual ridge is 6–7 mm, the surgeon can decide whether to use short implants or elevate the sinus floor 2–3 mm with the Summer's osteotome technique. The procedure, wrongly named "minor sinus elevation" is a blind procedure and should be considered with care. In order to elevate the sinus membrane with this procedure, any of the biomaterials can be equally used, since the primary

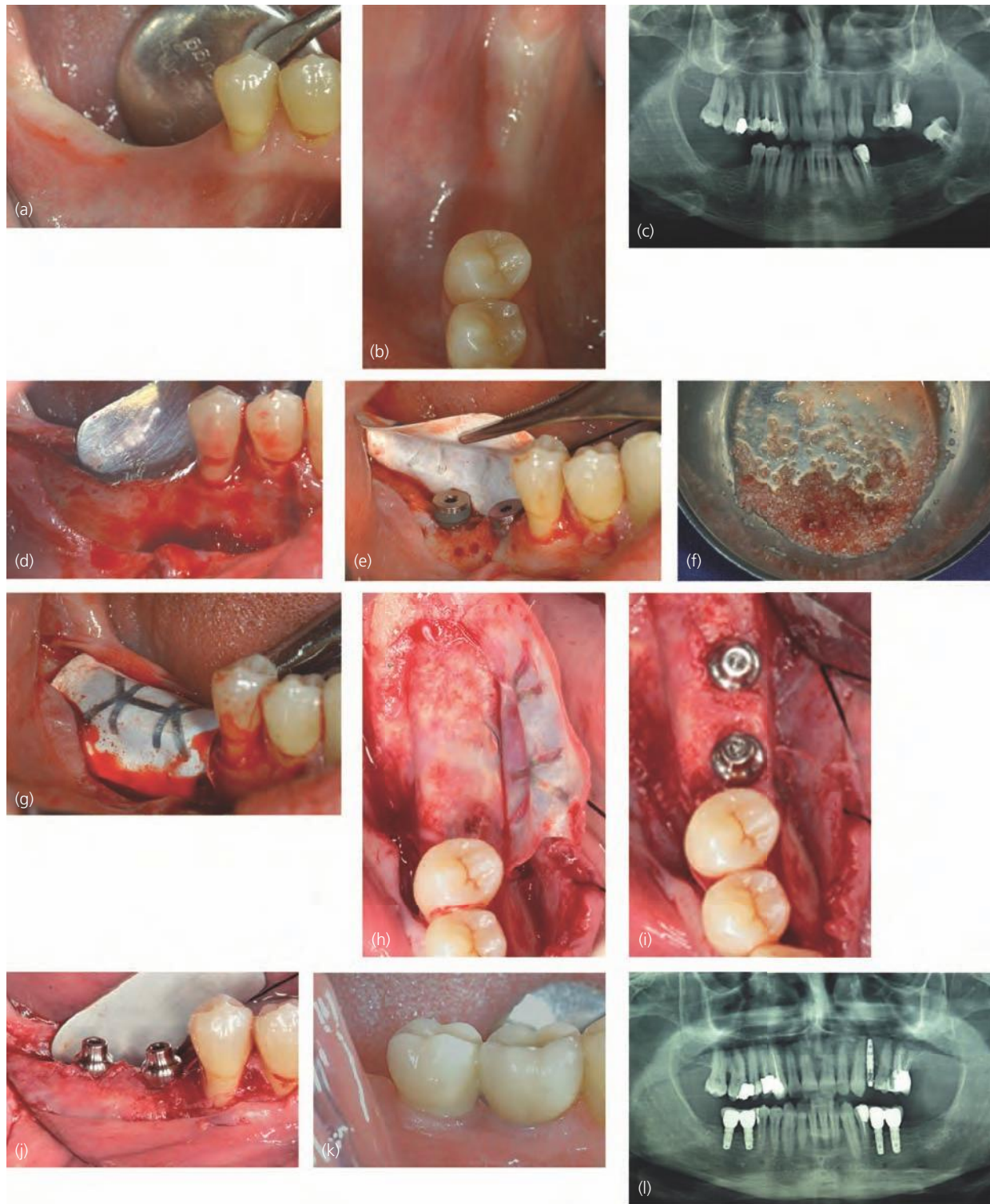


Fig. 26.4 Case 4. (a, b) Intraoral view: a horizontal and vertical defect can be appreciated. (c) Panoramic radiograph: vertical bone resorption can be seen in area 4.6–4.7. (d) Atrophic ridge after flap elevation: vertical bone resorption can be seen. (e) Two implants placed in the ideal position and non-resorbable membrane fixed to the lingual plate to perform vertical bone augmentation. (f) Anorganic bovine bone mixed in 1:1 ratio with autogenous bone harvested from the ramus. (g) Non-resorbable Gore-Tex membrane stabilized after grafting procedure. (h) Newly formed bone after 7 months' healing during membrane removal. (i, j) Healing abutment placed: the horizontal and vertical regeneration can be appreciated. (k) Final restoration. (l) 1 year follow-up panoramic radiograph.

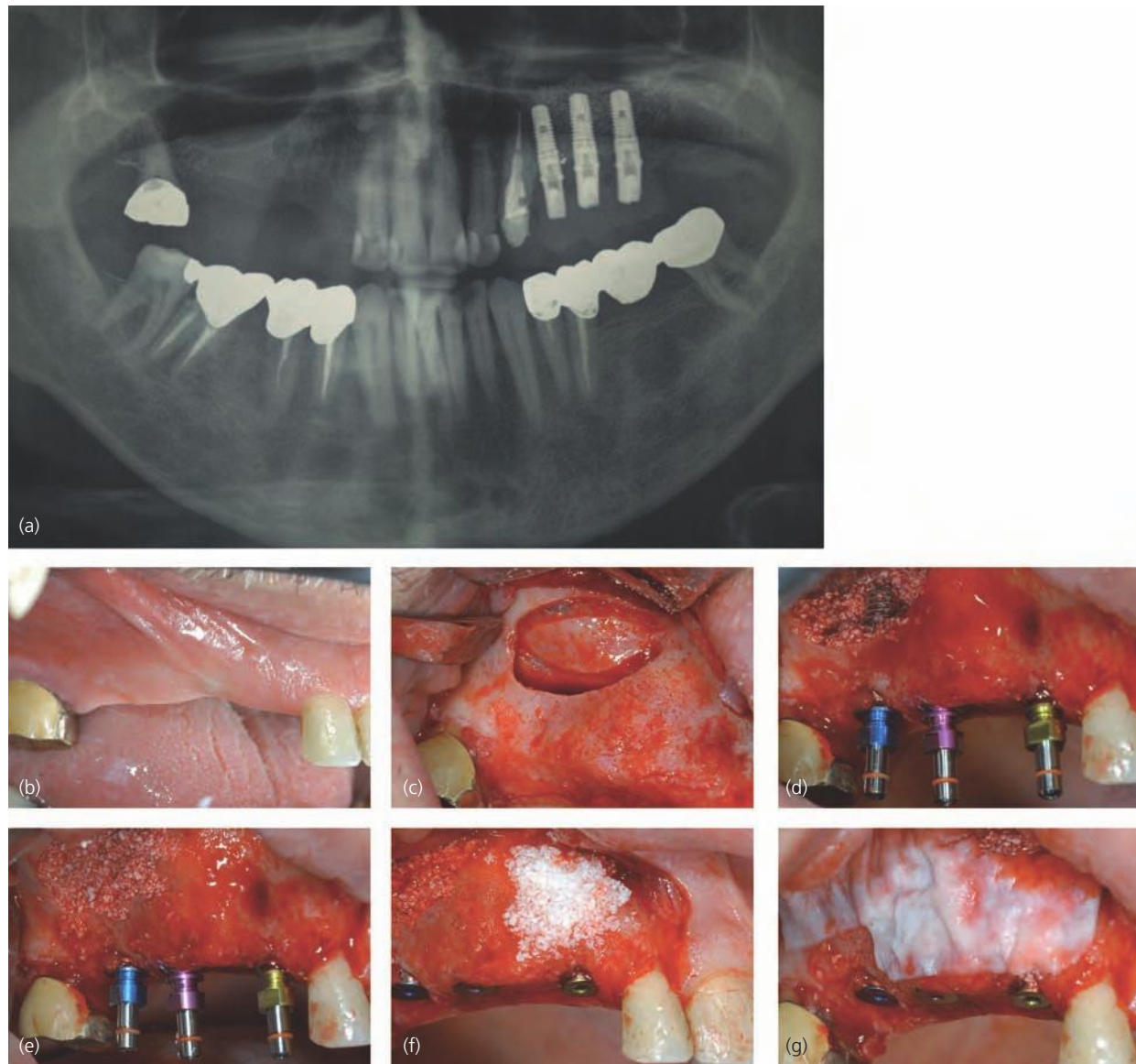


Fig. 26.5 Case 5. (a) Preoperative view: bone resorption in right upper jaw can be seen. (b) Intraoral preoperative view. (c) Sinus lift procedure: lateral approach to lift sinus floor. (d) Implant placement after grafting procedure in the medial portion of the subantral cavity by means of anorganic bovine bone. (e, f) Grafting procedure completed. (g) Resorbable membrane to stabilize the anorganic bovine bone.

stability of the implant is guaranteed by the residual ridge.⁴⁸ See Figs 26.5, 26.6.

Biomaterials in major advanced osseointegration

As described with regard to the intraoral autogenous blocks, biomaterials can be successfully used to reduce the iliac crest blocks with the same coverage technique. The author's clinical experience using anorganic bovine bone as an agent to limit autogenous

bone resorption allows him to conclude that the coverage technique can be routinely used with absolute predictability even in cases of a whole maxillary or mandibular reconstruction.³⁵

Another way to use biomaterials in major reconstructions is the combination of bone substitutes and cancellous iliac bone as described by Boyne in his presentation of the titanium mesh technique for the upper jaw.⁴⁹ The cancellous–anorganic bovine bone mix in a 1:1 ratio is used to fill up a titanium mesh model made in an individual tray (Fig. 26.7). The titanium mesh is secured to the palatal vault and kept in

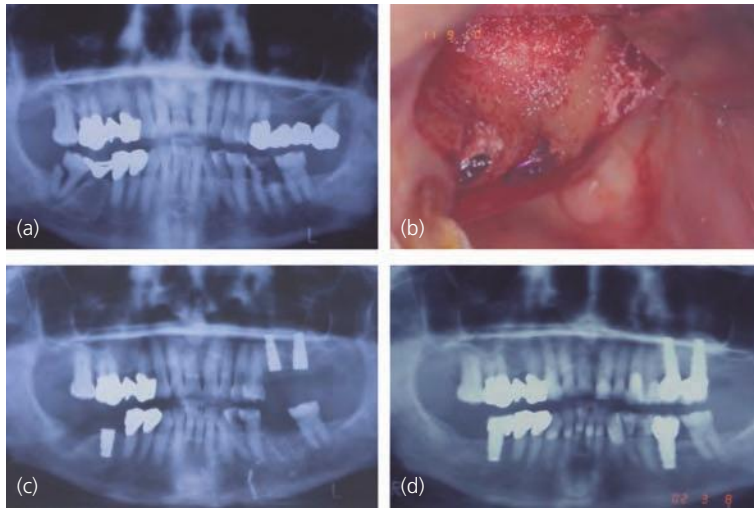


Fig. 26.6 Case 6. (a) Preoperative panoramic radiograph: before teeth extraction in the left upper jaw, 2 months before sinus lift procedure. (b) Sinus lift procedure and implant placement. Hydroxyapatite granules used to fill the subantral cavity can be seen. (c) Postoperative panoramic radiograph. (d) 1 year follow-up panoramic radiograph: no sign of graft resorption or peri-implant bone loss can be seen.

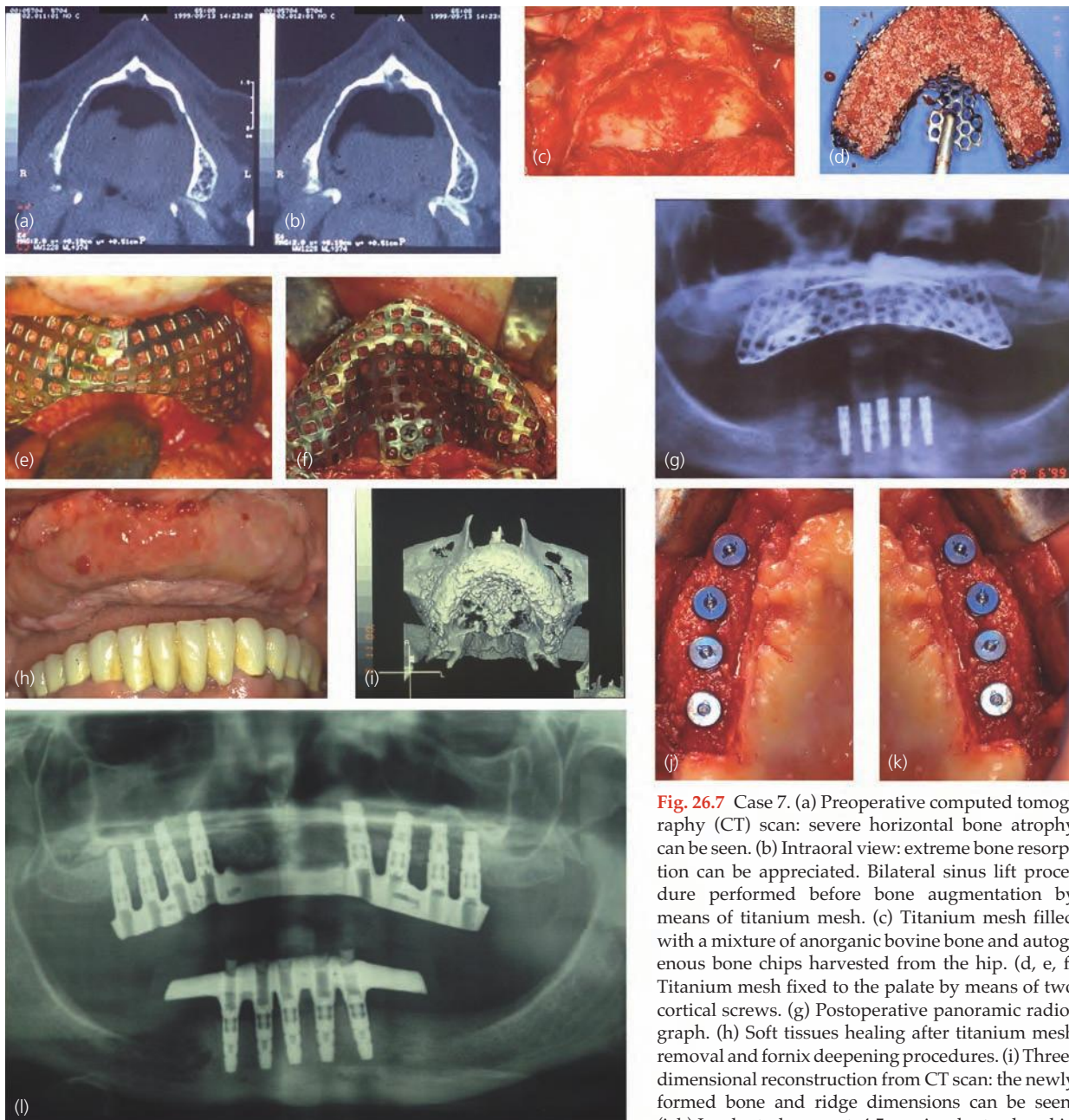


Fig. 26.7 Case 7. (a) Preoperative computed tomography (CT) scan: severe horizontal bone atrophy can be seen. (b) Intraoral view: extreme bone resorption can be appreciated. Bilateral sinus lift procedure performed before bone augmentation by means of titanium mesh. (c) Titanium mesh filled with a mixture of anorganic bovine bone and autogenous bone chips harvested from the hip. (d, e, f) Titanium mesh fixed to the palate by means of two cortical screws. (g) Postoperative panoramic radiograph. (h) Soft tissues healing after titanium mesh removal and fornix deepening procedures. (i) Three-dimensional reconstruction from CT scan: the newly formed bone and ridge dimensions can be seen. (j, k) Implant placement: 4.5 mm implants placed in the augmented alveolar ridge. (l) 5 year panoramic radiograph follow-up.

place for 5 months. The use of such a bone substitute helps to maintain the original volume of the graft during remodeling and contraction of the autogenous part, and so helps to determine increased firmness of the regenerated bone at implant placement time.⁴⁹⁻⁵¹

Developments: growth factors and BMP

A new field in implantology is developing with the aim of finding new ways to improve the osteoconductivity of bone substitutes and to study new molecules able to dictate cellular differentiation and improve bone regeneration. The so-called growth factors are biological mediators which promote cell proliferation. Some of these growth factors have been proved to have the ability to contribute to bone regeneration, PDGF (platelet-derived growth factor) in particular. PDGF is secreted by different cell types, such as platelets, osteoblasts, and activated macrophages,⁵² and is able to stimulate chemotaxis, cell proliferation, and protein synthesis.⁵³ Some clinical studies are currently running on the use of different carriers for PDGF in GBR. PDGF is also one of the factors contained in platelet-rich plasma (PRP), a platelet concentrate produced by plasmaphoresis using centrifugation; this is mixed with autogenous bone chips or bone substitutes in order to add osteoinductive properties to the graft. Different growth factors can be recognized within PRP: transforming growth factor (TGF- β), insulin-like growth factor (IGF), fibroblast growth factors (FGF), and others. The initial technique was presented by Marx, starting from a large blood sample.⁵⁴ Variations were applied in order to simplify the original procedure.⁵⁵ After the first promising results using PRP in different ways, studies confirmed that the activity of PRP is significant only in the presence of autogenous bone, while disappointing results are reported in cases where PRP is used in association with bone substitutes. In fact, no significant differences in terms of vital bone production in sinus elevation with anorganic bovine bone or DFDBA are reported.⁵⁶⁻⁶⁰

The real future in bone regeneration seems to be in connection with the BMPs, currently obtained by synthesis using recombinant DNA. BMPs are part of the TGF- β family but, different to the other growth factors, they have an osteoinductive property. They are exogenic cytokines and act on adult mesenchymal stem cells, which are responsible for all the bone healing processes; their effect, however, depends upon the vector used to deliver the fluid BMP into the site to be regenerated. Different carriers have been studied, such as collagen, polylactic acid granules, anorganic bovine bone, corals, etc., and the ability of BMP-2 and BMP-7 has been investigated.⁶¹⁻⁶³ In the first studies in humans by Boyne,⁶⁴ rhBMP-2 was used in sinus elevation, showing trabecular bone formation after 16 weeks and similar results in terms of

bone regeneration came from Cochran, Howell, and Jung.⁶⁵⁻⁶⁷ Recent data from Boyne⁶⁸ confirm the reliability of rhBMP-2 in sinus elevation when carried by a resorbable collagen sponge. Further data are necessary to confirm the preliminary results, but in the near future the use of BMPs will considerably change the approach and the treatment of maxillary atrophy.

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Chapter 27

The Zygoma Implant for the Totally Atrophied Maxilla

Chantal Malevez

The chapter describes the anatomic characteristics of the zygoma bone. Zygoma implants are described and possible indications for use in implant rehabilitation listed. The surgical technique is described in detail according to a standard protocol. Flapless, guided surgery as well as use of four zygoma implants are other methods included in the chapter. Immediate or delayed loading of the implants is another point of discussion. Zygoma implants have demonstrated a high success rate in the severely atrophied maxilla, however complications that may happen are discussed at the end of the chapter.

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Introduction

The concept of osseointegration provides viable treatment options to patients with partial or complete loss of teeth, and many patients benefit today from predictable oral rehabilitation by means of implants.¹

Patients' motivations for seeking implant-based therapy mostly involve issues of chewing, comfort, self-confidence, and appearance. Edentulousness has always been considered to be a normal effect of ageing, however most people avoid talking about this handicap or explaining how much it affects their professional as well as social life. People suffering total edentulousness restrict their social contact, losing self-confidence and developing fear of discomfort with their removable prosthesis in public, restrict-

ing the choice of the food they are able to chew, and also limiting their speech.

People want to live long and well, free from infirmity, impairment, disability, and handicap; the concept of quality of life has become fundamental in recent years. There is no universally accepted definition for quality of life. Oral health-related quality of life reflects four aspects of oral health: (1) speech and mastication; (2) pain and discomfort; (3) appearance and self-esteem; (4) intimacy, communication, and social relationships. These are of relevance even in maxillectomy cases.² Undoubtedly increasing life expectancy has heightened people's expectations. However edentulousness can occur early in life (20 years). At this age, the removable prosthesis is well tolerated and stability is obtained thanks to the volume of the maxilla and to the presence of retention sites.

It is very common to see patients of less than 60 years of age with a dramatic resorption of the maxilla and sometimes no more than 2 mm height under the nose and the maxillary sinuses. In terms of prosthetic reconstruction, fixed or removable, this kind of situation is no different from edentulousness at the age of 70 or 80: the handicap remains the same (no stability, sourness, difficulty of chewing, ageing face) but it appears earlier in life. Ageing depends on inherited factors, environmental factors, and personally controlled factors.

In many studies, the mean age of the patients treated by means of zygoma implants is 58 years^{3,4} confirming the distress of these patients. Most of the time, at this age, patients are still involved in professional life as well as in a social one. Their well-being is fundamental but, despite the fact that they are handicapped, they have no time to spend in lengthy treatments.

Treatment of the atrophic upper jaw depends on the severity of the atrophy and also on the patient's systemic condition, age and health, and on other functional parameters like interarch ratio, oral hygiene, and phonetic aspects. Regarding esthetic requirements, patients have to understand the advantages

and limitations of different types of prosthetic rehabilitation in terms of comfort, compensation for the lack of volume, and adequate lip support. Fixed rehabilitation by means of bridges seems to be the treatment of choice in young and middle-aged patients. Some clinicians consider that removable prostheses are adequate for older patients. However, ageing, with decline of muscular coordination, force, modification of eating habits, and compromised oral hygiene, increases the need for a fixed denture situation. There is no objective reason to provide older patients with removable dentures.

When the clinician takes time to try to understand the wishes of the patient, he or she will discover that younger people are more concerned with esthetics than older patients, but that correct chewing and easy cleaning are mandatory for older patients. However old patients do want to be good looking and avoid an ageing face due to the absence of lip support. They ask for esthetic results too, and they agree that artificial teeth provide them this lip support.

It is well known that providing the patient with a removable prosthesis is not satisfactory even if some studies try to prove that there is no significant difference between a removable prosthesis and a prosthesis fixed on implants. Providing standard implants in cases of relatively minimal bone resorption, which allow the construction of fixed bridges, considerably improves quality of life and patients recover self-esteem and confidence. They regularly say, "My life has changed." They have found a solution to all their complaints.

For many years, it was recommended to wait 6 months before loading the implants to obtain osseointegration; today the development of new surfaces, new implant designs, and modified surgical protocols has improved the quality or the primary stability⁵ leading to a final goal: immediate loading of the implants, rebuilding the occlusion, function, and esthetic lip support in one stage. This new approach completely changes the length and the philosophy of treatment.

For a normal rehabilitation of the totally edentulous upper jaw, standard implants are usually placed in the anterior part of the maxilla allowing the placement of four to six implants supporting a fixed bridge of 12–14 teeth.⁶ Bone loss is compensated for by the prosthesis with the use of longer teeth or a flange of pink acrylic simulating papillae and soft tissue. Tilted implants⁷ placed in the anterior maxilla can also be indicated in cases of deep maxillary sinus extensions, implants being placed tangential to the sinus borders. They are as long as possible, the implant entry point being at the level of the second premolar and the upper anchorage in the canine pillar.

Unfortunately loss of teeth is followed by loss of bone and lack of bone volume in the anterior part as well as in the posterior section of the maxilla. This situation can be a real problem for the rehabilitation of the patients either by means of removable prosthe-

sis or by means of implants. The amount of bone remaining under the sinus as well as under the nasal fossa can be as little as 1 mm. Patients who are completely edentulous at the age of 20 have to face this atrophy from the age of 40, so, consequently, this situation is very common in patients of 56–57 years of age.

Patients with a mobile removable denture develop anxiety and fear of losing their prosthesis at work as well as while socializing, during intimacy and contact with friends. In these cases with severe bone resorption, implant placement is impossible without additional techniques.

Some 20 years ago, the first solutions were bone volume augmentation with the hope of restoring the maxilla to how it was in the past.^{8,9} Many techniques have been proposed with different success rates: sinus grafts,^{10–12} onlay-inlay bone grafting, osteotomies. They all have the aim of augmenting the bone volume with different kinds of materials: autologous bone grafts, bovine bone grafts or artificial materials.^{9,13} Despite the fact that these treatments are valuable and well documented, they are lengthy, sometimes unpredictable, and cause discomfort for the patients depending on which kind of grafted material is used.

The zygoma implant (zygoma implant, zygomaticus fixture) was introduced by PI Brånemark in the early 1980s¹⁴ for severely atrophied maxillae without discontinuity, after his successful experience in the rehabilitation of defects of the upper jaw,¹⁵ where he imagined using the zygoma as an anchorage for oral implants.

Since 1997, the zygoma implant (the so-called zygomaticus fixture) has been developed throughout the world for totally edentulous patients; it was also later developed for partially edentulous cases. The zygoma implant was initially proposed as an alternative to sinus grafts, reducing morbidity, length of the treatment, discomfort, and improving quality of life of the patients. With time, survival rates appeared to be satisfactory.^{16–23} Moreover, as has been suggested for treatment in the lower jaw with the Brånemark Novum,²⁴ it is today possible to load standard implants^{25,26} and zygoma implants immediately, restoring chewing function, speech, and esthetics on the day of the surgery.^{27–29} The surgery is a safe procedure but it is necessary to have good knowledge of anatomy and to follow the guidelines proposed in the protocol.

The zygoma

The question: Can the zygoma be a satisfactory anchorage for oral implants?³⁰ The zygoma bone is attached to the frontal bone, the sphenoid, the temporal bone, and the maxilla, this last through the zygomatic apophysis. It looks like a pyramid with four faces and an inferior top (Fig. 27.1).



Fig. 27.1 View of the bulky form of the zygoma.

Features of the zygoma bone

Bone quality

In a study on the rabbit, Gosain showed the zygoma was a regular trabecular bone with high density.³¹ In a study on the human zygoma, Nkenke showed the zygoma had quite poor trabecular density but very strong cortical bone that could ensure primary stability of implants inserted in its cortex (Fig. 27.2).³²

Length and volume

In a study on 14 cadavers and clinical cases realized in February 2001, it was shown that the mean height of the zygoma is 14mm and the anterior-posterior width is 20.5mm.³³ Trabecular bone is found all along the total height of the zygoma bone and cortical bone is present at two levels in the lower part of the zygoma as well as in the upper part.³⁴ From the anatomic point of view, it can be concluded that the zygoma offers an anchorage that, according to Brånemark's definition, would probably provide osseointegration for a long period of time with long-term results.

Anchorage

The zygoma is a well known multidisciplinary anchorage:

1. In orthodontics: with the use of perizygomatic wire³⁵ placed around the zygomatic arch or mini-

- plates³⁶ screwed on the zygoma to obtain retraction of the incisors by progressive traction.
2. In maxillofacial prosthetics: the zygoma is used as an anchorage for extraoral transcutaneous implants supporting orbital prostheses.¹
3. In maxillofacial surgery: miniplates are used for repair of fractures and the zygoma provides a solid anchorage for the miniplates.³⁷
4. In research: in an experimental study on the monkey, the resistance of titanium implants inserted in the zygoma and submitted to heavy traction for mobilizing the maxillofacial complex after a Le Fort III osteotomy was shown.³⁸
5. In reconstructive surgery: PI Brånemark used the zygoma bone as an anchorage for maxillofacial prosthesis after tumor resection.³⁹

From this multidisciplinary experience, it is concluded that the zygoma bone resists different types of forces applied to it.

The zygoma implant

Extreme atrophy of the posterior maxilla makes it impossible to insert implants without augmenting the bone volume except if an anchorage can be found at a level other than the maxilla, i.e. the zygoma.^{22,34,40} Alveolar extension of the maxillary sinuses also reduces the length of the arch where regular standard implants could be inserted. With bone graft failures, bone volume is also reduced diminishing the possibility of using regular standard implants. Another issue is the rehabilitation of maxillectomized patients after tumor resection.

As seen previously, the zygoma offers a strong and stable anchorage^{34,41} at a level other than the maxilla. Two zygoma implants can be used in conjunction with two to four anterior standard implants when sufficient bone remains in the anterior part of the maxilla. In these cases, minimal bone height of the premaxilla should be 10mm and minimal width

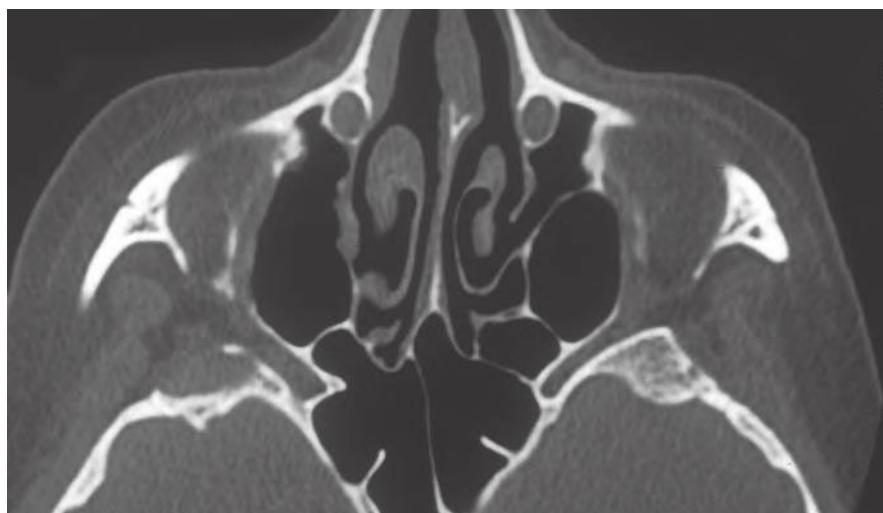


Fig. 27.2 Thick cortical bone and spongy trabecular bone.

4 mm. The zygoma implant has its emergence point at the level of the second premolar/first molar. Depending on the anatomy of the patient, the emergence point will be crestal or slightly palatal or slightly buccal.⁴² The zygoma implant has to be connected to other implants to support the load of the masticatory forces.⁵

Immediate loading avoids the need to wait for a long period before allowing regular masticatory function by means of a fixed prosthesis screwed on to the implants. Immediate rigid splinting of the tilted implants distributes the axial and lateral loads and stabilizes the whole system.

In extreme resorption of the maxilla (1 mm height) the majority of authors usually recommend large autogenous bone grafts. These reconstructions can offer a solution, but the use of four zygoma implants would seem to be the treatment of choice.²⁶ As described before, the volume of the zygoma is sufficient for the insertion of two zygoma implants with great stability, providing good support to a fixed prosthesis immediately screwed on to them.

Description

The zygoma implant is a self-tapping angulated implant (Nobel Biocare AB) made of cp titanium with two surfaces, one machined and one Tiunite,⁴³ and presenting two diameters on the same implant, 3.9 mm at the top and 4.6 mm at the maxillary level. It exists in different lengths: 30, 35, 40, 42.5, 45, 47.5, 50, and 52.5 mm. The 3.9 mm part corresponds to the mean volume of the zygoma and 4.6 mm to the width of the maxilla at the level of the second premolar/first molar.

The interesting 45° angulation corresponds to the angulation of the zygoma bone with the maxilla.



Fig. 27.3 The Tiunite zygoma implant and the machined implant with their fixture mount.



Fig. 27.4 The angulated edge for the maxillary level.

Some authors propose zygoma implants with 55° angulation.⁴⁴ In cases of extreme atrophy, this angulation can be corrected by additional angulated abutments (17° and 30°). The roughness of the Tiunite surface augments the mechanical interlocking at the level of the zygoma, allowing immediate loading and immediate function (Figs 27.3, 27.4).

Indications and aims

The zygoma implant is indicated in:

- situations where the anterior volume of the maxilla is still sufficient for the placement of two to four standard implants but where the posterior maxillary crest is too atrophied and would need bone grafting;
- situations where the atrophy of the maxilla is extreme, but the zygomas allow the insertion of four zygoma implants.^{26,45}

The aims of the zygoma implant are:

- to ensure a posterior stable anchorage in difficult conditions: type IV bone and heavy atrophy (more than 7 mm);
- to avoid heavy bone grafting;
- to offer an alternative to sinus grafts;
- to offer fixed bridges to patients handicapped by mobile removable prostheses;
- to rehabilitate patients who have undergone resections of the maxillae.

Radiological examination

In the dental field, different kinds of imaging techniques are available: orthopantomogram, profile radiographs, scanora, computed tomography (CT) scan, cone-beam CT, including software allowing virtual planning. Scanora, CT scan, and, more recently, cone-beam CT are the ideal radiological approaches for the preplanning of the surgery. These imaging techniques enable one to:

- appreciate the sinus health and the possibility of ventilation by investigating the whole maxillary sinus cavity – sinus pathology like chronic sinusitis, papillomas, and aspergillosis can be detected;
- determine the topography of the anterior wall of the posterior temporal fossa (posterior wall of the sinus and the zygoma);
- estimate the volume of the maxillary bone;
- estimate the concavity of the anterior sinus wall.

Dedicated software^{33,46} also provides the possibility of performing virtual insertion of the zygoma implant before the surgery and planning surgical guides allowing flapless surgery, immediate loading, and immediate function (Fig. 27.5).

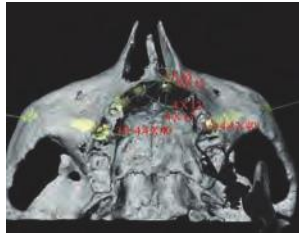


Fig. 27.5 Occlusal view of the zygoma with virtual insertion of the zygoma implant and of the other standard implants. The yellow color shows the security zone around the implant.

Surgery

Since 1997, when a protocol was first established for the insertion of zygoma implants, some modifications have been proposed by different clinicians. Despite the fact that the anatomy of the zygoma limits the orientation of the zygoma implants, the curve of the anterior wall of the sinus has also been considered to be a limiting factor. The more concave the sinus wall is, the more likely it is that the implant will have a palatal emergence point, that could jeopardize the

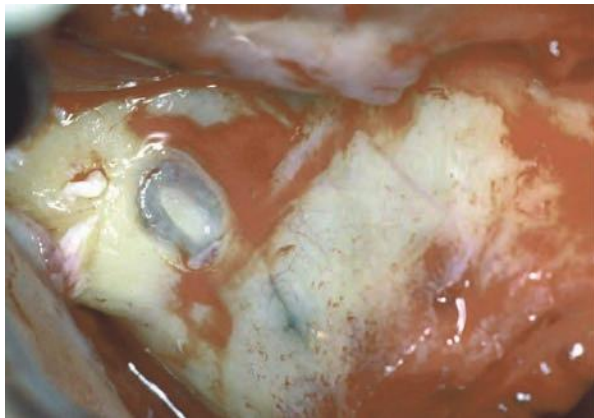


Fig. 27.6 After the flap is raised, opening of a small window.

prosthetic rehabilitation, encountering high cantilevered bridges. For these reasons, some authors proposed an extrasinusal insertion of the implants and a buccal position at the level of the maxilla.¹⁹ Mouth opening is also a factor limiting the possibilities of the zygoma implant insertion, because of the length of the drills and of the zygoma implants.

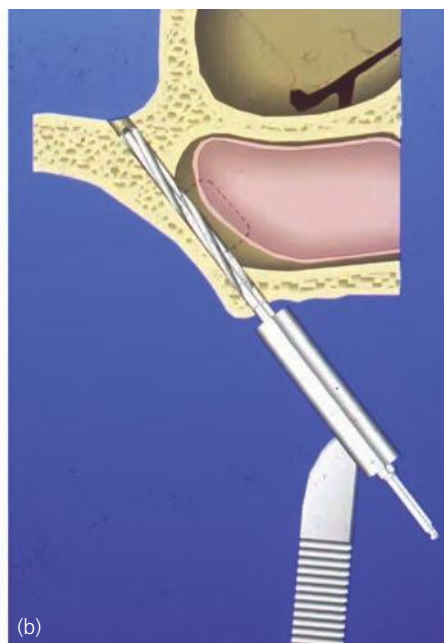
Conventionally following the classic protocol, the placement of zygoma implants can be performed as a day case or chairside under local or total anesthesia. The incision is sulcular (Le Fort I incision) or more often crestal or slightly palatal, with elevation of a full-thickness flap. The advantage of doing a palatal incision is to keep as much attached soft tissue as possible for covering the head of the implant after surgery.^{18,47} The flap is raised up to the zygoma, observing the suborbital nerve, the rim of the orbit, and the angle made by the zygomatic arch together with the zygoma and the zygomatic apophysis of the maxilla.^{17,46} A small window is opened in the anterior sinus wall at the level of the zygoma (Fig. 27.6).

This small window close to the level of the zygoma gives the surgeon the opportunity of a direct view of the whole zygoma and allows him/her to choose the orientation of the drilling procedure and of the insertion of the implant. A first round bur is then used for entering the maxilla and the zygoma and is followed by a sequence of three drills that will provide the bed of the zygoma implant (Fig. 27.7). The implant is then inserted up to the top of the zygoma with a low speed motor and stabilized with a special screwdriver ensuring the position of the implant perpendicular to the occlusal plane for prosthetic purposes (Fig. 27.8). Other standard implants (two to four) are then inserted in the anterior region (Fig. 27.9).

Where four zygoma implants are to be inserted, the anterior ones are positioned first. For this procedure, the window is a horizontal one that allows the



(a)



(b)

Fig. 27.7 (a) Drills for preparing the bed of the zygoma implant. (b) Drilling starts on the crest.

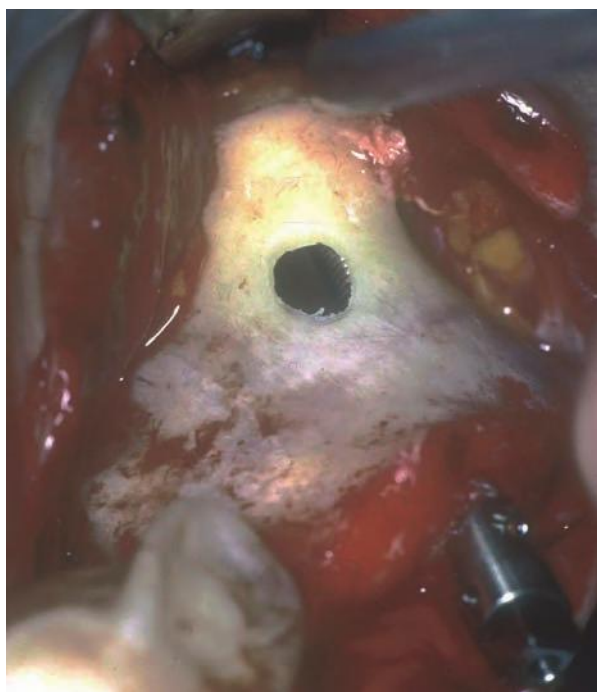


Fig. 27.8 Insertion of the zygoma implant. The head of the implant is seen at the top of the zygoma and the implant is seen through the window.



Fig. 27.9 Placement of four additional standard implants.

surgeon to have a view of the insertion of both implants (Fig. 27.10). The anterior one is positioned first after having detected the suborbital nerve. Its maxillary emergence point is at the level of the lateral incisor. The second implant, with its emergence point at the second premolar/first molar, is then inserted (Figs 27.11–27.13). The distance between both implants at the zygoma level should be at least 1 cm, for providing sufficient bone support and osseointegration.

Modifications of the surgical protocol have been proposed by different authors: sinus slot technique with a vertical window along the maxillary zygomatic apophysis,⁴⁸ vertical window pushing back bone and sinus membrane before drilling and insertion of the zygomatic implant,²⁸ with a technique comparable to the sinus graft technique, soft tissue punch at the level of the palatal mucosa combined with a Le Fort I incision,⁴² guided surgery for drilling and visual control of the insertion of the implant.²⁸

Insertion of the zygoma implant will depend on the anatomy of the anterior wall of the sinus and the

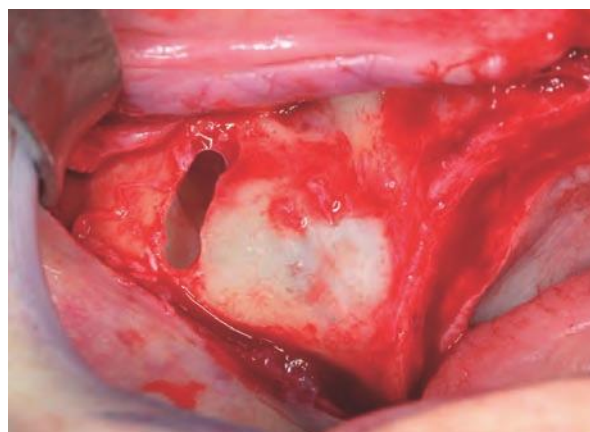


Fig. 27.10 Window where four zygoma implants are to be placed. Case realized in collaboration with R. Haas and G. Mailath-Pokorny.

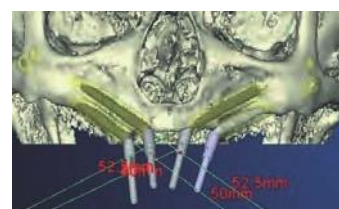


Fig. 27.11 Simulation and preplanning.

bulky zygoma bone. Where the concavity of the sinus wall is deep, the zygoma implant inserted totally through the sinus would have a palatal emergence point. To avoid the implant being too palatal, it is possible to insert the implant parallel and in close contact with the sinus wall. In this situation, the implant remains external to the sinus membrane, and implants will only be anchored in the zygoma, with a close contact with the maxillary crest for obtaining a crestal position of the implant.⁴² Osseointegration will only be obtained at the level of the zygoma. From a biomechanical point of view, it is well known that masticatory forces applied to the prosthesis screwed on the implants are transmitted to the bony anchorage at the level of the zygoma. That means that osseointegration is not required at the level of the atrophied maxilla. With this insertion protocol, the sinus membrane is not damaged, and the zygoma implant remains totally outside the sinus. A more crestal position of the implants is also favorable for prosthetic reconstruction.

Flapless guided surgery (Nobel Biocare AB) is also being developed to minimize surgical degloving of the soft tissue and to improve the postoperative condition of the patient, with less swelling, less pain, no sutures, and the possibility of immediate loading.

Complications

Complications are few. Some authors⁴⁹ seem to have found deep pathological pockets around the neck of

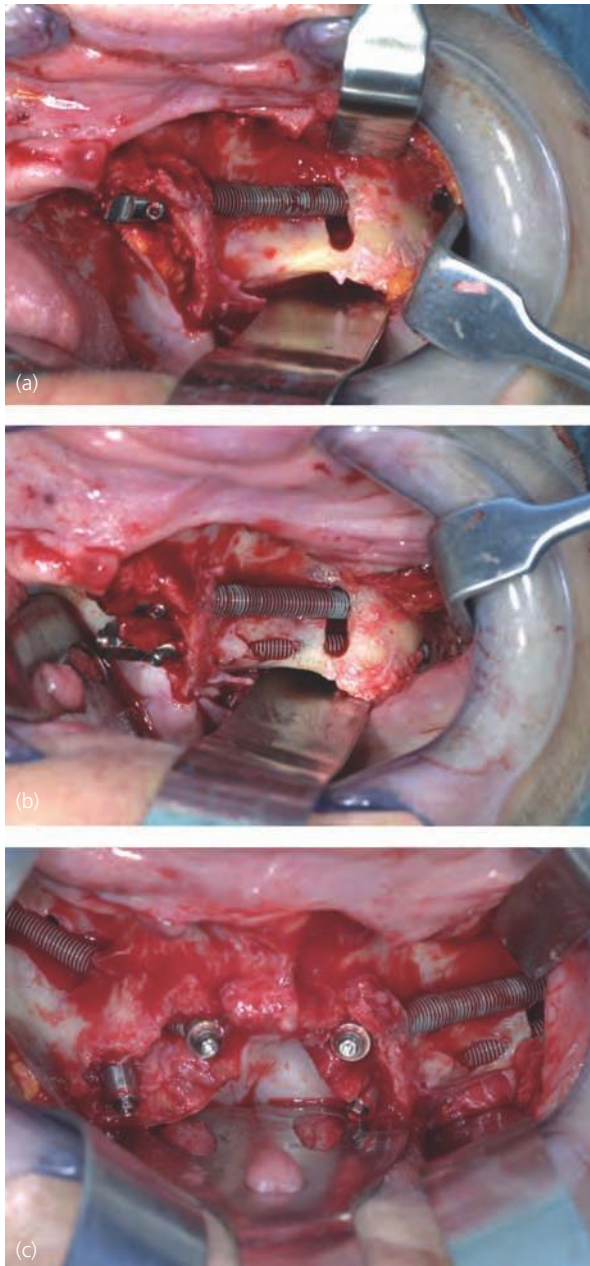


Fig. 27.12 Case of four zygoma implants and extreme atrophy of the maxilla. Preplanning and surgery. (a) Placement of the first left implant. (b) Placement of the second left implant. (c) View of the four implants.



Fig. 27.13 Placement of a provisional prosthesis screwed on the four implants the same day. Case realized in collaboration with V. Choquet and T. Thomas.

the zygoma implants. These “pockets” are due to the fact that the zygoma implant emerges slightly palatal at a level where the healthy soft tissue containing fat cells can be 5–6 mm thick. Controlled hygiene avoids inflammation at this level and keratinized epithelium ensures a seal around the implants.

Most of the time, zygoma implants penetrate the sinus. This results in two risks: the first is the opening of the floor of the sinus so that, at the time of the surgery, it communicates with the mouth, and the opening of the zygoma at the top of the sinus. The second risk is the insertion of a foreign body⁵⁰ that could result in inflammatory reactions. Some authors have described 2–4% sinus inflammatory reactions or more.^{3,22,51}

Symptoms of sinusitis are swelling of the midface and suppuration from the nose. Microbiological tests and endoscopy can confirm the diagnosis. For resolution of the inflammation, control of the infection with reduction of swelling is necessary to correct ventilation and sinuses drainage.⁵² Antibiosis, corticosteroids, and local treatment can be proposed initially,⁵² before considering surgical treatment like meatotomies.

One hypothesis of the cause of sinusitis is the fact that the zygoma implants generate chronic inflammation of the sinus that diminishes sinus drainage and increases the amount of bacteria.

Another hypothesis is the creation of a communication between sinus and mouth due to the possible flexibility of the zygoma implant and a weak point at the maxillary bony level. Bacteria from the mouth could enter the sinus by this route. In the standard machined zygoma implants, there is a perforation of the head of the implant which houses the abutment screw, and this could facilitate the migration of bacteria.

Most of the studies show a maximum of 5% sinusitis. One study on a small number of patients describes nine patients out of 16 who suffered from sinusitis, but in this study poor hygiene as well as inflammation of the emergence point of the implants were implicated.⁵¹ The percentage of infections is comparable to the percentage of infections in sinus graft procedures.⁵⁰ Nevertheless, endoscopies also show very healthy sinuses.⁵³

Sinusitis involving osteitis of the zygoma has not been described. In cases where the integrated zygoma implant had to be eliminated because of recurrent sinusitis, it was impossible to remove it and it had to be cut at the level of the zygoma.

Since the development of this zygoma implant protocol in 1997, a number of modifications have been developed to improve the whole procedure:

- Better evaluation of the sinus health can be done with examination of the sinus meatus by CT scan before performing the surgery.
- With the new protocols of immediate loading, there is a single one-stage surgery without reopen-

ing of the soft tissue and an immediate stabilization of the whole rehabilitating system.

- The implant with the Tiunite surface does not have a perforation.
- Surgery can also be performed without entering the sinus, as described above.

All these modifications could lead to a diminished number of cases of sinusitis.

Other complications, e.g. acute sinusitis, hematoma, and transitional hypoesthesias of the suborbital nerve, have also been described. Fracture of implant is very uncommon and has so far been described by PI Brånemark in one case. Failure of osseointegration has been reported with results up to 9%.¹⁷ The palatal emergence site of the machined and Tiunite zygoma implant has been histologically analyzed, showing particularly healthy sites (Figs 27.14–27.16). The epithelium is a short, keratinized or parakeratinized epithelium very similar to the keratinized palatal epithelium. The lamina propria, i.e. the connective tissue underlying the oral mucosa, contains thick collagen

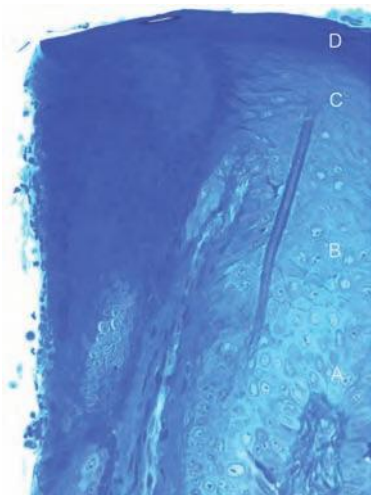


Fig. 27.14 Keratinized palatal epithelium.

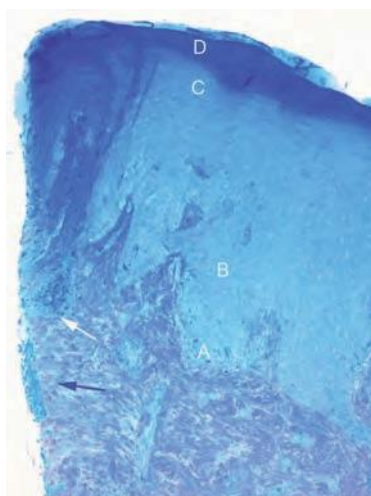


Fig. 27.15 Thick palatal mucosa with: A, stratum germinativum; B, stratum spinosum; C, stratum granulosum; D, the stratum corneum.

bundles oriented towards the implant surface. No accumulation of bacteria or inflammatory response was visible at the interface.

Functional rehabilitation

As for the standard rehabilitation on standard implants, there are three possibilities available to the clinician:

- immediate loading and immediate function,^{5,24,25,29,54}
- early loading and immediate function with provisional prosthesis;^{25,29}
- two-stage surgery and delayed loading.

Immediate loading can be performed with a permanent prosthesis or a provisional one.^{27,28} To allow immediate loading, a complete removable prosthesis is prepared before the surgery in the usual way: occlusion, interarch relationship, vertical dimension, and masticatory conditions are evaluated. A surgical guide in clear acrylic can also be made to give more information for placing the implants in the ideal position.²⁶

The patient's immediate rehabilitation is performed exactly the same way as for standard implants. Immediately after the surgery, an impression is taken of the implants by means of transfers screwed on the abutments. The provisional prosthesis prepared before the surgery is attached to the transfers with an acrylic template, and a low-viscosity, silicon-type impression material is injected to copy as many details as possible. The prosthesis is then treated in the laboratory where it can be reinforced by carbon fibers or a metallic framework. The prosthesis is then inserted in the mouth of the patient and screwed on the implants within 24 hours and the occlusion is adjusted.

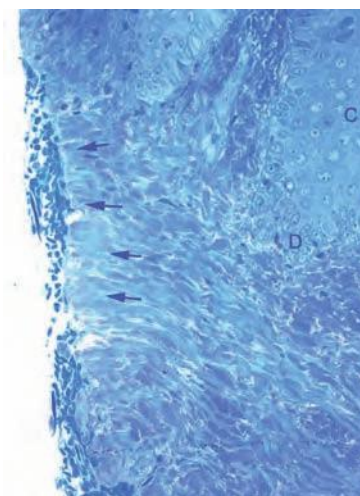


Fig. 27.16 Collagen fiber bundles in a case of zygoma quads oriented towards the implant surface. Courtesy of P. Schupbach.

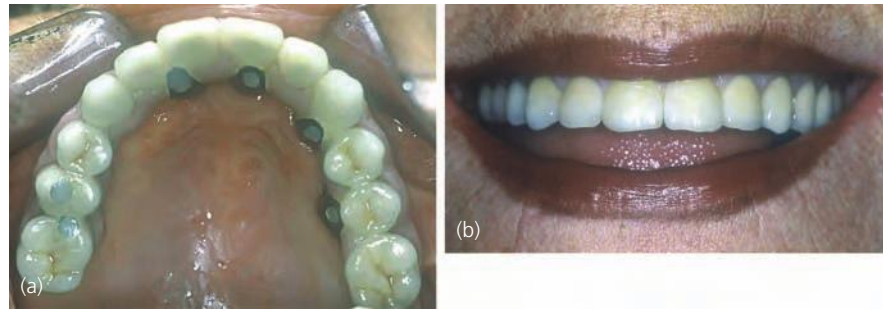


Fig. 27.17 (a) Final prosthesis made of gold and porcelain on two zygoma implants and three standard implants. (b) Smile of the patient showing good lip support.

Most of the time, fixed bridges are screwed on the implants to offer the patient comfort and precise application of occlusal forces. Some clinicians prefer to provide a bar system and removable prosthesis, considering this procedure more adequate for hygiene especially in old patients. However practitioners should remember that most of the studies show a mean age of 56–58 years, an age at which patients would probably prefer fixed bridges.

After 3 months, definitive prostheses are constructed with gold or titanium or zirconium frameworks and acrylic or ceramic teeth to ensure cosmetic results for the patient and compensate for the lack of volume due to the atrophy (Figs 27.17, 27.18).

Guided and minimally invasive surgery

Dedicated software provides the possibility of producing the provisional or definitive prosthesis before the surgery and screwing it on immediately, to allow immediate function.^{25,46} Virtual positioning of the implant is performed before the surgery and a surgical guide is fabricated following the NobelGuide procedure. At the time of the surgery, the guide is inserted in the mouth and fixed to the maxilla with several pins (Figs 27.19–27.22). Drilling is performed through the guide as well as the zygoma implant insertion by means of special hardware that places the zygoma implant in its exact position. The definitive prosthesis is screwed on the implants.



Fig. 27.18 Titanium framework on four zygoma implants.

The advantages of this technique are:

- Reduction of time for the surgery and the prosthetics. The patient encounters less swelling and pain that limits discomfort.
- Restoration of chewing function same day with a fixed prosthesis that gives the patient a feeling of security and self-esteem improving quality of life.
- Reduction of costs, limiting chairside treatment to a minimum.

This procedure is still in development, and will benefit from more scientific research for validating the surgical guide and the flapless procedure.

Conclusions

Zygoma implants have demonstrated a high survival rate in severely atrophied maxillae, with minimal complications. This alternative to sinus grafting and other kinds of bone volume augmentation shortens the surgical procedure, reduces costs, diminishes the length of the rehabilitation, and there is the possibility of immediate loading and function, improving quality of life for the patient.

Clinicians should be aware of the fact that edentulousness can start very early in life, affecting chewing function, speech, esthetics, well-being, and self-esteem.

Whatever the surgical approach is, crestal or paracrestal, slightly buccal or palatal, the point of

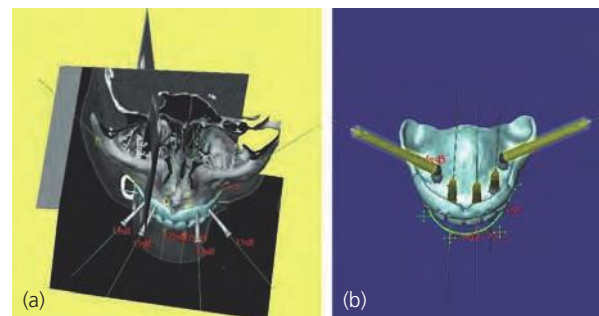


Fig. 27.19 Virtual simulation before guided surgery. (a) Pre-planning in three dimensions. (b) A view of the radiological guide with the implants.

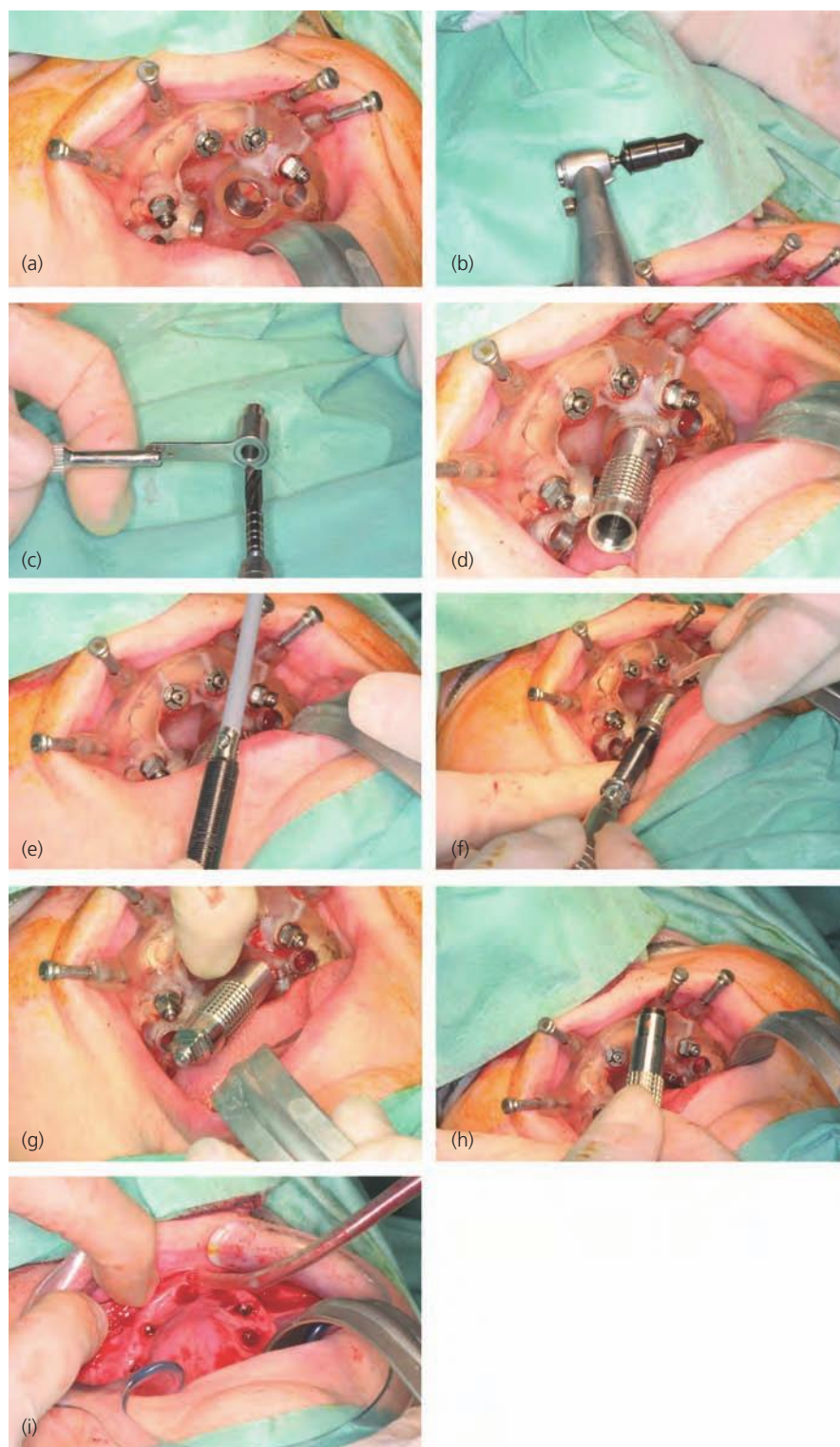


Fig. 27.20 Sequences of the guided surgery. (a) Positioning of the Nobelguide after CAD/CAM preplanning and Teeth in an Hour[®] protocol. (b) Use of the punching drill for removing the soft tissue. (c) Use of sleeves for drilling. Sleeves are inserted in the zygoma implant guide. (d) Insertion of the zygoma implant guide. (e) The Tiunite zygoma implant is screwed on a special fixture mount. (f) The implant is inserted in the guide. (g) The implant is in place. (h) The zygomatic guide is removed. (i) The surgical guide is removed.

emergence of the zygoma implants is usually at the level of the second premolar/first molar. This position allows the use of cantilevered fixed bridges, up to the first or second molar, reconstructing a full arch for functional and esthetic purposes.

Where four zygoma implants are used, the anterior ones emerge at the level of the canine/lateral incisor

and the posterior ones at the level of the second premolar. The use of four zygoma implants increases the strength of the anchorage and offers a distribution of the masticatory load in the zygoma, a bony structure of excellent quality.

Strict hygiene is mandatory to avoid inflammation of the soft tissues around the emergence point of the

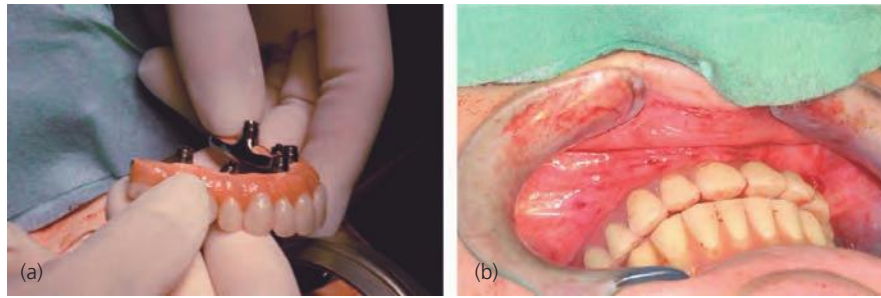


Fig. 27.21 Prosthesis is prepared before surgery and inserted immediately. (a) Procera® implant bridge (PIB) with the adjustable abutments in place. (b) Prosthesis screwed on the implants.



Fig. 27.22 Profile showing good lip support.

implants that could create possible fistulas. However, in cases where inflammation was observed, it never compromised the osseointegration of the zygoma implants.

More studies, especially on partial edentulousness, are needed to validate this procedure and give another alternative to patients. Development of new technologies like guided surgery should be validated to diminish the risks and possible complications of these protocols and establish guidelines for the clinicians. Modifications of surgical protocols as proposed by the author should diminish complications like sinusitis that jeopardize the treatment.

Zygoma implants have yet to find their place in the routine treatment of patients suffering from extreme atrophy of the maxilla that jeopardizes their professional and social life.

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Chapter 28

The Role of Implants in Maxillofacial Reconstruction

Arun B. Sharma and John Beumer III

The chapter is a comprehensive overview of the role of implants in the reconstruction of major defects in the orofacial region. Malignant tumors, with their extensive resections, are frequently the cause of disfiguring facial defects. A variety of prosthetic techniques to treat such patients is presented.

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Treatment of oral and facial cancers can result in severe oral dysfunction and facial disfigurement but today it is possible to restore many patients to near normal form and function, enabling them to continue to have useful and productive lives. In the late 1980s several technical improvements were made – for example the development of osseointegrated dental implants and free vascularized flaps – but in recent times the most significant improvements have been the result of improved collaborations between medical and dental researchers and clinicians. In the leading cancer centers of the world, prosthodontists see and examine the patient prior to their cancer treatment and work with their colleagues in surgical oncology, reconstructive surgery or radiation oncology to minimize post-treatment morbidities and to develop plans to rehabilitate the patient. Presently with a proper multidisciplinary approach to patient care, almost all patients, treated with surgical resection and/or radia-

tion for oral or facial cancer can be very effectively rehabilitated, retaining their ability to speak, swallow, masticate and control their saliva, enabling them to interact socially with family, friends, and professional colleagues.

Head and neck cancer patients who require maxillofacial rehabilitation may be arbitrarily classified according to their post-treatment surgical defects and morbidity, which include maxillary, tongue–mandibular, and facial defects. This chapter will concentrate primarily on the role of implants in the restoration of defects secondary to surgical ablation of head and neck tumors.

Maxillary defects

Most tumors arising from the paranasal sinus, palatal epithelium, or minor salivary glands require either a partial or radical maxillectomy (Fig. 28.1a). Defects of the hard or soft palate produce a variety of problems: hypernasality makes speech unintelligible; mastication efficiency is compromised, particularly for the edentulous patient, because teeth and denture-bearing tissue surfaces are lost, and support, stability, and retention of the maxillary prosthesis are compromised; swallowing is awkward, since food and liquids may be forced up into the nasal cavity and out the nose; nasal and sinus secretions collect in the defect area; and facial disfigurement can result from lack of midface bony support.

Rehabilitation after resection of the hard or soft palate is best accomplished prosthetically. Customarily, a temporary prosthesis, known as an immediate surgical obturator, is placed at the time of surgery. During the healing period, this prosthesis is relined periodically with temporary denture liners to compensate for tissue changes secondary to organization and contracture of the tissues adjacent to the defect. When these tissues are well healed and dimensionally stable (usually 3–4 months after surgery), the definitive prosthesis is made. Inadequate retention, stability, and support are the main problems associated

with the use of an obturator prosthesis. The remaining teeth, therefore, become extremely valuable in providing support, retention, and stability for these restorations. If there are adequate numbers of teeth remaining after the resection to provide the support, stability, and retention for the obturator prosthesis, then surgical reconstruction should not be considered (Fig. 28.1).

Almost all defects of the hard palate are restored effectively with a prosthesis. Best results are achieved when the surgeon prepares the defect for the obturator prosthesis by skin grafting the defect, sparing the premaxilla when possible, removing unwanted turbinates enabling appropriate access to the defect, and making alveolar bone cuts in such a fashion as to maximize the effectiveness of abutment teeth adjacent to the defect.¹ When the patient is edentulous or the remaining teeth do not have a favorable prognosis, osseointegrated implants (Fig. 28.2) can be used to enhance support, stability, and retention of the obturator. The purpose of these prostheses is to restore the physical separation between the oral and nasal cavities, thereby restoring speech and swallowing to normal, and to provide support for the lip and cheek.

Prosthodontic treatment

It is essential that the prosthodontist examines and consults with the patient before surgery. The sequence of treatment should be explained to the patient, and



Fig. 28.1 (a) Large maxillectomy defect. (b) Maxillary obturator. (c) Prosthesis in position. Speech, mastication, and swallowing are restored to normal.



Fig. 28.2 Edentulous patient with partial palatectomy defect. Implants provide retention, stability, and support for obturator prosthesis. Note rests (arrows) on the bar and resilient attachments.



Fig. 28.3 Implants being inserted following maxillectomy.

diagnostic casts and appropriate radiographs should be obtained. With this information, the prosthodontist is ready to consult with the surgeon. In edentulous patients, the placement of osseointegrated implants into available maxillary sites should be considered for patients not scheduled for postoperative radiation. These implants can be used later to facilitate the retention and stability of, and support for, the future obturator prosthesis (Fig. 28.3).

Teeth greatly improve the retention and stability of the obturator prosthesis. In the absence of teeth, osseointegrated implants can provide similar retention and stability for the obturator. Speech, swallowing, mastication, and facial contour can be restored with proper extensions and obturation. The obturator should extend maximally up the lateral wall of the defect (Fig. 28.4). This high lateral extension increases retention and stability and helps recontour the lip and cheek.

The movement of the medial side of the ramus into the distolateral area of the defect must be accounted for during impression procedures. The extension superiorly along the medial margin of the defect should not exceed the level of the repositioned palatal mucosa or the floor of the nose. Further superior extension will impede normal nasal airflow during speech and breathing, and may result in unnecessary and painful ulceration of the respiratory mucosa lining the nasal septum. In some patients, extension across the nasal surface of the soft palate or into the nasal aperture may be necessary to provide acceptable retention.

The placement of osseointegrated implants dramatically improves function of the obturator prosthesis, particularly for edentulous patients.^{2,3} The most desirable locations are the premaxillary segment and the maxillary tuberosity (Figs 28.2 and 28.5). Careful attention must be paid to the design of the tissue bar. Fewer implant sites are available and the anterior-posterior spread of those placed into the premaxilla is limited, subjecting these implants to an increased risk

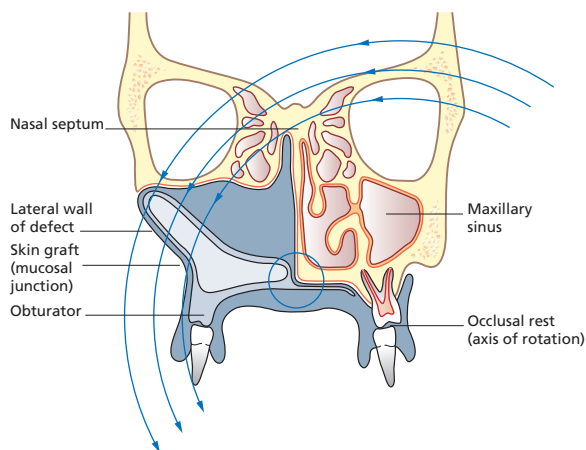


Fig. 28.4 Cross-section illustration showing peripheral extension of obturator against lateral wall of surgical defect. In total maxillectomy defect this peripheral extension will exhibit the greatest range of motion.

of implant overload if designs are implant supported as opposed to implant assisted. The authors suggest that occlusal rests be placed on the tissue bars (Fig. 28.2) and resilient attachments be attached to both ends of the bar. Implants are splinted together and this design directs the occlusal forces on the unresected side (where the patient will actually masticate) along the long axis of the implants (Fig. 28.2). Patients are advised to masticate on the unresected side. Forceful occlusion on the defect side will result in rapid wear of the attachments securing the obturator prosthesis to the tissue bar and may also result in bone loss around the implants, particularly the implant adjacent to the defect.

When the prosthesis is completed, speech and swallowing are restored to normal limits. Most prostheses require relining within the first year because of slow but continuous changes of the tissues on the periphery of the surgical defect.

For the edentulous patient who will be undergoing extensive resections involving most of the hard palate, the choice of prosthetic vs surgical reconstruction is dependent upon the availability of suitable implant sites. The data indicate that implants have been reasonably successful in this group of patients. In the

Roumanas report² 102 implants were placed in 26 patients with acquired maxillary defects secondary to resection of palatal and paranasal sinus tumors. Six patients with 19 implants were not available for follow-up because either the patient expired prior to second-stage surgery or they developed recurrence of their tumors. Of the 83 implants remaining for study, four were buried and one patient with one implant had not undergone second-stage surgery. Seventy-eight implants with known status were available for study. The implant survival rate for these 78 implants was 69.2%. The implant survival rates were 63.6% for the irradiated group (67.0% before radiation and 50% after radiation) (mean dose of 5000 cGy) and 82.6% for the non-irradiated group. Anterior implants demonstrated 2.7 times greater number of exposed threads than posterior implants.

If palatal or alveolar bone sites are not feasible implant sites, another option to consider would be the use of implants placed in the zygoma (zygoma implants). Zygoma implants, 30–50 mm long, have been employed successfully in patients with large or total palatotomy defects. In the UCSF report,⁴ 28 zygoma implants were placed in nine patients with large maxillary defects. Six implants failed (most in irradiated patients), and five of the patients were eventually restored with an implant-retained obturator prosthesis. When restoring total palatotomy defects these clinicians recommend that two implants be placed into each residual zygoma and that all four be splinted together with a tissue bar (Fig. 28.6). Landis reported on the use of 36 zygoma implants in 15 patients.⁵ Twenty-four conventional implants were also placed in oral sites in these patients. Twelve of the 15 patients presented status postresection of large tumors of the maxilla and three presented with congenital defects. Nine of the patients were edentulous. Six of the twelve oncology patients received



Fig. 28.5 Solitary implant in maxillary tuberosity of an almost total palatotomy defect.

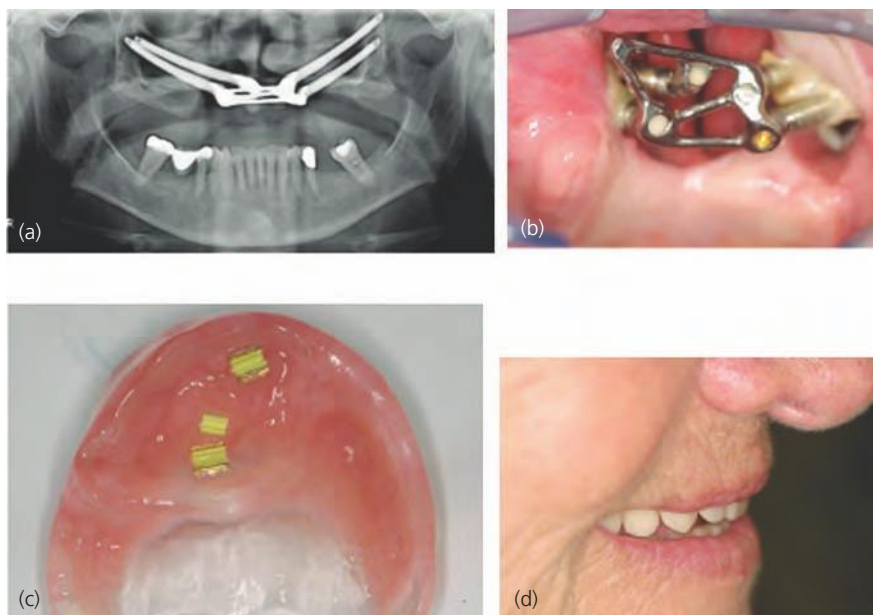


Fig. 28.6 Zygoma implants. (a) Two are placed on each side. (b) All four implants are splinted together with tissue bar. Note accumulation of plaque and calculus. (c) Completed prosthesis. (d) Prosthesis in position.

chemotherapy and seven received radiation therapy during the course of their treatments. Radiation doses ranged from 45–61 Gy. Nine of the patients were edentulous. The prostheses were retained with telescopic crowns associated with, when necessary, angled abutments. Follow-up ranged from 13–102 months. Three zygoma implants were lost secondary to overloading and/or chronic infection and five others were removed secondary to resection of recurrent disease.

It is possible to obturate hard palate defects with free vascularized flaps and this may be desirable in potentially non-compliant edentulous patients with large defects who may have difficulty tolerating a large removable prosthesis. However, in almost all situations surgical reconstruction of such defects is undesirable and actually impairs oral function. Restoration of partially dentate patients with unilateral defects of the hard palate with vascularized flaps is contraindicated in almost all instances. These flaps distort the palatal contours and compromise the tongue space (Fig. 28.7). Bulky soft tissue flaps preclude replacement of missing dentition in many patients and lack of tongue space compromises speech articulation. In addition, secretions accumulate on the sinus side of the flaps. These secretions become crusted, and are subsequently colonized by microorganisms. These mucous crusts become odiferous and also cause local infections. Total palatal defects are the exception to this rule. Free flap selection should be governed by the need to create viable implant sites. Fibula free flaps are preferred and subsequently implants are placed and an implant-retained prosthesis is fabricated that replaces the dentition and restores normal palatal contours and, if



Fig. 28.7 Partial palatotomy defect obturated with a radial forearm flap. Palatal contours are excessive and speech articulation was affected.

necessary, restores the partition between the nasal and oral cavities.

Definitive soft palate prosthesis

Defects of the soft palate and velopharyngeal complex require different and more complex prosthetic treatment. Velopharyngeal closure normally occurs when the soft palate elevates and contacts the contracting lateral and posterior pharyngeal walls of the nasopharynx. When a portion of the soft palate or lateral pharyngeal wall is excised or when the soft palate is perforated, scarred, or neurologically impaired, effective velopharyngeal closure cannot occur. Speech becomes hypernasal, and normal swallowing is not possible. With a properly extended and contoured pharyngeal obturator, the patient will be able to re-establish velopharyngeal closure if a residual portion of the velopharyngeal muscular mechanism still exhibits some functional movement. The obturator must not interfere with breathing, impinge upon soft tissues during postural movements, or hamper the tongue during swallowing and speech.

The soft palate obturator remains in a fixed position in the nasopharynx and does not attempt to duplicate normal movements of the soft palate. The inferior surface of the obturator should be level with the hard palate contour, which in most patients is approximately the level of the anterior tubercle of the atlas. The inferior margin of the posterior surface of the obturator contacts Passavant's pad, if present, and extends approximately 10 mm superiorly into the nasopharynx. During breathing and the production of nasal speech sounds, the space around the obturator reflects the potential for muscular contraction. During swallowing and the production of other speech sounds, this sphincteric muscular network moves into contact with the stationary acrylic resin obturator, establishing velopharyngeal closure (Fig. 28.8). A correctly constructed obturator and one accurately positioned in the nasopharynx to properly interact with the residual velopharyngeal musculature, will result in the return of normal speech and swallowing.

In edentulous patients a conventional complete denture may lack retention which prevents the obturator prosthesis from effectively interacting with the residual velopharyngeal musculature. The addition of implants provides sufficient retention to ensure accu-

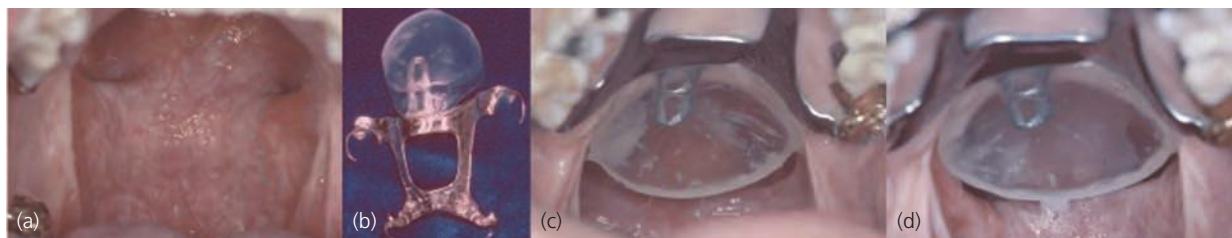


Fig. 28.8 (a) Soft palate defect. (b) Pharyngeal obturator prosthesis. (c) Prosthesis in position with the tissues at rest. (d) During contraction the obturator comes in contact with the residual velopharyngeal musculature and permits the production of normal oral sounds and swallowing.

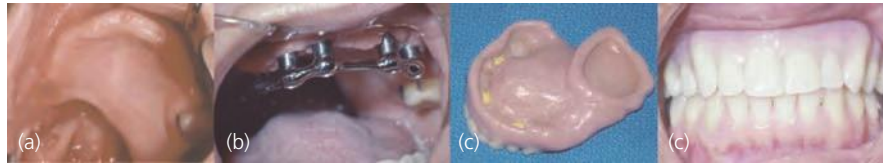


Fig. 28.9 (a) Combination hard-soft palate defect. (b) Implant tissue bar in position. (c) Implant-retained overlay denture with obturator. (d) Prosthesis in position. Speech, swallowing, and mastication are restored.

rate positioning of the obturator prosthesis in relationship with the residual velopharyngeal musculature and will enable effective velopharyngeal closure and normal velopharyngeal function (Fig. 28.9).

Edentulous patients with unrepaired clefts or those who do not have adequate numbers of teeth to support and retain the prosthesis can also benefit from the use of osseointegrated implants.

Tongue-mandible defects

Disabilities resulting from tongue-mandibular resections include impaired speech articulation, difficulty swallowing, deviation of the mandible during functional movements, poor control of salivary secretions, and often cosmetic disfigurement. If the surgical wound is closed primarily (primary closure) the functional disabilities are compounded. Advanced tumors of the oral tongue and floor of the mouth often require extensive resection of soft tissue and adjacent mandible. This loss of bulk and mobility combined



Fig. 28.10 Patient following resection of lateral tongue, floor of mouth, and mandible. The fibula free flap failed. Note the resultant mandibular deviation.



Fig. 28.11 The mandible and the lateral third of the tongue have been reconstructed with a fibula free flap.

with the loss of motor and sensory innervation leads to misarticulation of most speech sounds, inability to control saliva, impaired mastication, and seriously impacts the patient's ability to manipulate complete dentures. Deglutition is less impaired, and most patients learn to swallow fairly efficiently unless they receive chemoradiation following their surgical resections. In addition, the motor and sensory innervation of the lower lip on the resected side is often compromised, further impairing speech, mastication, control of saliva, and use of conventional complete dentures.

If much of the mandible is removed and not reconstructed the remaining functional mandibular segment will be retruded and deviated towards the surgical side. When the jaw is opened this deviation increases. These factors, combined with impaired tongue function, prevent effective mastication. The severity and permanence of mandibular deviation varies (Fig. 28.10). However some patients can attain reasonable occlusal relationships, although some frontal plane rotation will be observed. It is, therefore, highly desirable to reconstruct the mandible at the time of tumor ablation with a free vascularized flap.

From the prosthodontic perspective the first priority, however, following surgical ablation, should be restoration of the soft tissue deficit, particularly reconstruction of the tongue. With proper use of free flaps, tongue bulk can be restored and the mobility of the reconstructed tongue (Fig. 28.11) results in acceptable speech, swallowing, and saliva control.⁶ The advantage of free tissue transfers (free flaps) over musculocutaneous flaps is the improved blood supply, enhancing wound healing and flap survival. Most important with regards to function, there is considerably less scarring associated with free flaps, which permits the residual tongue-flap combination to move laterally, elevate, and interact with the maxillary dentition and the palatal vault during speech, mastication, and swallowing. If large mandibular segments need to be restored, the fibula is the preferred donor site unless the soft tissue deficit is unusually large. The osteotomized fibula provides sufficient length and bulk of bone and osseointegrated implants can be placed to retain and support a prosthesis (Fig. 28.12). The authors tend to favor removable overlay prostheses with milled tissue bars in these restorations because this design allows for more flexibility with regard to development of proper lip and cheek contours and is more easily serviced in the event of damage to the prosthesis (Figs 28.12 and 28.13).

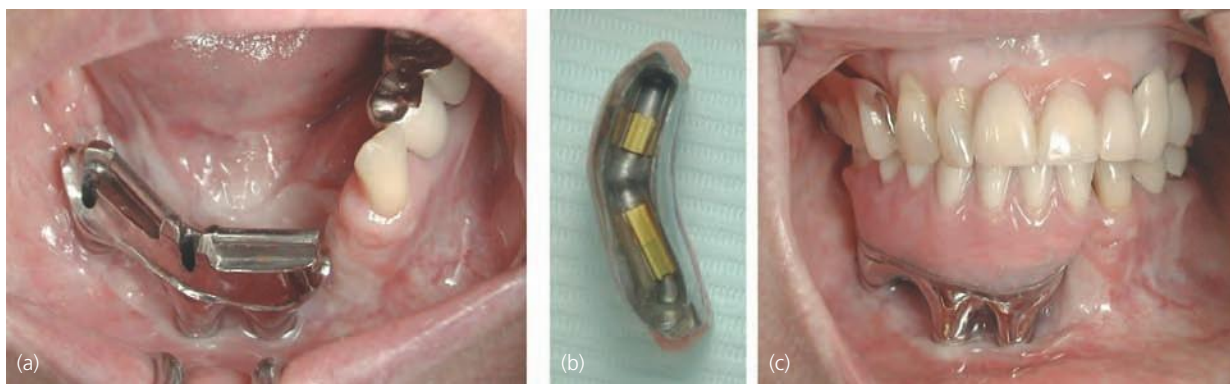


Fig. 28.12 Mandibular defect reconstructed with fibula free flap. Tongue, and hypoglossal and lingual nerves are intact. Flangeless overlay prosthesis. (a) Tissue bar milled to a 3° taper. (b) Overlay prosthesis. Clip housings incorporated within metal substructure. (c) Prosthesis in position.



Fig. 28.13 (a) Implants placed in a free graft. (b) Milled tissue bar. (c) Overlay prosthesis. Note presence of flange support for the lip. (d) Prosthesis in position.

Free bone grafts

Free grafts are rapidly being replaced by free vascularized flaps as a means of restoring mandibular discontinuity defects. These grafts are limited by the fact that most tumors leading to mandibular resections are malignant, and arise from the oral mucosa; they require extensive resection of the soft tissues of adjacent tongue, floor of mouth, and alveolus. Many other patients require postoperative radiotherapy or chemoradiation which further compromises the blood supply to the local tissues which must house the graft. In these situations either the volume of the residual soft tissues is insufficient to house the graft or the residual vasculature is insufficient to support the graft. However, free grafts can be effectively employed to restore mandibular continuity following resection of benign tumors such as ameloblastomas, when the resection is confined to the mandible itself.

Autogenous graft sources include iliac crest, rib, and clavicle. Most commonly the defects are restored either with particulate autogenous marrow housed in a metal tray or with a block of bone, both usually obtained from the iliac crest. The use of myocutaneous flaps for closure of the initial wound facilitates free bone grafting of continuity defects by enhancing the volume and vascularity of the recipient soft tissues. These grafts are excellent sites for placement of osseointegrated implants (Fig. 28.13). The long-term success rates of implants in free grafts exceeds 90% in most clinical follow-up studies.⁷⁻¹⁰

Free bone grafts demonstrate a homogeneous calcification pattern that results in excellent bone anchorage. Implant position should be prosthetically driven

and should be based on a diagnostic wax-up and a surgical template.

A major challenge is to create a zone of thin, immobile, and, if possible, keratinized tissue around the implants as the restoration emerges through the peri-implant tissues. If the peri-implant tissues are excessively thick and movable, these tissues are subject to hypertrophy and local infection. This is particularly an issue in patients reconstructed with free vascularized flaps, because the soft tissues overlying the bone may be up to 1 cm thick. Thinning of these tissues is best accomplished at second-stage implant surgery. A stent secured to the implant fixtures will facilitate healing following this procedure. Abutments emerging through skin must be highly polished to avoid peri-implant tissue inflammation and hypertrophy.

Free vascularized flaps

A major advance in mandibular reconstruction has been the development of improved techniques in microvascular surgery, which allow for composite grafting of larger volumes of tissue. In microvascular free tissue transfer (free flaps), bone, muscle, connective tissue, and skin can be autogenously grafted and remain viable. The grafting can be accomplished simultaneously with resection of the tumor, with excellent results.⁶

The composite fibula flap is the preferred donor site for most complex mandibular discontinuity defects but the surgeon must realize that the skin paddle may not always be sufficient for defects that involve significant portions of the anterior two thirds of the tongue. Multiple osteotomies may be per-

formed without devascularizing the bone segments, to replicate the arch of curvature of the replaced mandible. The contours of the fibula and its well developed cortices make it an excellent recipient of osseointegrated implants. Success rates of implants in free fibula grafts are generally above 90% in most clinical follow-up studies.¹¹ In this group of patients, the implant-retained restoration is most effective if there is sensory innervation remaining on the defect side, particularly the lateral border of the tongue. When the lingual nerve has been resected, or in patients where the lateral portion of the oral tongue is reconstructed with a free flap, the patient will be unable to detect and manipulate the food bolus, and, as a result, mastication will not be as efficient on the resected side. If such is the case and the patient has healthy dentition remaining in the unresected portion of the mandible, a removable partial denture is probably in the best interest of the patient.

Definitive prosthetic restoration

The functional outcomes of implant-retained prostheses for partially edentulous patients with resections of the tongue and mandible depend primarily upon the sensory status locally and the function of the residual tongue. In some patients with poor tongue function, only appearance and oral competence can be improved, while in others with good tongue function, mastication is a reasonable objective.^{12,13}

Complete dentures for edentulous patients with unrestored discontinuity defects of the mandible may provide esthetic improvement by replacing teeth and improving lip and cheek contours, but mastication is generally not possible even if the patient has reasonably good tongue control. A number of factors affect

the ability of these patients to masticate with so-called resection dentures:

- stability, support, and retention for the mandibular denture are compromised;
- compromised quality and quantity of saliva secondary to postradiation xerostomia;
- the angular pathway of mandibular closure, which induces lateral forces upon the dentures, tending to dislodge them;
- abnormal maxillomandibular relationships that may prevent ideal placement of the denture teeth over their supporting structures;
- impairment of motor and/or sensory control of the tongue, lip, and cheek, limiting the patient's ability to control dentures during function.

Implant-assisted resection dentures can overcome many of these difficulties, particularly those associated with compromised retention, stability, or support.

Implant-assisted overlay dentures

Patients with reasonable tongue bulk and mobility and with motor and sensory innervation intact on at least one side gain the most from implant-retained overlay dentures, whether the patient presents with either a resected or unresected discontinuity defect. The patients shown in Fig. 28.14 would not derive significant benefit from implant placement if improved mastication is the primary goal of the patient and the clinician. On the other hand, the patient in Fig. 28.15 would stand to derive significant benefit because his tongue has been reconstructed and has good mobility. In such patients mastication is improved because the tongue is no longer required to

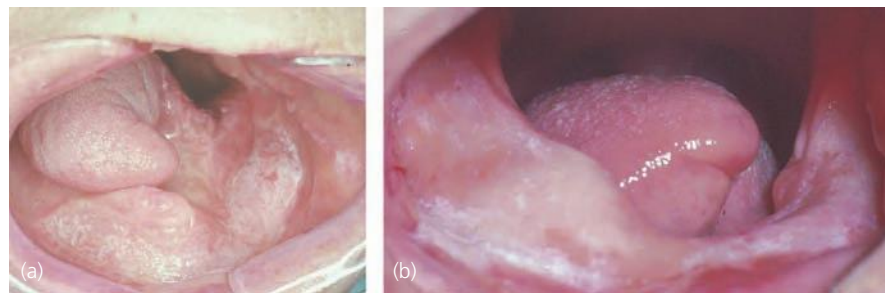


Fig. 28.14 In both patients the mandible has been reconstructed but not the tongue. (a) Tongue immobilized. (b) Tongue unable to elevate to the level of the occlusal plane. Result: prognosis for mastication is very poor for both patients even if implants are employed to retain the lower denture.



Fig. 28.15 (a–c) Partial glossectomy defect restored with radial forearm flap. Portion of bearing surface resurfaced with flap. Movement of tongue is excellent. Prosthodontic prognosis is excellent, particularly if implants are employed to retain and stabilize the mandibular denture.

control the denture, and can be employed to manipulate the food bolus during mastication and swallowing. If the patient's speech is intelligible, this is a sign of good tongue mobility and control, and the prognosis for effective bolus manipulation and mastication with an implant-retained prosthesis is therefore going to be favorable (Fig. 28.15).

In unreconstructed discontinuity patients, if implants are to be placed into the mandible to retain and support an overlay prosthesis, consideration should also be given to placing implants in the opposing maxilla. The unilateral occlusal forces and increased lateral forces generated during the chewing cycle tend to dislodge the upper denture. In addition, xerostomia secondary to radiation therapy may compromise the peripheral seal of the maxillary complete denture. Therefore, implants should be considered if the retention and stability of a conventional maxillary denture is marginal (Fig. 28.16).

In most patients, the most favorable implant sites available in the edentulous resected mandible are located in the symphyseal region. A minimum of two implants should be placed. However, more may be desirable, if space allows. The authors prefer to splint the implants together and fabricate implant-assisted tissue bar designs for such defects in order to minimize the forces delivered to the implants. Examples of implant-assisted designs are shown in Figs 28.16 and 28.17.

Facial defects

Surgical reconstruction vs prosthetic restoration

Surgical reconstruction and prosthetic restoration both have limitations. The surgeon is limited by the availability of tissue, damage to the local vascular bed, and the need for periodic visual inspection of an oncologic defect. The prosthodontist is limited by movable tissue beds, and difficulties in retaining very large prostheses. The method of facial restoration should be considered before surgery, and patients should participate in the decision-making process and have realistic expectations.

Surgical reconstruction of small facial defects is preferable. However, it is difficult for the surgeon to contour a facial component that is as effective in appearance as a well made prosthesis. Osseointegrated implants in facial defects have changed patient perceptions about facial prostheses because of the retention achieved.¹⁴ Additionally, when a large resection is necessary and recurrence of tumor is probable, it is advantageous to be able to monitor the surgical site closely, which a prosthesis permits. Even when surgical reconstruction is deemed possible, most surgeons prefer to wait at least 1 year before surgical reconstruction. Also, surgical restoration of large defects is technically difficult and requires multiple procedures



Fig. 28.16 Mandibular discontinuity defect opposing the severely resorbed maxilla. (a) Implants placed into opposing maxilla to prevent tipping and loss of retention of maxillary denture during function. (b) Overlay prosthesis. (c) Prostheses inserted.

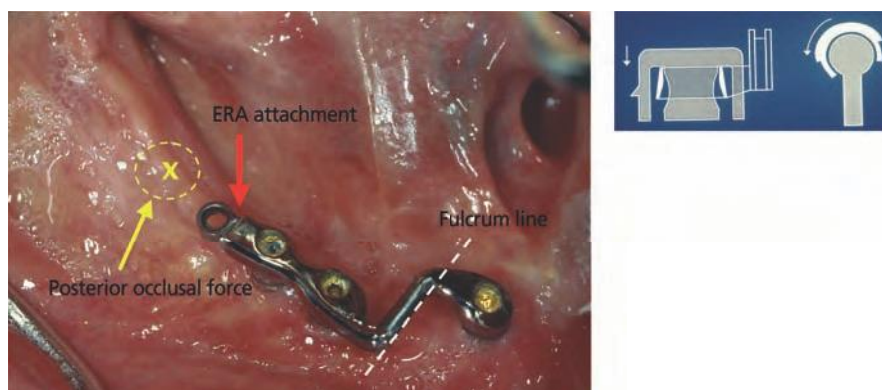


Fig. 28.17 When occlusal force is applied in extension area (x), prosthesis is designed to rotate around Hader bar (fulcrum line) into the posterior denture bearing surfaces, minimizing forces delivered to implants.

and hospitalizations, which may be further complicated if radiotherapy has been included.

Alterations at surgery to enhance the prosthetic prognosis

The key to creating esthetic facial prostheses is properly designed surgical defects. If surgical reconstruction of a facial defect following tumor ablation is not anticipated, several factors need to be addressed. The key to an esthetic facial prosthesis is to create a defect with minimal distortion of facial contours adjacent to the defect (Fig. 28.18). During nasal resections, the nasal bones should be resected and all raw tissue surfaces should be lined with split-thickness skin grafts. The anterior segment of the nasal septum should be removed and the anterior portion of the floor of the nose lined with skin. Primary closure of skin to mucosal margins should be avoided, for this practice distorts nasolabial folds, cheek and lip contours. In resections of the ear, total as opposed to partial resection is preferred. When possible however, the tragus should be retained because this structure will hide the anterior margin of the prosthesis. During orbital exenteration the eye lids should be removed and the orbit lined with a skin graft.

Covering facial defects with free flaps precludes the fabrication of prosthetic restorations in almost all situations and should be avoided.

Prosthetic facial restorations

The challenge to the prosthodontist is to fabricate an esthetically pleasing restoration. A conspicuous prosthesis may produce more anxiety and permit less social readjustment than a simple facial bandage or eye patch. The most critical period is the first 2–3 days after delivery. The patient must understand that a prosthesis has two different roles: for family, close friends, or business associates, it can only cosmetically replace the excised tissues; for the public at large, it generally provides enough concealment to render the reconstructed defect inconspicuous.

Materials and methods of retention for facial prostheses

Current materials all possess some undesirable characteristics.¹⁵ The materials most often used are the silicone elastomers, which have achieved wide clinical acceptance. Adhesives can be used to retain most facial prostheses but implant retention is preferred. Osseointegrated implants result in extremely well retained prostheses that permit vigorous physical activities. Patients favor implant-retained facial prostheses over adhesive-retained prostheses by a wide margin.¹⁴

Craniofacial implants

The use of osseointegrated implants has significantly improved patient acceptance of facial prostheses.¹⁴ The retention provided by implants makes it possible to retain large prostheses and makes it possible to fabricate thinner margins, which enhance esthetics by blending and moving more effectively with adjacent mobile tissues. Other benefits include elimination of the occasional skin reaction to skin adhesives, ease and enhanced accuracy of prosthesis placement, improved patient comfort, and decreased daily maintenance, which also increases the lifespan of the facial prosthesis.

Treatment planning

The implants must be positioned within the confines of the proposed facial prosthesis. In most patients it is desirable to sculpt a wax replica of the future prosthesis and to use this replica to fabricate a surgical template. This template is sterilized and used as a guide during surgery to ensure proper implant position and angulation (Fig. 28.19).

The number and arrangement of implants and possible bone sites are determined. In large extensive defects, computed tomography (CT) scans and three-dimensional models are useful in evaluating potential bone sites and important adjacent structures. The health of skin and soft tissues circumscribing osseointegrated implants is easier to maintain if these tissues

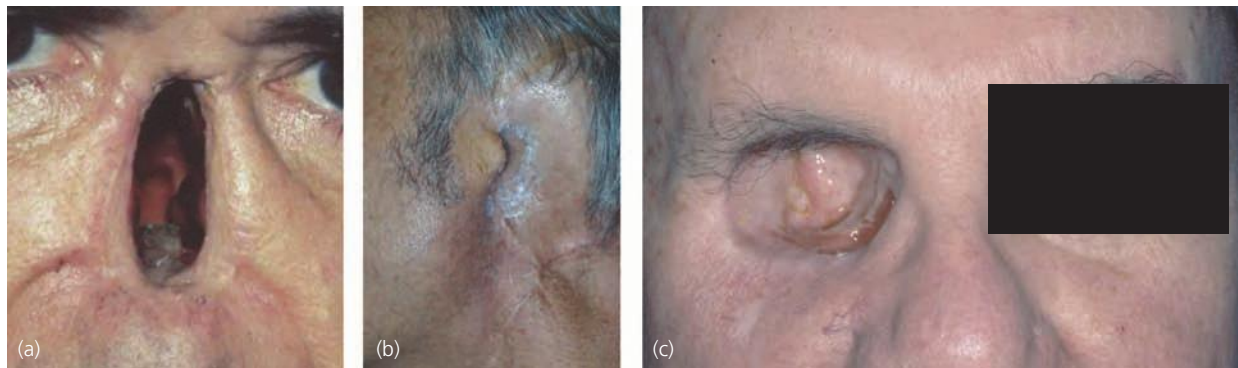


Fig. 28.18 (a) Favorable nasal defect. Nasal bones and anterior septum removed, raw tissues lined with skin. (b) Favorable auricular defect. Tragus retained. (c) Favorable orbital defect. Orbit lined with skin.

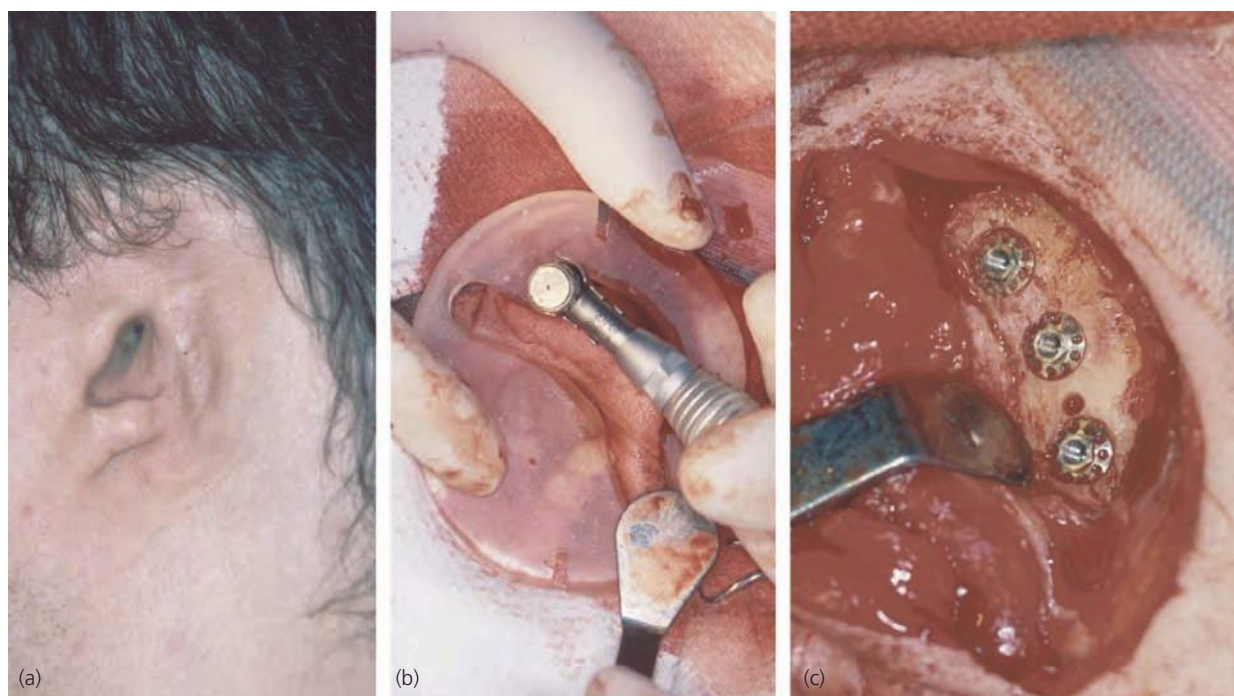


Fig. 28.19 (a) Auricular defect. (b) Implant placement in mastoid bone using surgical template. (c, d) Implants inserted.

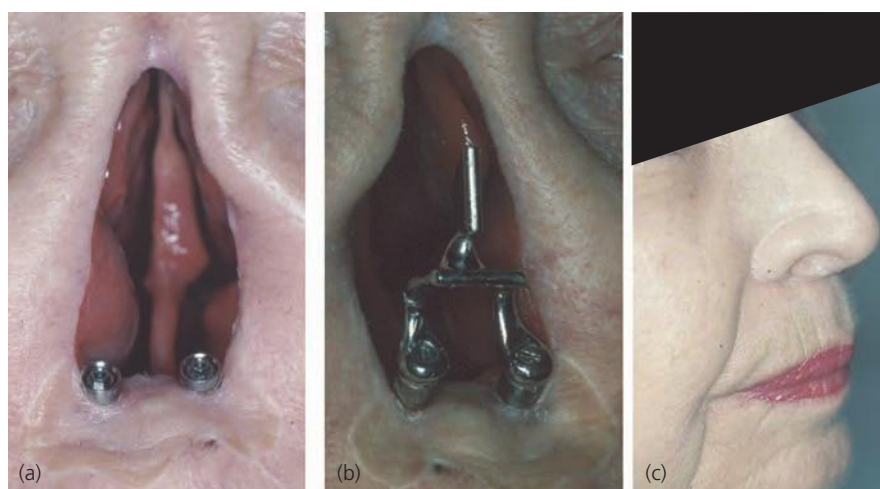


Fig. 28.20 (a) Implants placed into the nasal floor. (b) Tissue bar in position. Note horizontal and vertical "Hader" bar configuration. (c) Completed prosthesis.

are thin (less than 5 mm thickness) and attached to underlying periosteum. If the skin contains hair follicles or scar tissue from past reconstructive procedures, these tissues should be removed and replaced with skin grafts.

Surgical placement

Craniofacial implant fixtures are fabricated from titanium. They are available in either 3 or 4 mm lengths, with a 5 mm diameter flange. The short lengths are designed to permit placement in areas with limited available bone. The flange facilitates initial stabilization of the implant and prevents penetration into interior components. For ear defects, two or three implants are positioned posteriorly and superiorly to the ear canal (Fig. 28.19). For nasal defects, the preferred fixture location is the anterior portion of the

floor of the nose (Fig. 28.20). Care should be taken to avoid the roots of the teeth in the area. For orbital defects, the preferred location is the lateral portion of the superior orbital rim.

Several attachment systems have been used to connect the facial prosthesis to the implants, such as bar-clips, magnets, and O-ring types. For auricular and nasal or large midfacial prostheses the authors prefer the bar-clip systems because they provide superior retention (Figs 28.20 and 28.21). They prefer magnetic retention for orbital prosthesis, because the ease of insertion of magnetically retained prostheses outweighs the possibilities of magnet corrosion and decreased retention over time. An acrylic resin substructure retaining the attachments is designed to fit within the contours of the silicone facial prosthesis. It should possess sufficient surface area so that the

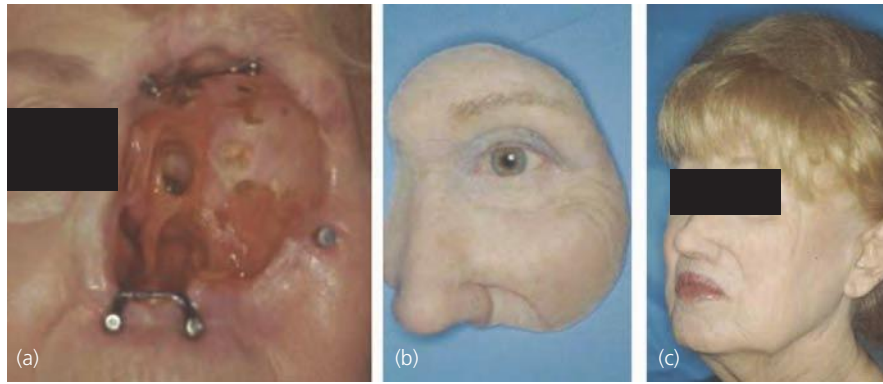


Fig. 28.21 (a) Large facial defect with tissue bars. (b) Facial prosthesis. (c) Prosthesis in position.

bond between the acrylic resin substructure and the silicone prosthesis will not fail during insertion or removal of the prosthesis.

Success rates for auricular sites have exceeded 90% in most studies and few complications have been encountered. Success rates of the floor of nose sites are around 80%. Success rates in the orbit have ranged from 50–70%. Success rates are diminished if implant sites have been irradiated previously particularly in the orbit (Tables 28.1 and 28.2).¹⁶

Implants in irradiated tissues

Irradiation of head and neck tumors predisposes to changes in bone, skin, and mucosa, which affect the predictability of osseointegrated implants. The most significant of these changes are loss of vasculature and impaired function of osteoprogenitor cells in the marrow. Long-term function of osseointegrated implants is dependent upon the presence of viable bone that is capable of remodeling and turnover as the implant is subjected to stresses associated with supporting, retaining, and stabilizing prosthetic restorations. The viability of irradiated bone may not be sufficient to ensure a long-term predictable result, particularly in anatomic sites such as the supraorbital rim and the mandible. Even in the maxilla remodeling and turnover of bone subjected to high dose radiotherapy (above 5000 cGy) may be adversely affected to the point where an implant subject to functional stresses cannot be sustained.

When implants are considered for the irradiated patient several issues require careful consideration such as the risk of osteoradionecrosis, the potential benefit provided by the implants, the potential morbidity associated with implant failure, and the potential usefulness of hyperbaric oxygen.

Predictability of implants in irradiated bone

Preliminary reports and our own experience indicate that the success–failure rate of osseointegrated implants in irradiated bone appears to be dependent

upon the anatomic site selected, the dose to the site, and the use of hyperbaric oxygen. Animal experiments have shown that the bone-implant contact area is reduced at irradiated sites.^{17,18} In addition, Nishimura has shown that the quality of bone in the implant appositional zone is compromised, particularly at high radiation dose levels.¹⁹ In his study 44 adult New Zealand rabbits were assigned at random to six test groups. The animals received radiation to either the proximal or diaphyseal segments of both tibias. Equivalent doses ranged from 4000–7000 cGy. Three months following completion of radiation treatment, 5 mm screw-type implants were placed into each half of the left tibia of the surviving 38 rabbits. Polyfluorochrome labeling was performed 3 months postimplant placement and 2 days prior to sacrifice. Ground non-decalcified sections were prepared and evaluated. Results revealed a steady decrease in the label, especially when the equivalent dosage exceeded 5800 cGy, indicating reduced cellular activity. Also, when viewed under polarized light, the specimens receiving the highest radiation levels showed a preponderance of woven bone as compared to the dense lamellar bone seen in the controls and in specimens receiving lower doses.

The early trends seen in recent clinical reports appear to substantiate the concerns raised in the animal studies; namely, implants placed in irradiated maxilla and mandible have a significantly lower success rate.^{2,20–24} Implants in irradiated tissues used to retain facial prostheses appear to have a significantly lower success rate than implants in non-irradiated tissues (Tables 28.1 and 28.2).¹⁶ Roumanas reported on the results of 33 implants placed in the irradiated maxillae of 13 patients.² All patients received at least 5000 cGy to the implant sites. Eleven of the 33 failed and were removed and two others were buried beneath the mucosa for a success rate of 60.6%. Many of the remaining implants demonstrated moderate to severe bone loss (bone loss extending to at least the level of the fourth thread). In the mandible the success rates have varied from 65%²³ to 84%.²¹

Because of these results some clinicians^{20,23,25} have attempted to improve the viability of bone with hyperbaric oxygen treatments prior to implant place-

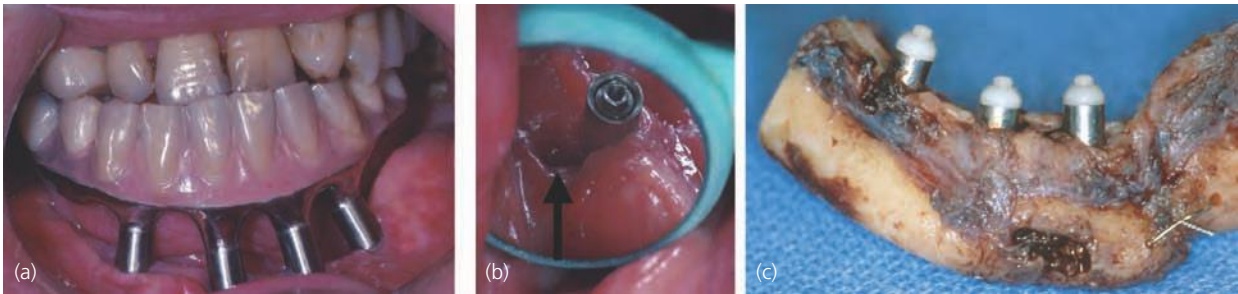


Fig. 28.22 (a) This patient received 6600 cGy for a squamous carcinoma of the lateral tongue. Three years later implants were placed. (b) Three years after implant placement the patient developed an infection associated with the left posterior implant. (c) Eventually, the patient developed an osteoradionecrosis and a pathologic fracture of the mandible; subsequently the mandible was resected. (From Beumer J, Sung E, Kagan R. 2004. Dental Management of the Irradiated Patient. An electronic program of instruction issued by the ISMR. Reproduced with permission.)

Table 28.1 Non-irradiated craniofacial implants. Data from Roumanas.¹⁶

Implant titles	Patients treated	Number of implants				Survival rate
		Placed	Uncovered	Buried	Failed	
Auricular	35	111	97	8	5	94%
Nasal	16					
Piriform		27	25	0	5	80%
Glabella		6	6	0	4	33%
Orbital	9	28	25	2	7	70%
Overall	60	172	153	10	21	85%

Table 28.2 Irradiated craniofacial implants. Data from Roumanas.¹⁶

Implant titles	Patients treated	Number of implants				Survival rate
		Placed	Uncovered	Buried	Failed	
Auricular	2	6	6	0	0	100%
Nasal	4					
Piriform		8	6	0	1	83%
Glabella		2	2	0	2	0%
Orbital	6	19	15	0	11	27%
Overall	12	35	29	0	14	52%

ment. Granström²⁰ treated 13 patients, who had previously been irradiated, with hyperbaric oxygen. Each patient received 20 treatments, implant surgery was performed, followed by 10 more hyperbaric treatments. Only one implant fixture was lost (2.0% of the total). In his most recent report Granström²³ reported only 29 implants lost of 349 placed in irradiated patients who received hyperbaric oxygen whereas 117 out of 291 placed in irradiated patients who did not receive hyperbaric oxygen were lost.

In summary, it is clear from the current data that osseointegration is impaired in bone that has received doses in excess of 5000 cGy. Success rates, based on short-term clinical reports, are reduced as compared to non-irradiated sites. The success rates are lower than in normal individuals even in the maxilla with its excellent blood supply. In addition, preliminary animal studies referred to previously appear to indicate that the bone–implant interface may be signifi-

cantly compromised making the implant less able to tolerate functional loads. Hyperbaric oxygen appears to help revitalize the bone, leading to improved success rates, but its high cost precludes its use in most patients.

Risk of osteoradionecrosis in the mandible is probably best determined by an analysis of the bone necrosis rate seen in postradiation extractions. Based on this data it should be relatively safe to place implants in irradiated mandibular sites if the dose is less than 5500 cGy. The risk would be quite high for doses above 6500 cGy (Fig. 28.22). In such patients, if implants are desirable, the authors recommend a course of hyperbaric oxygen as described by Granström.²³ In patients with doses to bone sites between 5500 and 6500 cGy, individual patient factors such as the dose per fraction, a previous radical neck dissection, and so on, may be important cofactors to consider when assessing the risk. In these

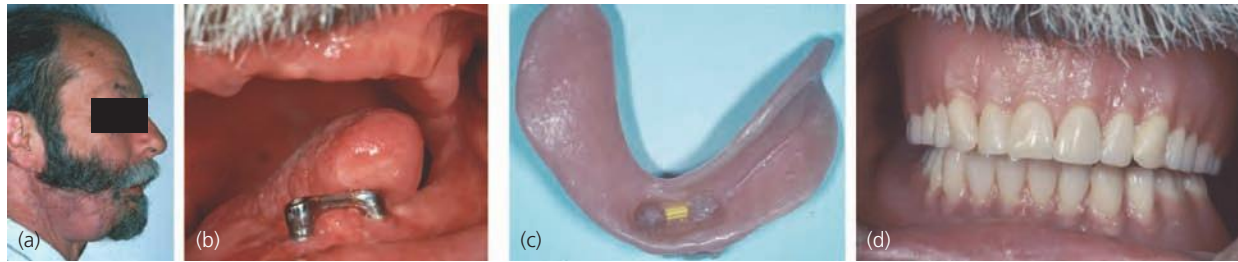


Fig. 28.23 (a, b) These implants were placed anterior to the field of radiation (note distribution of hair in patient's beard). (c) Implant-retained overlay denture. (d) Prostheses in position.

patients, if implants are considered elective they should be deferred. If implants are deemed essential, the authors recommend they be placed in conjunction with hyperbaric oxygen. It should be noted that most patients do not receive radiation to the symphyseal region. Therefore, implants can be placed with a high degree of predictability in this region (Fig. 28.23).

In the maxilla the risk of bone necrosis is probably negligible. The use of hyperbaric oxygen can be justified only on the basis of improving success rates.

Irradiation of existing implants

Irradiation of titanium implants already in place results in backscatter and, therefore, the tissues on the radiation source side of the implants receive a higher dose than the other tissues in the field. The dose is increased about 15% 1 mm from the implant (Fig. 28.24). Because of backscatter and the increased numbers of elderly patients receiving implants,^{26,27} clinicians ask if osseointegrated implants should be removed in patients about to be irradiated for head and neck tumors. Granström²⁰ addressed this question in his report of 11 patients with 33 existing titanium implants who were scheduled to be irradiated. Dosages ranged from 5000 to 6600 cGy. Based on his findings Granström²⁰ recommended that all abutments and superstructures be removed prior to radiation and skin and/or mucosa should be closed over



Fig. 28.24 Implants were placed simultaneous with tumor resection and reconstruction of this large body-symphyseal defect with a fibula free flap. The patient received 6000 cGy postoperatively. Several months later and just after delivery of the tissue bar, the tissues on the labial surfaces of the implants dehiscid and the bone overlying the implants sequestered leading to loss of the implants. The graft remained viable and mandibular continuity was maintained. (From Beumer J, Sung E and Kagan R. 2004. Dental Management of the Irradiated Patient. An electronic program of instruction issued by the ISMR. Reproduced with permission.)

the implant fixtures. When healing is complete, radiation therapy can begin. Following completion of radiation, abutments and the superstructure are reattached and the prosthesis remade or readapted.

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Part 4: Infections

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Tomoari Kuriyama, Michael A.O. Lewis and David W. Williams

Chapter 29

Infections of the Oral and Maxillofacial Region

Tomoari Kuriyama, Michael A.O. Lewis, and David W. Williams

The term “infection” is defined as the detrimental colonization of a host organism by a foreign microorganism, and “inflammation” is the term describing the host’s response to stimuli including those of infection. The onset of infection depends on the balance between the virulence of microorganism and the host’s defenses.¹ Infection resulting from microorganisms invading from outside of the host is termed as an “exogenous infection” whilst an infection caused by microorganisms already residing within the body is called an “endogenous infection”.

Oral and maxillofacial infections generally have the following characteristics: (1) the large majority are endogenous, and commonly involve microorganisms residing in the mouth; (2) the infections frequently originate from existing dental and periodontal diseases.

In this chapter, the etiology, clinical features, diagnosis, and treatment of bacterial, fungal, and viral infections in the oral and maxillofacial region are described. Infection related to salivary glands will be presented in a separate chapter.

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Biomedical sciences

An overview of the basic concepts of biomedical sciences is necessary in order to fully understand the etiology and clinical features of oral and maxillofacial infection, and the implementation of appropriate clinical management.

Anatomy

Oral and maxillofacial bacterial infections often originate from dental diseases and sometimes spread to the jaw, fascial spaces, paranasal sinuses, and lymph nodes. A description of the anatomy of these structures is provided below.

Tooth

The tooth comprises enamel, dentin, cementum, and pulp (Fig. 29.1).² In terms of infection, concerns are perhaps greatest when there is involvement of the pulp. The pulp is the central part of the tooth, and is filled with soft connective tissue containing blood vessels and nerves that enter the tooth from a hole at the apex of the root. There are also accessory canals that branch from the main canal. The pulp fulfills a variety of roles including provision of nutrients to the tooth and formation of dentin. Teeth with pulp necrosis and those where the pulp has been completely removed by endodontic therapy are called non-vital teeth. The hard tissues of a non-vital tooth tend to weaken making them more likely to crack and break when compared with vital teeth. The color of non-vital teeth usually changes to a purple-gray, as the pulp dies and the blood cell components degenerate.

Periodontium (periodontal tissues)

The periodontium describes the specialized tissues that surround and support the teeth, thereby maintaining them within the bones. The four major tissues that comprise the periodontium are the gingiva, alveolar process (alveolar bone), periodontal ligament, and cementum (Fig. 29.1). The alveolar process is the bony portion of the maxilla and mandible where the teeth are embedded and through which the roots of the tooth are supported. The bones, including the alveolar process, consist anatomically of cortical and cancellous bone.³ The cortical bone is the outer

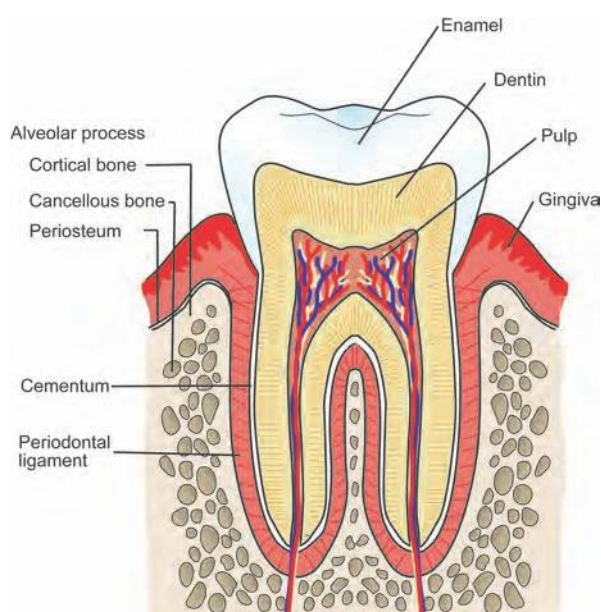


Fig. 29.1 Anatomy of tooth and tissues that surround and support the tooth.

compact layer and provides strength and rigidity. The cancellous bone is the inner spongy bone, which consists of a network of trabeculae arranged to resist external forces. The interstices of cancellous bone are filled with bone marrow. The thickness of cortical bone and density of cancellous bone in the mandible are greater than in the maxilla. A connective tissue membrane called the periosteum surrounds the bone. Periosteum is continuous with muscle attachments, joint capsules, and the deep fascia, and it provides the underlying bone with nutrition.

Fascia spaces

Fasciae are broad sheets of dense connective tissue and function to separate structures that pass over each other during movement and serve as pathways for the course of vascular and neural structures. Fasciae can be divided into two types, superficial and deep.⁴ Superficial fascia is a mixture of loose areolar and adipose tissues that unite the dermis of the skin to the underlying deep fascia. The deep fascia is a membranous layer of connective tissue around the muscles and other deep structures. Between the dense layers of deep fascia in the neck is a loose connective tissue that forms potential spaces that can be eroded or distended by blood, pus, purulent exudates, and air (gas). Thus, fascial spaces are the potential spaces that normally do not exist but become filled under special conditions, including those of infection or bleeding. The arrangement of the fascia determines the pattern of spread of infection. The locations of the most important fascial spaces are discussed later in the section Fascial space infections.

Paranasal sinuses

Paranasal sinuses are air-filled spaces within the bones of the skull and face. They exist bilaterally, and have four subgroups: the frontal sinuses, the ethmoid sinuses, the sphenoid sinuses, and the maxillary sinuses (Fig. 29.2).² These sinuses are lined with mucoperiosteum. Amongst the paranasal subgroups, it is the maxillary sinus that is perhaps the most significant for dentists and oral surgeons. The maxillary sinus is the largest of the paranasal sinuses, and it is pyramidal in shape. The maxillary sinus is very small at birth, but enlarges and becomes fully formed in adolescence (around 18 years of age).⁵ The maxillary sinus opens into the middle meatus of the nose through the hiatus semilunaris. The mucus produced by the glands of the mucous membranes is transferred to the nose by ciliary action of the columnar cells. Drainage of the mucus is also achieved by the siphon action created during nose blowing. The cilia and mucus play a significant role in draining foreign substances including bacteria from the sinus to the nasal cavity. The apices of the second maxillary premolar and the first and second molar teeth are located in close relation to the floor of the sinus and may be

separated only by mucous membrane and thus permit dissemination of agents from dental infection.

The lymph nodes

The lymph nodes are components of the lymphatic system. Lymph nodes act as filters, or traps, for foreign particles. When the host is fighting an infection, characteristic swelling of the lymph nodes may be observed (lymphadenopathy). The lymph nodes in the head and neck are made up of several regional groups and a terminal group. The regional groups comprise the occipital, retroauricular (mastoid), parotid, buccal, submandibular, submental, anterior cervical, superficial cervical, retropharyngeal, laryngeal, and tracheal nodes (Fig. 29.3).^{4,6} The terminal group of nodes receives all the lymph vessels of the head and neck, either directly or indirectly, via one of the regional groups. The terminal group is closely related to the carotid sheath and, in particular, to the internal jugular vein, and is referred to as the deep cervical group. Important lymph nodes for consideration by dentists are outlined below.

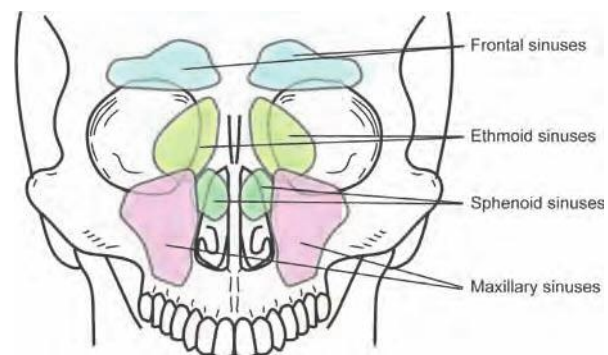


Fig. 29.2 Paranasal sinuses have four subgroups: the frontal sinuses, the ethmoid sinuses, the sphenoid sinuses, and the maxillary sinuses.

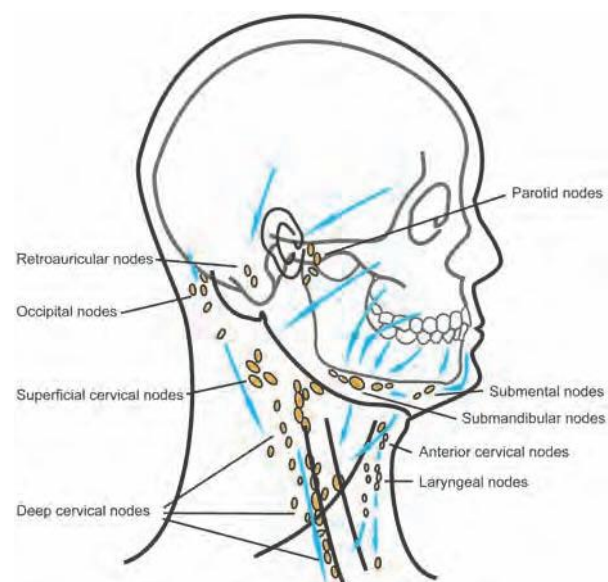


Fig. 29.3 Anatomic location of lymph nodes and lymph drainage of the head and neck.

Submandibular lymph nodes

These lymph nodes lie superficially to the submandibular salivary gland, just below the lower border of the body of the mandible. When enlarged, these nodes are easily palpable. The nodes receive lymph from a wide area including the front of the scalp, the nose, the cheek, the upper and lower lips (except the center part of the lower lip), the frontal, maxillary, ethmoid sinuses, the upper and lower teeth (except the lower incisors) and the associated gingiva, the floor of the mouth, and the vestibule. The efferent lymph vessels drain into the deep cervical lymph nodes.

Submental lymph nodes

Submental nodes are located just below the chin and above the body of the hyoid bone. When enlarged, these nodes are easily palpable. Submental nodes receive lymph from the tip of the tongue, the floor of the anterior part of the mouth, the incisor teeth and the associated gingiva, the center part of the lower lip and the skin over the chin. The efferent lymph vessels drain into the submandibular and deep cervical lymph nodes.

Deep cervical lymph nodes

The deep cervical lymph nodes form a vertical chain along the course of the internal jugular vein within the carotid sheath. When enlarged, these nodes are palpable. These lymph nodes receive lymph from neighboring structures and from all the other regional lymph nodes in the head and neck. The efferent lymph vessels from the deep cervical lymph nodes join to form the jugular trunk, which drains into the thoracic duct or the right lymphatic duct.

Microbiology

Numerous bacterial species survive within the body as commensal microorganisms. A commensal microorganism can be described as an organism that is often isolated from the body, but is rarely implicated in infection. It is not surprising therefore, that most commensal microorganisms have limited virulence. Within the community of the commensal flora, bacterial interactions will occur and a degree of stability in its composition will develop. Some microorganisms promote the growth and survival of other members by an inhibition of the host defense mechanisms, the provision of essential nutrients, and the modulation of the local environment to one more conducive to the survival of other organisms.^{1,7} On the other hand, the microflora can also exhibit microbial antagonism that suppresses the growth of its members through competition for essential nutrients and production of inhibitory substances. Microbial antagonism results in the prevention of overgrowth of potentially pathogenic microorganisms and the invasion of exogenous pathogens into the body. On occasions, however, members of the commensal microflora can be involved

in infections. Although the majority of infections involving commensal microorganisms are opportunistic, some infections, including odontogenic infections and sinusitis, occur even in individuals whose immunity is not significantly impaired.

The composition and features of the microflora vary between anatomical sites and the microbiology of the oral cavity, skin, and paranasal sinus is discussed separately.

Microbiology of the oral cavity

Over 500 bacterial taxa, several fungal species, some protozoan genera, and many viruses have been demonstrated to reside within the mouth, whilst numerous others are believed to remain undetected.⁸

Bacteria

Various host factors, including age, the presence or absence of teeth or prosthesis, periodontal status, medicine intake, systemic diseases, and lifestyle habits, can all influence the composition of the oral bacterial microflora.¹ Different sites in the oral cavity of one individual are also frequently colonized by different and distinct microflora.

Bacteria are by far the most predominant type of microorganism in the human mouth. Bacterial genera that are commonly found in the mouth are presented in Table 29.1. Members of viridans streptococci are generally the most predominant bacteria in the mouth, although the exact type varies with the intra-oral location.¹ Examples of viridans streptococci include *Streptococcus sanguinis*, *S. salivarius*, *S. mitis*, and the "*S. anginosus (milleri)* group". It should be noted that the genus *Streptococcus* is a very heterogeneous group of bacteria. *Streptococcus pneumoniae* and *Streptococcus pyogenes* are well known streptococcal pathogens of human infectious diseases. However, the pathogenicity and the antimicrobial susceptibility profiles of viridans streptococci are different from those of other streptococci.⁹

The oral environment of the neonate tends to favor aerobic and facultatively anaerobic bacteria.¹ Strictly anaerobic bacteria establish themselves with the formation of the gingival sulcus that results from tooth eruption. As sites with more anaerobic conditions appear, the number of strict anaerobes increases. In the case of strictly anaerobic bacteria, members of the genera *Fusobacterium*, *Porphyromonas*, *Prevotella*, *Veillonella*, *Actinomyces*, *Eubacterium*, and *Peptostreptococcus* are commonly recovered from the mouth. Some *Bacteroides* species including *B. forsythus* (*Tannerella forsythensis*), *B. ureolyticus*, and *B. distasonis* are also sometimes detected. These species have different pathogenic features and antimicrobial susceptibility profiles compared with the *Bacteroides fragilis* group. Although *Porphyromonas* and *Prevotella* species were formerly classified as *Bacteroides* species, these species are not bile-resistant organisms like *B. fragilis*, and their pathogenicity and antimicrobial

Table 29.1 Principal microbial genera/groups of the commensal human oral microflora.

Aerobic and facultative bacteria	Anaerobic bacteria	Fungi
<i>Viridans streptococcus</i>	<i>Peptostreptococcus</i>	<i>Candida</i>
<i>Staphylococcus</i>	<i>Actinomyces</i>	<i>Aspergillus</i>
<i>Corynebacterium</i>	<i>Bifidobacterium</i>	<i>Geotrichum</i>
<i>Enterococcus</i>	<i>Eubacterium</i>	<i>Hemispora</i>
<i>Micrococcus</i>	<i>Lactobacillus</i>	<i>Penicillium</i>
<i>Neisseria</i>	<i>Propionibacterium</i>	<i>Scopulariopsis</i>
<i>Campylobacter</i>	<i>Veillonella</i>	<i>Rhodotulula</i>
<i>Pseudomonas</i>	<i>Prevotella</i>	<i>Saccharomyces</i>
<i>Moraxella</i>	<i>Porphyromonas</i>	
<i>Enterobacter</i>	<i>Fusobacterium</i>	
<i>Eikenella</i>	<i>Bacteroides</i>	
<i>Rothia</i>	<i>Leptotrichia</i>	
<i>Haemophilus</i>	<i>Selenomonas</i>	
<i>Capnocytophaga</i>	<i>Centipeda</i>	
	<i>Tanarella</i>	
	<i>Treponema</i>	
	<i>Aggregatibacter</i>	

susceptibility patterns are different from those of the *B. fragilis* group.

Since the subgingival environment is anaerobic, it will support the growth of strictly anaerobic microorganisms. The composition and bacterial quantity of the subgingival microflora will vary depending upon periodontal status.¹ Gram-positive cocci are predominant in the initial stage of periodontitis. Spirochetes appear as the condition develops and the proportion of strictly anaerobic Gram-negative bacilli also frequently increases. Most of the spirochetes detected belong to the genus *Treponema*, although numerous uncultivated species exist. The favored habitats of the oral *Treponema* species are the periodontal pockets.

Dental plaque is a biofilm that builds up on the teeth and consists of bacteria and their products all in close proximity to the surface of the teeth (Fig. 29.4). It is important to recognize that it is the bacteria in the plaque and not the saliva that cause dental caries and periodontal diseases. Since a biofilm structure affords protection to bacteria from antimicrobials and antibiotics, the effectiveness of administered antimicrobial mouthwashes can be diminished. Therefore, as is the case with other types of biofilms, the physical or mechanical removal (tooth-brushing and flossing) often represents the most effective way to control dental plaque. Although various bacterial species are present in dental plaque, the predominant type of bacteria varies depending on the intraoral location and the maturity level of the dental plaque. The proportion of oxygen-tolerant bacteria is higher in supra-gingival plaque than in subgingival plaque. In contrast, strict anaerobes are predominant in subgingival plaque. Plaque can calcify through the presence

**Fig. 29.4** Dental plaque.

of calcium and phosphorus in the saliva and the resulting calcified deposit is called calculus, within which there are no living microorganisms.

Yeasts

Up to 80% of healthy individuals harbor yeasts in their mouths.¹ The prevalence of yeast carriage and the actual numbers of yeast generally increase with patient age, although high numbers of yeast are also readily detected in the mouths of infants due to their relatively immature immune system.¹⁰ Colonization rates and quantity of yeasts are influenced by various factors.¹¹ Depression in normal host defense levels is undoubtedly a major factor. Oral status, denture wearing, reduction in saliva production, receipt of systemic antibiotics, and antifungal agents can also all influence the likelihood of yeast carriage and level.

Candida species, and in particular *Candida albicans*, are the predominant oral yeasts.^{9,11} In general, *C. albicans* has a higher pathogenic potential compared with other *Candida* species. Importantly, *C. albicans* is normally susceptible to most antifungal agents that are currently administered.¹² The majority of non-*albicans* *Candida* exhibit a lower degree of virulence. However, some species including *Candida glabrata* and *Candida krusei* can be highly resistant to antifungal agents, especially those belonging to the azole class of antifungals.^{12,13} Fungal infection by *Candida* is a classic example of human opportunistic infection. Fungal infections, including oral candidosis, do not occur in the vast majority of yeast carriers even if their yeast levels are elevated. However, if an imbalance between the host defense levels and *Candida* virulence expression occurs, then fungal infections may develop.

Viruses

Although virology of the oral cavity has not been fully clarified, most viruses are thought to be oral transients, present only during active infections or during asymptomatic carrier states. It is the herpes group (Herpesviridae family) that is most frequently associated with the human oral diseases. These viruses include human herpes simplex virus (HSV), cytomegalovirus, varicella zoster virus (VZV), and Epstein-Barr virus (EBV).⁹

HSVs are classified either as HSV-1 or -2. HSV-1 can be involved in infection of oral mucous membranes (oral herpes), although it can also be a cause of genital herpes. HSV-2 is involved in infections of the genitalia and is rarely encountered in the mouth. The transmission of HSV usually results from direct contact with infected secretions from the carriers. The prevalence of HSV-1 infection increases from childhood, and reaches 44–85% in later adult years.¹⁴ Its initial primary infection is followed by a latent infection of neuronal cells in the dorsal root ganglia, and subsequent viral reactivation is accompanied by viral excretion from the original mucocutaneous site of infection, with or without the concomitant appearance of clinical signs and symptoms. The secondary and recurrent form of HSV-1 oral infection is herpes labialis, more commonly referred to as cold sores.

VZV is the virus involved in chicken pox (varicella) and shingles (zoster). Normally, this virus is not detectable in the mouth. However, during infection, varicella zoster virus may be detected in the oral environment, and can even be detected after clinical symptoms have resolved. Significantly and in a similar fashion to HSV, VZV remains dormant in the nervous system of the host in the trigeminal and dorsal root ganglia. In some cases, VZV reactivates later in life producing a disease known as herpes zoster or shingles.

EBV is one of the most common viruses of humans, and is believed to be acquired via saliva during childhood. Individuals infected with EBV often become asymptomatic carriers throughout their lifetime. In certain individuals, EBV can however be involved in several diseases including infectious mononucleosis, lymphomas, and cancers.⁹

Microbiology of the skin

The surface of the skin itself comprises of several distinct environments. Some areas, such as the axilla and the perineum provide moist environments, which allow colonization by a large number of microorganisms. However, the majority of the skin's surface tends to be dry and, generally, the overall quantity of bacteria on the skin surface is much lower than in the mouth. Aerobes and facultative bacteria, including *Staphylococcus epidermidis* and *Corynebacterium* species, are predominant, whilst some strict anaerobes, in particular *Propionibacterium* species, are also found colonizing the skin (Table 29.2).^{7,9,15}

Microbiology of the paranasal sinuses

The microbial flora of the paranasal sinuses commonly includes *Corynebacterium* species, *Haemophilus influenzae*, *Haemophilus parainfluenzae*, staphylococci, viridans streptococci, *Moraxella* species, and *Streptococcus pneumoniae* (Table 29.3).^{15,16} In addition, strict anaerobes including *Prevotella*, *Fusobacterium*, and *Propi-*

Table 29.2 The principal genera of commensal skin microbial flora.

Aerobic and facultative bacteria	Anaerobic bacteria	Fungi
<i>Staphylococcus</i>	<i>Peptostreptococcus</i>	<i>Candida</i>
<i>Streptococcus</i>	<i>Propionibacterium</i>	<i>Absidia</i>
<i>Micrococcus</i>	<i>Lactobacillus</i>	<i>Mucor</i>
<i>Enterococcus</i>	<i>Prevotella</i>	<i>Malassezia</i>
<i>Corynebacterium</i>		
<i>Serratia</i>		
<i>Aerococcus</i>		
<i>Enterobacter</i>		
<i>Acinetobacter</i>		
<i>Pseudomonas</i>		

Table 29.3 The principal members of the commensal sinus microbial flora.

Aerobic and facultative bacteria	Anaerobic bacteria
Viridans streptococcus	<i>Peptostreptococcus</i>
<i>Streptococcus pneumoniae</i>	<i>Propionibacterium</i>
<i>Micrococcus</i>	<i>Prevotella</i>
<i>Staphylococcus</i>	<i>Fusobacterium</i>
<i>Corynebacterium</i>	
<i>Neisseria</i>	
<i>Moraxella</i>	
<i>Haemophilus</i>	

onibacterium species can also be recovered from non-inflamed maxillary sinuses.

Immunity and inflammation

Host defense systems

Although host defense systems have not been fully elucidated, our understanding of host immunology has increased greatly in recent years. In this section, the general concepts of host defense systems are concisely described.^{1,3,7,17–19}

Local barriers against infection

Skin, mucous membranes, saliva, and the commensal microbial flora act as local defense systems, and provide the first barriers against pathogens through mechanical, chemical, and biological processes. The epithelial lining is the principal mechanical barrier at all body sites exposed to the external environment.

Dryness may limit the overgrowth of certain microorganisms on the skin, whilst lactic acid and fatty acids in sweat and sebaceous secretions, and their low pH, can inhibit the long-term survival of bacteria. Saliva and mucus not only remove microbes through a mechanical flushing effect, but also contain antimicrobial peptides such as lactoferrin, lysozyme, and immunoglobulin A. The resident microbial flora serve as biological barriers to incoming microorganisms. The normal microflora exhibit an inherent

microbial antagonism that suppresses the growth of many potentially pathogenic microorganisms by competition for essential nutrients and the production of inhibitory substances.

Immune system

Once a pathogen breaches the primary barriers and invades into the body, the immune system responds immediately. The most important role of the immune system is to distinguish between “self” and “non-self” molecules that have entered into the body, and to trigger a series of immune responses. A molecule that stimulates an immune response is called an antigen. Proteins and polysaccharides that are constituents of a microorganism, such as surface coats, capsules, cell walls, flagella, and toxins of bacteria and viruses, are representatives of antigens, as are non-microbial exogenous substances, such as pollen, proteins contained in food, and transplanted tissues.

Various types of host cells and proteins are involved in the immune system. The immune system can be divided into humoral and cell-mediated immunity in terms of the components, but in practice there is substantial overlap.

- *Cell-mediated immunity.* Cell-mediated (cellular) immunity is defined as the protective function of immunization associated with cells. Cell-mediated immunity primarily involves leukocytes (white blood cells). The leukocytes consist of neutrophils,

eosinophils, basophils, monocytes (macrophages), and lymphocytes (B-cells, T-cells, and natural killer cells) (Table 29.4). Macrophages are monocytes that migrate from the bloodstream to the tissues, and are present throughout the body. Dendritic cells, which are derived from the bone marrow, are also involved in cell-mediated immunity where they play a key role in the processing and presentation of certain antigens.

- *Humoral immunity.* The immune system is the protective function of immunization mediated by protein components in the cell-free body fluid or serum. The complement proteins are also prime components of humoral immunity. Antibodies are produced by B-cells mediated by signals from T-cells, and bind specifically to certain antigens.
- *Complement.* The complement system is a biochemical cascade that helps clear pathogens from an infected individual. This system consists of a group of more than 20 plasma proteins and protein fragments, which normally circulate as inactive zymogens. Many kinds of microbes directly activate the complement system through the so-called alternative pathway. This pathway contrasts with complement activation mediated by antibody–antigen interaction, and this latter type of reaction is referred to as the classical pathway. Activation of the complement system is a cascade reaction. Similar to the coagulation cascade, one component of the reaction triggers off the activa-

Table 29.4 The subtypes of human leukocytes and their functions.

Subtypes	% in blood	Functions/properties
Neutrophils	52–64%	Phagocytosis Initial responders to microbial infection Also called polymorphonuclear leukocytes (multi-lobed nucleus which appear like multiple nuclei) Short-lived cells Dead neutrophils are components of pus
Eosinophils	0–3%	Involved in protecting against parasitic infections and allergic reactions
Basophils	0–1%	Involved in allergic response by releasing the chemical histamine
Lymphocytes	30–40%	Divided into three types: B-cells: B-cells are produced and mature in the bone marrow. B-cells produce antibodies T-cells: T-cells mature in the thymus Helper T-cells manage and control immune responses Cytotoxic T-cells eliminate virus-infected and tumor cells NK cells: NK cells patrol throughout the body and combat tumor cells and cells infected by viruses
Monocytes	1–9%	Monocytes (macrophages) are located in the blood and become tissue macrophages after migrating from the bloodstream Phagocytosis Remove dead cell debris Present processed antigens of pathogens to T-cells (acts as activators of adaptive immune systems) Unlike neutrophils, macrophages have a longer active life

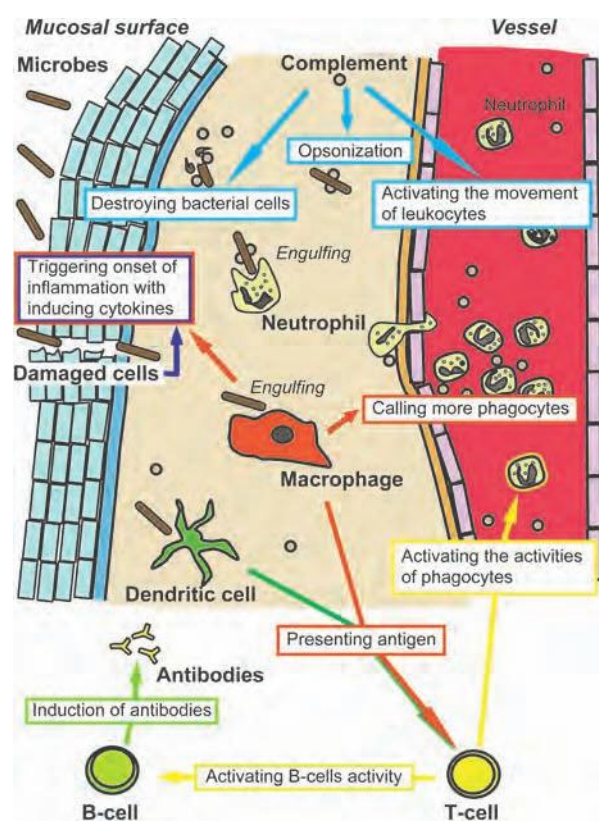
Table 29.5 Summary of differences between innate and adaptive immunity.

	Innate immunity	Adaptive immunity
Specificity of reaction	Non-specific	Specific for each pathogen
Onset of response	Immediate after antigen exposure	Lag time between exposure and maximal response
Involved components	Cellular and humoral components	Cellular and humoral components
Immunological memory	No	Yes

tion of subsequent members in the complement pathway. Unlike antibody, binding of complement to antigen is a non-specific reaction.

All components of the immune system are associated with the recognition of antigen, presentation of antigen information to other components of the immune system, and the destruction and elimination of the pathogen. The actions of the immune response do not occur independently and close interaction between processes takes place.

The immune system can be divided into innate and adaptive immunity (Table 29.5 and Fig. 29.5). It is the innate immunity that first responds and combats a pathogen that has invaded the body. Innate immunity does not require previous exposure to a pathogen. Activation of adaptive immunity follows innate immunity, its effect is more specific to the targeted pathogen, and often “memory” of the pathogen occurs following a successful adaptive response.

**Fig. 29.5** Mechanism of immune response.

Innate immunity

The innate immune response occurs immediately after pathogens enter the body and is not specific to any particular pathogen. Innate immunity requires no previous exposure to a pathogenic organism. The major roles of the innate immune response are the elimination of pathogens and the activation of adaptive immunity. Macrophages, neutrophils, natural killer cells, dendritic cells, and the complement system play a significant role in innate immunity.

The most important role of the cellular components in innate immunity is phagocytosis. Phagocytes are cells that have the ability to phagocytose microorganisms or other cellular targets. Key phagocytotic cells are macrophages, neutrophils, and dendritic cells. Phagocytes normally patrol the body searching for pathogens. During infection, these cells are recruited to the site of pathogen invasion.

When tissues have been damaged by microbes or toxins produced by microbes, various cells including macrophages present at the infection site release cytokines, which are soluble proteins and peptides that mediate communication between different types of cells. Cytokines trigger the onset of inflammation (the pathology of inflammation is described later). Initially, circulatory disturbance and exudation of plasma and cellular components from blood vessels in the infected tissue occur. Phagocytes travel to the infection site in response to the release of cytokines by host cells. During the initial stages, neutrophils, which are the predominant subtype of leukocytes, are the primary phagocytes. These cells first attach to the endothelial lining of the blood vessel and, by a process referred to as diapedesis, exude from the vessel, migrating towards the inflammation site. Phagocytes are short-lived cells and will soon die after engulfing and killing pathogens. The dead neutrophils are the major constituents of pus.

The complement system is the humoral component of innate immunity. It has three major roles: (1) complement proteins adhere to the surface of microbes (antigen) to enhance leukocyte phagocytosis, a process called opsonization; (2) the complement system activates the movement of leukocytes to the infection site; (3) complement directly damages the pathogens through contact with their cell surface.

Natural killer cells are cytotoxic and large granular lymphocytes with a characteristic morphology and cytotoxic functional role. Natural killer cells normally patrol throughout the body and once they

encounter tumor cells and cells infected by viruses, the natural killer cells destroy the target cells by releasing small cytoplasmic granules of proteins which trigger apoptotic killing. Therefore, natural killer cells play a major role in the prevention of tumor development and spread of viral infection.

The presentation of antigen to lymphocytes is another major role of innate immunity. Macrophages, dendritic cells, and cytokines are primarily associated with this role. These cells present antigen to lymphocytes (T-cells), which are key cells of the adaptive immune system. Although innate immunity itself plays an important role in defense, it therefore also acts as an activator of adaptive immune systems.

Adaptive immunity

When innate immunity is insufficient to defend against invasion of pathogens, the adaptive immune system, which is a specific and strongly defensive reaction, is triggered. There is generally a time lag between exposure and maximal response, because of the requirement to be activated by the innate immune system. The major functions of the adaptive immune system include the recognition of specific (non-self) antigens during the process of antigen presentation, the generation of responses that are tailored to maximally eliminate specific pathogens or infected cells, and the development of immunological memory, which allows rapid elimination of the pathogen during subsequent exposure to the organism.

Lymphocytes are a key subtype of leukocyte cells involved in the adaptive immune system. The peripheral blood contains 20–50% of all circulating lymphocytes, and the rest circulate within the lymphatic system. B-cells and T-cells are subclasses of lymphocytes. B-cells are produced and matured by stem cells in the bone marrow; whilst T-cells travel from the bone marrow to the thymus where they mature. Immature T-cells develop into several mature types when the adaptive immune system is triggered. Helper T-cells (CD4+ lymphocytes), play an important role in establishing and maximizing the capabilities of the adaptive immune response. These cells lack cytotoxic or phagocytic activity so they do not kill either infected cells or eliminate pathogens directly. However, helper T-cells manage and control the immune response by directing other cells to perform these tasks. Once helper T-cells have received and recognized the antigens, they secrete cytokines that induce activation of many types of cells associated with host defense, including macrophages. The cytotoxic T-cell (killer T-cell; CD8+ lymphocyte) is another major type of mature T-cell. Cytotoxic T-cells perform the function of eliminating cells infected with viruses, and damaged or dysfunctional cells.

B-cells are involved in the production of antibodies that circulate in blood plasma and lymph, and play an essential role in humoral immunity. Antibodies (immunoglobulin, Ig) are large Y-shaped proteins that serve to identify and neutralize foreign

antigens. There are five basic types of antibody (IgA, IgD, IgE, IgG, and IgM), which can be distinguished by biological properties. B-cells identify pathogens when the cell's surface antibodies bind to the specific target antigen. The antigen–antibody complex is processed into peptides and the B-cells subsequently display these antigenic peptides on their surface to attract helper T-cells. The helper T-cells release cytokines (lymphokines) and activate the B-cells, and, as a result, enormous amounts of the specific and required antibody are produced. The antibodies bind to pathogens expressing the antigen and “mark” them for destruction by complement activation or for specific destruction by phagocytes. Antibodies also neutralize challenges directly by binding to bacterial toxins or by interfering with the receptors that pathogens use to infect cells.

Some members of B-cells and T-cells ultimately mature into memory cells. These memory cells form a database of effective B- and T-lymphocytes; upon interaction with a previously encountered antigen, the appropriate memory cells are selected and activated. Consequently a stronger immune response can be produced quickly during second and subsequent exposures to that particular antigen.

Compromised host defenses and association with infection

As described earlier, infections in oral and maxillofacial regions are opportunistic. A compromised host defense increases the likelihood of infection, and provides an elevated risk of extensive progression and a probable prolonged period before resolution. Various congenital and acquired factors can compromise the host defense mechanism. Bacterial infection is generally associated with cellular and/or humoral immunity defects, whilst fungal and viral infections are thought to largely relate to cellular immunity defects.

Inflammation

Pathology of inflammation

It is widely accepted that inflammation is one of the manifestations associated with the host response to maintain biological homeostasis against stress. A variety of factors, including microbial, physical, and chemical stimuli, can be the origin of this stress. In oral and maxillofacial infections, microbes or their toxins initiate a series of inflammatory reactions. Although the inflammation itself is protective for the host, it sometimes acts harmfully or leads to unwanted damage to the host. Swelling, redness, pain, heat, and loss of function are all classic signs of inflammation. Inflammation that is short term is described as an acute inflammation, whilst a prolonged period of inflammation is referred to as chronic inflammation.³

Once inflammation has been triggered by certain harmful stresses, circulatory disturbance will ensue. As a consequence, the dilation of blood vessels (vasodilation) and congestion occurs following the initial

constriction of the vascular smooth muscle. Moreover, alteration of endothelium results in elevated vascular permeability (vasopermeability). Vasopermeability leads to exudation of plasma from blood vessels and a resulting increase in blood viscosity. The increase in vasopermeability allows the exudation of plasma containing albumins, globulins, and fibrinogen, and the extravasation of leukocytes (mainly polymorphonuclear leukocytes) through the endothelium and basement membrane of the blood vessel into the tissue at the infected site. Exudation of plasma fluid induces inflammatory swelling (edema). The leukocytes migrate along a chemotactic gradient to reach the target foreign bodies, such as microbes, involved in the infection. The majority of exudated leukocytes in the initial stages of inflammation are neutrophils, and macrophages and lymphocytes emerge relatively later.

Through a series of reactions, a variety of biochemical cascade systems associated with inflammatory mediators (e.g. histamine, serotonin, prostaglandin, leukotriene, and bradykinin) act to propagate and mature the inflammatory response. These cascade reactions include the complement, coagulation, and fibrinolysis systems. The mediators are derived from plasma substances or directly from cells, and some of them are involved in the onset of pain.

The final act of the inflammatory process is proliferation of cells at the injured site. Monocytes, lymphocytes, fibrocytes, and vascular endothelial cells are recruited to the site of tissue damage where they play an important role in angiogenesis and tissue repair with the formation of granulation tissue.

Removal of the cause of injury generally halts the response of the inflammatory mechanisms. Acute inflammation generally terminates without obvious proliferation of cells. However, in cases where inflammation fails to restore homeostasis, chronic inflammation will ensue. Chronic inflammation is characterized by simultaneous destruction and healing of the tissue.

Clinical signs of inflammation

Most symptoms of oral and maxillofacial infections result directly from the inflammatory reaction.

Local signs

The classic signs of inflammation include swelling, redness, pain, heat, and loss of function (dysfunction) (Table 29.6). Swelling results from circulatory disturbance (edema) and in the case of chronic inflammation, proliferation of cells may be also associated. The redness and heat result from the circulatory disturbance, in particular congestion. Several factors are related to the onset of pain. Some inflammatory mediators, such as prostaglandin, change in tissue pH, and physical stress to nerves resulting from swelling may be involved. In the case of bacterial infection, intensity of pain generally depends on the pressure of fluid within the tissue. Pain and swelling

Table 29.6 The relationship between classic signs and symptoms of acute inflammation and pathological change.

Signs and symptoms	Association with pathological change
Redness	Circulatory disturbance (congestion)
Heat	Circulatory disturbance (congestion)
Swelling	Circulatory disturbance (edema), proliferation of cells
Pain	Release of inflammatory mediators, change in pH of tissue, physical stress to nerve
Function loss	Pain and swelling, inappropriate proliferation of cells, tissue destruction

are major causes of dysfunction, although inappropriate proliferation of cells and tissue destruction resulting from the inflammatory reaction can be involved in loss of function. Trismus is the term describing inability to open the mouth normally. It is the most common oral dysfunction and is largely due to inflammation of the masseter and other masticatory muscles.

Systemic signs

Infection affects the host systemically and results in the expression of various systemic reactions. Fever, an increase in pulse rate, and fluctuation in hematological components are prime examples of subsequent systemic responses to infection.

- **Fever.** Fever is defined as an increase in internal body temperature to levels that are above normal, and often results from the systemic effects of inflammation. Variations in normal body temperature between individuals are not uncommon. Various non-pathogenic factors, including the time of day, location, situation within which the temperature is being measured, age, gender, and the stage of menstrual cycle, may also influence body temperature. Body temperature is also different at various sites within the same individual and is most frequently measured within the mouth, axilla or rectum. Fever results from an elevated thermoregulatory set-point regulated in the hypothalamus. A substance that induces fever is called a pyrogen. Pyrogenic agents may be endogenous or exogenous.^{3,7} The lipopolysaccharide (LPS; endotoxin) that is a cell wall component of Gram-negative bacteria is representative of a typical exogenous pyrogen. The LPS molecule may directly affect the hypothalamic thermoregulatory center. With regards to endogenous pyrogen, some cytokines including interleukin (IL)-1 and IL-6 that are released by macrophages following exposure to bacterial endotoxin, viruses or lymphocyte products stimulate prostaglandin E₂ (PGE₂) synthesis in the hypothalamic thermoregulatory center.⁷ As a result, the thermoregulatory center that controls

body temperature is altered. The majority of non-steroidal anti-inflammatory drugs block the fever response by inhibiting cytokine (IL-1)-stimulated PGE₂ synthesis in the hypothalamus.

- **Elevation in pulse rate.** A normal pulse rate for a healthy adult under resting conditions is around 60 beats per minute, although it ranges widely between individuals and fluctuates due to various non-pathogenic factors. The pulse rate in patients with infection or inflammation is generally seen to increase and is also associated with an elevation in body temperature.
- **Fluctuation in haematological data.** Infection may influence various components of the blood:
 - **Leukocytes.** Leukocytes, especially neutrophils, play a prime role in the elimination of causative agents of infection. The bone marrow supplies large numbers of these cells to combat infection. An elevation in the blood leukocyte count (leukocytosis) is frequently seen in cases of bacterial infection and tissue injury, and is triggered by specific mediators (IL-1 and tumor necrosis factor (TNF)- α) released by macrophages and other cells. In the case of more severe infection, an increase in immature neutrophils, which results from irritation of the bone marrow with regeneration or insufficient response of the bone marrow to the host's demand on production of more leukocytes, may be observed. This is referred to as a left shift (Fig. 29.6).
 - **Serum protein.** In the initial stage of infection, IgM production may be promoted, and subsequently, levels of IgG and IgA may be elevated. C-reactive protein (CRP) is produced by the liver in the acute phase of an infection.²⁰ CRP was originally discovered as a substance bind-

ing the C polysaccharide of *Streptococcus pneumoniae*. Although its function has not been fully elucidated, it is thought to play an important role in immune systems.²¹ As CRP levels rise during inflammatory processes, it is readily detectable for diagnostic purposes.

- **Erythrocytes.** Notable fluctuation in the erythrocyte count does not occur during infection. The erythrocyte sedimentation rate (ESR) is the rate at which erythrocytes fall when anticoagulated blood is placed in an upright tube. The ESR is measured in mm/h and generally increases in the presence of infection or inflammation. The effect probably arises due to an elevated production of fibrinogen and an increase in blood viscosity as a result of inflammation.

Pharmacology

Various kinds of medicines may be used for management of oral and maxillofacial infections: this section discusses antibiotics, antifungals, antivirals, and analgesics.

Antibiotics

Antibiotics are substances produced by various species of microorganisms (bacteria and fungi) that suppress the growth of other microorganisms and eventually may destroy them. However, common usage often extends the term "antibiotics" to include synthetic antibacterial agents, such as the sulfonamides and quinolones that are not the products of microbes. It is important to recognize that antibiotics work selectively against microorganisms, and that they are different from disinfectants and antiseptics

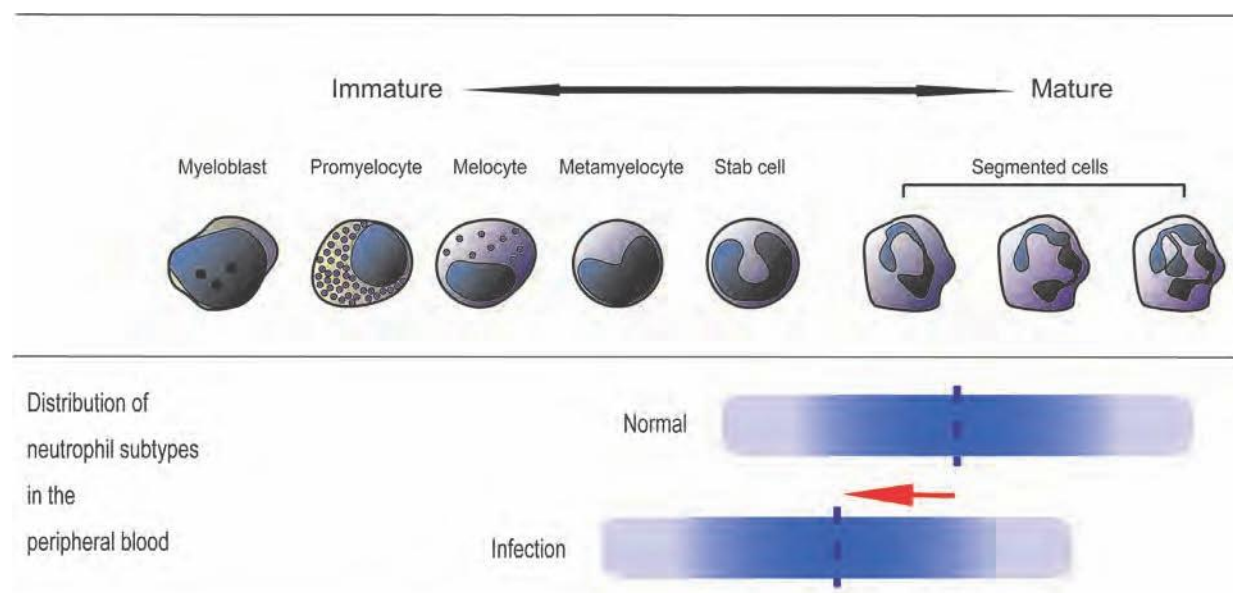


Fig. 29.6 An increase in the number of immature neutrophil band cells in the peripheral blood, is commonly seen in the course of an active infection. This is because of an insufficient response of the bone marrow to the host's demand on production of more leukocytes.

which can have a high toxicity both to human cells and the microorganism.

General considerations

Antibiotics are classified into bactericidal and bacteriostatic agents based upon their antimicrobial properties. Bactericidal agents directly kill bacteria, whilst bacteriostatic agents interfere with reproduction of bacteria and subsequently it is the host immune system that eliminates the bacteria.²² In general, the activity of antibiotics that inhibit cell wall synthesis by bacteria is bactericidal. Although the majority of antibiotics inhibiting synthesis of bacterial proteins have bacteriostatic activity, some do exhibit bactericidal activity (Table 29.7). Although use of bactericidal agents has been recommended for treatment of acute infections in oral and maxillofacial regions, their clinical effectiveness is not notably different from that of bacteriostatic agents when administered to patients with normal host defense mechanisms.

The *in vitro* activity of antibiotics can be expressed using the minimal inhibitory concentration (MIC) determined against target bacteria. An antibiotic that exhibits a low MIC is generally regarded to be effective against the target organism, as the growth of bacteria should be inhibited by low antibiotic concentrations. The pharmacological properties of antibiotics are also important, however. The antibiotic concentration achieved at the infected site must exceed the MIC for the target bacteria but not cause significant toxicity to the host. Antimicrobial agents can be divided into two main groups, based on antimicrobial pharmacodynamic characteristics: (1) agents that exhibit time-dependent bactericidal activity that

have little relationship to the magnitude of the drug concentrations, as long as the concentrations are above a minimally effective level (e.g. β -lactam antibiotics, macrolides, tetracyclines, and vancomycin); (2) agents that exhibit concentration-dependent bactericidal activity over a wide range of drug concentrations (e.g. aminoglycosides and fluoroquinolones). Some kinds of antibiotics have prolonged persistent effects (postantibiotic effect) in which the bactericidal action continues for a period of time after the antibiotic level falls below the MIC. Time-dependent antibiotics tend to have minimal to no postantibiotic effect, whilst concentration-dependent antibiotics usually have an associated dose-dependent postantibiotic effect. However, although the antimicrobial action of macrolides and tetracyclines is time-dependent, these antibiotics also have concentration-dependent features because of the occurrence of a relatively large postantibiotic effect.^{22,24}

The intervals for drug administration are determined in accordance with drug metabolism and pharmacokinetics. For concentration-dependent antibiotics, a high once-daily dose could be the best way to eradicate pathogens unless it causes significant toxicity. In contrast, in the case of time-dependent antibiotics, it is necessary to maintain the serum concentration above the MIC for at least 40–50% of the dosage interval.²⁵

Classes and characters of antibiotics

Antibiotics are usually classified based on chemical structure (Fig. 29.7). The following antibiotics are considered to have significance in the oral and maxillofacial surgical field.

Table 29.7 The properties of antibiotics that may be used in the management of oral and maxillofacial infections

Antibiotic	Antibacterial action	Pharmacodynamic category	Notable side-effects	FDA pregnancy risk categories*
Penicillins	Bactericidal	Time-dependent	Hypersensitivity	B
Cephalosporins	Bactericidal	Time-dependent	Hypersensitivity	B
Carbapenems	Bactericidal	Time-dependent	Hypersensitivity	B (Imipenem, C)
Macrolides	Bacteriostatic	Time-dependent	Drug interaction	B (Clarithromycin, C)
Clindamycin	Bacteriostatic	Time-dependent	Diarrhea, pseudomembranous colitis	B
Tetracyclines	Bacteriostatic	Time-dependent	Discoloration of teeth	D
Aminoglycosides	Bactericidal	Concentration-dependent	Nephrotoxicity, ototoxicity	D
Fluoroquinolones	Bactericidal	Concentration-dependent	Arthralgias, damage, drug interaction with NSAIDs	C
Glycopeptides	Bactericidal	Time-dependent	Nephrotoxicity	C
Metronidazole	Bactericidal	Concentration-dependent	Headache, nausea, disulfiram-like effect	B

* Pregnancy risk categories provide by the US Food and Drug Administration (FDA).²³

A, Controlled studies show no risk.

B, No evidence of risk in humans.

C, Risk cannot be ruled out.

D, Positive evidence of risk.

X, Contraindicated in pregnancy.

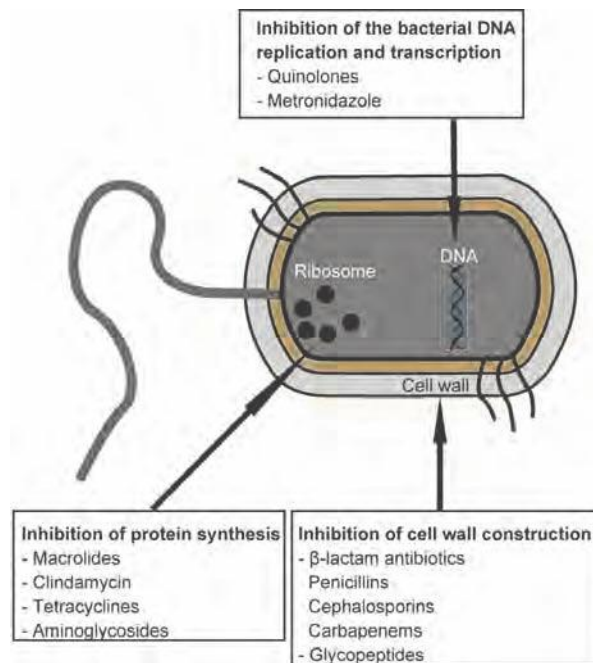


Fig. 29.7 Target sites of antibiotics.

β-lactams

β-lactam antibiotics have been widely used in the prevention and treatment of a variety of human infections. Although β-lactam antibiotics can be classified into penicillin, cephalosporin, carbapenem, and monobactam classes, all have a chemical structure called a β-lactam ring, and have bactericidal activity by binding to penicillin-binding proteins (PBP) and inhibiting synthesis of the bacterial peptidoglycan cell wall.^{7,26}

β-lactam antibiotics are generally regarded to be safe agents, because they target the cell wall in bacteria, which does not exist in human cells. However, hypersensitivity to antibiotics should be considered. The most likely form of hypersensitivity is a dermatological reaction. It should be noted that anaphylactic reaction is rare, but under certain conditions it can be serious and lead to a fatal hypersensitive reaction.

Bacterial resistance against the β-lactam antibiotics is increasing at a dramatic rate.^{27,28} The mechanisms of resistance include: (1) production of β-lactamases, which are bacterial enzymes that hydrolyse the β-lactam antibiotic; (2) expression of an altered PBP; (3) decreased antibiotic entry or active effluxing of the antibiotic from the bacterial cell.^{26,29}

- **Penicillin.** The penicillin class of antibiotics has a long history, and remains one of the most important antibiotics, although numerous other antimicrobial agents have been produced since the first penicillin was discovered in 1928 by Alexander Fleming. Members of the penicillin group have minimal direct toxicity. Hypersensitivity reactions are the most common adverse effects. Overall incidence of the reactions to penicillins varies from 0.7–10% in different studies. Hypersensitive reactions may occur with any dosage form of

penicillin. Penicillin G, penicillin V, ampicillin, and amoxicillin are commonly used for oral and maxillofacial infections, because of their historical effectiveness and minimal toxicity. Penicillin G and penicillin V have a narrower spectrum of activity than ampicillin and amoxicillin. The gastrointestinal absorption and peak serum level of amoxicillin are excellent, whilst the financial cost is higher compared with the other members.

- **Cephalosporin.** The cephalosporin class of antibiotics is another important group of β-lactam antibiotics. Some of the older antibiotics have a relatively narrow spectrum, but newer antibiotics generally possess a broader spectrum of activity and have higher stability against various types of β-lactamases. Cephalosporin antibiotics are classified into four generation classes.²⁶ The first-generation agents have good activity against Gram-positive bacteria such as streptococci and staphylococci and relatively modest activity against Gram-negative bacteria. This class includes cefazolin and cephalexin. The second-generation agents have somewhat increased activity against Gram-negative bacteria but have weaker activity against Gram-positive bacteria compared with the first-generation agents. Cefuroxime, cefaclor, cefpodoxime, and cefmetazole are good examples of such antibiotics. The third-generation cephalosporins are generally even less active against Gram-positive bacteria than the second-generation agents, whilst they are much more active against Gram-negative bacteria including some β-lactamase-producing organisms, such as *Pseudomonas aeruginosa* and *Enterobacter* species. Cefotaxime, ceftriaxone, cefoperazone, and cefdinir belong to the third-generation agents. Although the fourth-generation cephalosporin antibiotics have a similar spectrum to the third generation agents, these agents are more stable to some extended-spectrum β-lactamases. Cefepime is regarded as a fourth-generation agent. It is worth highlighting that although the stability of the third- and fourth-generation cephalosporin antibiotics is high against many types of β-lactamases, the β-lactamase produced by *Prevotella* species (a major pathogen of orofacial infections) hydrolyses most cephalosporin antibiotics, regardless of agent generation.^{30,31} The toxicity level of cephalosporin antibiotics is low. However, as with penicillin, hypersensitivity is the most commonly found side-effect, and the incidence of allergic reaction is reported to be 1–3% of patients who are exposed.³² The majority of allergic reactions to cephalosporins are rashes, although anaphylaxis can occur. Due to the similarity in structure of penicillin and cephalosporin groups, patients who are allergic to one class of these agents may manifest cross-reactivity when a member of the other class is administered. In general, the likelihood of cross-reactivity of the second-, third-, and fourth-generation

cephalosporins with penicillin is lower than that of the first-generation cephalosporins.³² It should be noted that concomitant penicillin and cephalosporin allergy is not always due to cross-reactivity, as penicillin-allergic patients have a three-fold higher rate of allergic reactions to any drug, even structurally unrelated agents.³² Therefore, the histories of "penicillin or cephalosporin allergy" reported by patients might not be actually due to immunologic drug-related side-effects. The increased risk of an allergic reaction to certain cephalosporins in penicillin-allergic patients may be smaller than previously postulated, and 5–10% of penicillin-allergic patients have allergy to cephalosporins.³³ There is controversy over clinical use of cephalosporin in penicillin-allergic patients. Evidence-based guidelines provided by the American Academy of Pediatrics, have recently endorsed the use of cephalosporin antibiotics for patients with reported allergies to penicillin, for the treatment of acute bacterial sinusitis and acute otitis media.³² Nevertheless, the use of cephalosporin antibiotics in patients with possible penicillin allergy requires careful consideration.

- **Carbapenems.** The members of the carbapenem group of antibiotics have an extremely broad spectrum of antimicrobial activity and are resistant to a variety of β -lactamases. Antimicrobial activity of carbapenem antibiotics is extremely high and the group is typified by imipenem and meropenem antibiotics. Both of these agents are administered parenterally. As carbapenem antibiotics are structurally related to penicillin, caution should be used when administering these agents to a penicillin-allergic patient. It has been reported that the incidence of carbapenem hypersensitivity among penicillin-allergic patients is 9–11%, which is similar to that reported for other β -lactam agents.^{34,35}
- **β -lactamase inhibitors.** β -lactamase inhibitors are substances that bind to β -lactamases and inactivate them. These inhibitors include clavulanic acid and sulbactam. β -lactamase inhibitors themselves have little direct antimicrobial activity, but when combined with an antibiotic, they extend the antibiotic's spectrum of activity and increase stability against β -lactamases.²⁶ Augmentin[®] is a product of amoxicillin combined with clavulanate, and Unasyn[®] is a combination of ampicillin and sulbactam. Although these antibiotics are not resistant to all types of β -lactamases, it has been demonstrated that they are effective against β -lactamase producing *Prevotella* strains involved in oral infections.³¹

Macrolides

Macrolide antibiotics have a common structure of a macrocyclic lactone ring to which are attached one or more deoxy sugars. Macrolide antibiotics are bacteriostatic agents that inhibit bacterial protein synthesis by binding reversibly to 50S ribosomal subunits of

sensitive microorganisms. Although the majority of bacteria involved in orofacial infections are susceptible to macrolide antibiotics, *in vitro* studies have demonstrated an increased prevalence of macrolide-resistant bacteria.^{36,37} Moreover, *Fusobacterium* species are highly resistant to macrolide antibiotics.^{37,38} On the other hand, the clinical response to macrolide antibiotics has generally been good and therefore these agents are often used for treatment of oral and maxillofacial infections. Erythromycin is the best known example of a macrolide, and clarithromycin and azithromycin are also available for treatment of orofacial bacterial infections.

Serious untoward effects are only rarely caused by erythromycin, although cholestatic hepatitis is the most striking side effect.²⁶ Other possible side-effects are hypersensitivity reactions, manifesting in the form of fever, eosinophilia, and skin eruptions. Macrolide antibiotics have a relatively high likelihood of drug interaction, because they are potent inhibitors of the cytochrome P450 system, which constitutes a complex set of drug-metabolizing enzymes.³⁹ Macrolide antibiotics have the potential to enhance the effects of various drugs (e.g. corticosteroids, carbamazepine, ciclosporin, digoxin, and warfarin).

As a unique pharmacokinetic property, azithromycin provides a much higher drug concentration in tissue (infection site) compared with the simultaneous serum concentration.⁴⁰ It has been suggested that fibroblasts serve as a reservoir of this drug in the tissue, permitting activity against the target organisms and possibly transferring antibiotic to phagocytes for activity against intracellular pathogens and delivery to infection sites.⁴¹ Azithromycin has an extremely long elimination half-life, which allows a large single dose to be administered, giving bacteriostatic levels in the infected tissue for several days.

Recently, the anti-inflammatory effects of 14-membered macrolides were discovered.⁴² An exploitation of this property involves a novel therapy, whereby a subantimicrobial dose of macrolide is used in the control of diffuse panbronchiolitis and management of chronic sinusitis in some countries.^{43,44}

Tetracyclines

The tetracyclines possess a wide range of antimicrobial activity against aerobic and anaerobic bacteria. The activity of these antibiotics is generally bacteriostatic by inhibition of bacterial protein synthesis through reversible binding to the 30S ribosomal subunit, thus blocking the interaction of aminoacyl-transfer ribonucleic acid with the ribosome.

Dairy products, antacids containing calcium, aluminium, zinc, magnesium or silicate, and vitamins with iron, can all interfere with the absorption of tetracyclines from the gastrointestinal tract when taken simultaneously with the drug.⁴⁵

Although gastrointestinal irritation, photosensitivity, hepatic toxicity, and renal toxicity are the notable side-effects, effects on teeth may, for

dentists, be the most recognizable side-effect caused by systemic administration of tetracyclines. Children receiving these agents may develop permanent brown discoloration of the teeth. If given to pregnant women, discoloration of the teeth may occur in their children. As a consequence, members of the tetracycline group should not be prescribed to pregnant women or children.

Interestingly, tetracycline antibiotics have non-antimicrobial properties including collagenase inhibition, anti-inflammatory actions, inhibition of bone resorption, and facilitating effect on fibroblastic growth.^{46,47} Some members of these agents are topically used as adjunctive treatment for periodontitis.

Clindamycin

Clindamycin binds exclusively to the 50S subunit of bacterial ribosomes and suppresses protein synthesis. Since clindamycin has excellent activity against viridians streptococci and strictly anaerobic bacteria, this agent is very useful in prevention and management of oral and maxillofacial infections. Clindamycin is well absorbed following oral administration and is widely distributed in many tissues including mandibular bones.⁴⁸

Clindamycin has sometimes been associated with causing diarrhea. The incidence of diarrhea following clindamycin treatment ranges from 2–20%.^{26,49,50} Pseudomembranous colitis is the most striking side-effect. This colitis is caused by the toxin from the organism *Clostridium difficile* that overgrows in the colon due to microbial substitution resulting from systemic antibiotic therapy.⁵¹ Pseudomembranous colitis is characterized by abdominal pain, diarrhea, fever, and mucus and blood in the stools, and can be severe, causing toxic megacolon, or even fatal. The incidence of this colitis following administration of clindamycin is reported to be 0.01–10%.^{26,52} Significantly, however, it has been reported that there is not a notable difference in the risk of the antibiotic-related colitis between clindamycin and β -lactam antibiotics.⁵² Skin rashes are reported to occur in approximately 10% of patients treated with clindamycin.²⁶

Aminoglycosides

Aminoglycosides are bactericidal inhibitors of protein synthesis, although the majority of bacterial protein synthesis inhibitors are bacteriostatic. These antibiotics include amikacin, gentamicin, kanamycin, streptomycin, and tobramycin. Aminoglycosides have a strong antimicrobial activity and are used primarily to treat infections caused by aerobic Gram-negative bacteria, including *Pseudomonas aeruginosa*, which tend to be resistant to multiple antibiotics. However, since the transport of aminoglycosides across the cytoplasmic membrane of bacteria is an oxygen-dependent active process, aminoglycosides have little activity against anaerobic bacteria because of the lack of the necessary transport system.^{7,26}

Aminoglycosides are relatively toxic compared with other classes of antibiotics. Nephrotoxicity and ototoxicity, that can involve both the auditory and vestibular functions of the eighth cranial nerve, are clinically notable and serious side-effects.

The quinolones

The older class of quinolones is of relatively minor interest now because of their limited antimicrobial spectrum and therapeutic use, and the rapid development of bacterial resistance. However, the introduction of fluorinate 4-quinolones has extended the antimicrobial activity and resulted in a particularly important therapeutic advance. The term quinolone is usually synonymous with fluoroquinolones.

Quinolones (fluoroquinolones) are bactericidal agents that inhibit bacterial DNA replication and transcription. As mentioned earlier, the quinolone antibiotics have an extremely broad antibacterial spectrum. These agents are well absorbed after oral administration and are widely distributed at various body sites.⁵³ The incidence of side-effects is relatively low. Nausea, abdominal discomfort, headache, and dizziness are the most common side-effects. Skin rashes including photosensitivity reactions can also occur. Arthralgias and joint damage can also develop in children receiving these agents.²⁶ Clinical use for prepubertal children and pregnant women should, therefore, be avoided. It has been reported that the risk of central nervous system side-effects, particularly convulsion, increases when receiving non-steroidal anti-inflammatory drugs (NSAIDs) during therapy with quinolones.⁵⁴ The relatively high cost of these agents may also limit clinical use in treatment of oral and maxillofacial infections.

Glycopeptides

A glycopeptide antibiotic is composed of glycosylated cyclic or polycyclic non-ribosomal peptides. Although several agents are currently available, vancomycin and teicoplanin are amongst the most important.

Glycopeptide antibiotics act primarily by inhibiting cell wall synthesis of Gram-positive bacteria, and this action is time-dependent. Vancomycin and teicoplanin exhibit strong antimicrobial activity against almost all types of Gram-positive organisms including methicillin-resistant *S. aureus*, although their spectra of activity are limited to Gram-positive organisms. This unique antimicrobial spectrum and also a level of high toxicity restrict the clinical use of glycopeptide antibiotics. Glycopeptide antibiotics are indicated for the treatment of serious, life-threatening infections by Gram-positive bacteria which are unresponsive to other less toxic antibiotics.

Glycopeptide antibiotics were historically regarded as the last effective line of defence. However, there are increasing reports of resistant enterococci strains.⁵⁵ Moreover, staphylococcal strains with high-level resistance to glycopeptides have increasingly been isolated from clinical specimens.⁵⁶

Nephrotoxicity is the most clinically important and serious adverse effect and its occurrence and severity are dependent on the dose. Therefore, measurement of the levels of the antibiotic in the plasma is required to monitor and reduce adverse events. Other possible adverse effects include anaphylaxis, toxic epidermal necrolysis, erythema multiforme, red man syndrome, super infection, neutropenia, leukopenia, thrombocytopenia, tinnitus, dizziness, and ototoxicity.

Metronidazole

Although metronidazole is used as an anti-protozoan agent, it is also available for the treatment of infections involving strictly anaerobic bacteria.²⁶ Metronidazole has a high antimicrobial activity against almost all of the strictly anaerobic bacteria, although this agent is not active against aerobes and facultative bacteria.⁵⁷ Therefore, metronidazole is effective for treatment of serious infections due to strict anaerobes. This agent is also a useful adjunct to antibiotics with an aerobic spectrum of activity (usually β -lactams and macrolide) in treatment of mixed aerobic (or facultative) and anaerobic infections or for empirical treatment of odontogenic infections.

Occurrences of serious side-effects are rare. The most common complications are headache, nausea, and metallic taste.²⁶ Vomiting, diarrhea, and abdominal distress occasionally occur. Metronidazole has a disulfiram-like effect, resulting in some patients experiencing abdominal distress, vomiting, flushing or headache if they drink alcohol during therapy. Patients should be cautioned against consuming alcohol during metronidazole treatment. There is conflicting evidence regarding possible teratogenic effects of metronidazole. The use of this agent during the first trimester of pregnancy should be avoided.

Antifungals

Antifungal agents are drugs that inhibit the growth of fungi. In contrast with bacteria, fungi are eukaryotic organisms and are therefore cytologically more similar to those of the host. As a consequence, there are limited numbers of drugs that affect fungal cells without a degree of toxicity to host cells. In general, more side-effects are likely to occur with antifungal agents compared with antibiotics.

Polyenes

Amphotericin B is the best known of the polyene antifungals. The mechanism of action of amphotericin B is fungal membrane disruption induced by the agent binding to a sterol moiety, primarily ergosterol, in the cell membrane of the fungi. Amphotericin B has a strong antifungal activity against a variety of fungal species. Absorption of amphotericin B from the gastrointestinal tract is negligible. Therefore intravenous administration of amphotericin B is employed in systemic therapy. The major acute reac-

tion to intravenous amphotericin B is fever and chills. Nephrotoxicity is another significant side-effect. Topical amphotericin B is a useful option for treatment of superficial candidosis and is considered safe.

Nystatin is another polyene antifungal agent that provides effective topical treatment of oral candidosis. It is usually administered in the form of oral suspension. Topical therapy with nystatin rarely causes side-effects.

Azoles

The azole agents are frequently used in the prevention and treatment of fungal infection because of their relatively broad antifungal spectrum of activity and lower toxicity compared with the polyene antifungals. These antifungals impair the synthesis of the fungal cell membrane by inhibition of sterol synthesis. The azole antifungals include two broad classes, imidazoles and triazoles. Miconazole is an imidazole agent that is widely used clinically, and fluconazole and itraconazole are triazoles. Although both classes share the same antifungal spectrum and mechanism of action, the systemic triazoles are more slowly metabolized and have lower toxicity to human cells compared with the imidazoles.

Fluconazole and itraconazole are administered systemically in the treatment of oral fungal infections. These agents are readily absorbed from the gastrointestinal tract. However, the absorption of itraconazole is greatly reduced in individuals who have reduced gastric acid production, so this agent should preferably be taken during or after meals.²⁶ Fluconazole and itraconazole are generally well tolerated. Gastrointestinal distress, nausea, vomiting, skin rash, and headache are major side-effects, but these can be managed with dose reduction. Importantly, these agents can interact with other systemically administered drugs. Miconazole is usually used topically which is considered safe, even during pregnancy.

Echinocandins

Echinocandins inhibit the synthesis of glucan (an important constituent of the fungal cell wall) and have a broad antifungal spectrum. Anidulafungin, caspofungin, and micafungin all belong to this group of antifungals. *Candida glabrata* and *Candida krusei*, which are not susceptible to the azole antifungals, are highly susceptible to echinocandins.⁵⁸ Serious side-effects occur rarely and adverse interactions with other drugs are less frequent compared with other types of antifungals.⁵⁹ Since all members of this class of agents are administered parenterally, echinocandins are mainly used for treatment of invasive fungal infections.

Flucytosine (5-fluorocytosine)

Flucytosine is transported into the fungal cell where it is deaminated to 5-fluorouracil (5-FU). 5-FU is further metabolized and then impairs the synthesis of

fungal DNA. 5-FU is also used as an anticancer agent, where it is administered in its active form because mammalian cells do not convert flucytosine to 5-FU. Although the antifungal spectrum of flucytosine is relatively narrow, this drug is active against *Candida* species. Flucytosine is well absorbed from the gastrointestinal tract, and widely distributed in the body.

Since flucytosine has similar properties to the 5-FU agents used in cancer treatment, flucytosine may depress the function of bone marrow and lead to the development of leukopenia and thrombocytopenia. This toxicity is thought to be the result of conversion of flucytosine to 5-FU by the microbial flora in the intestinal tract of the host.²⁶ Other side-effects including skin rash, nausea, vomiting, diarrhea, and severe enterocolitis have been noted. This agent is currently not often used in clinical practice in many countries.

Antiviral agents

Some viruses contain enzymes that initiate viral replication inside a host cell. As viruses have no metabolic machinery of their own, they use the enzymes of their host cell. Antiviral agents often inhibit virus-specific replicative events or preferentially inhibit "virus-directed" rather than "host cell-directed" nucleic acid or protein synthesis. Although various kinds of antiviral agents are currently available, the pharmacology of aciclovir is discussed in this section because the vast majority of viral infections in oral and maxillofacial regions involve members of the herpes virus group and aciclovir is the most commonly used antiviral agent in the treatment of herpes infection.

Aciclovir is an acyclic guanine nucleoside analog drug. Many generic forms of aciclovir are currently available. It is one of the most commonly used antiviral drugs and has been primarily used for the treatment of herpes simplex virus and varicella zoster infections. The mechanism of action of aciclovir is inhibition of viral DNA synthesis.

Aciclovir is generally well tolerated. Oral aciclovir has been associated infrequently with nausea, vomiting, diarrhea, rash, and headache, and very rarely with renal insufficiency or neurotoxicity. No increased frequency of abnormalities has been recognized in infants born to women exposed to aciclovir during pregnancy.²⁶ Intravenous aciclovir may cause renal insufficiency and, on rare occasions, central nervous system side-effects. Valaciclovir is an aciclovir pro-drug. It is converted *in vivo* to aciclovir.

Analgesics/antipyretics

Patients with infections often suffer from pain and fever. Paracetamol and NSAIDs are often used in control of these symptoms.

Non-steroidal anti-inflammatory drugs

NSAIDs are compounds that possess anti-inflammatory, analgesic, and antipyretic effects. Although

members of these classes of drugs are often heterogeneous and chemically unrelated, they nevertheless share certain therapeutic actions and side-effects.

Mechanisms of action

Most currently available NSAIDs inhibit both cyclooxygenase 1 (COX-1) and cyclooxygenase 2 (COX-2) activities. Cyclooxygenase is the enzyme responsible for the biosynthesis of prostaglandins, which are closely associated with the development of inflammation and certain autacoids. Therefore, inhibition of this enzyme is thought to be a major mechanism of NSAIDs' activity. COX-1 is a constitutive isoform found in blood vessels, stomach, and kidney, whilst COX-2 is induced at sites of inflammation by cytokines and inflammatory mediators. The inhibition of COX-2 is thought to mediate, in part, the anti-inflammatory, analgesic, and antipyretic actions of NSAIDs. However, the simultaneous inhibition of COX-1 results in unwanted side-effects, in particular induction of gastric ulcers, which result from decreased prostaglandin and thromboxane formation.

Classification

Table 29.8 provides a classification of NSAIDs and other analgesic and antipyretic agents based on chemical categories. Although individual agents inhibit cyclooxygenase by differing mechanisms, agents that have similar chemical properties generally share certain therapeutic properties and side-effects.

Shared therapeutic activities

All NSAIDs are antipyretic, analgesic and anti-inflammatory, but there is an important difference in their activities. NSAIDs do not change the perception of sensory modalities other than pain. Postoperative

Table 29.8 Classification of non-steroidal analgesics.

Group	Analgesics
Salicylates	Aspirin
Para-aminophenol derivative	Paracetamol
Acetic acid derivatives	Indomethacin
	Sulindac
	Etodolac
	Femansates
	Tolmetin
	Ketorolac
	Diclofenac
Propionic acid derivatives	Meclofenamate
	Ibuprofen
	Naproxen
	Fenoprofen
	Ketoprofen
	Flurbiprofen
	Oxaprozin
Enolic acid derivatives	Piroxicam
	Meloxicam
	Nabumetone
COX-2 selective inhibitors	Celecoxib
	Valdecoxib

pain or pain arising from inflammation can be controlled by NSAIDs. NSAIDs reduce the body temperature in febrile state but do not affect normal body temperature in the absence of fever.

Side-effects

The clinically important side-effects that are shared by NSAIDs are generally dose-dependent.

- *Gastrointestinal tract.* Irritation of the gastrointestinal tract is the most common side-effect. Complications include epigastric distress, stomach pain, nausea, vomiting, dyspepsia, gastric ulceration, and gastrointestinal bleeding. Gastric ulceration is sometimes severe and can be accompanied by anemia from the resultant blood loss. It should be remembered that gastrointestinal side-effects are sometimes painless, although they can result in admission to hospital, surgery, and possibly death. The risk factors include advanced age (especially >65 years old), previous peptic ulcers or ulcer complications, concomitant use of glucocorticoids or anticoagulants, and high-dose or prolonged NSAID administration.^{60,61}
- *Blood.* Ingestion of NSAIDs prolongs the duration of bleeding due to a blockade of platelet aggregation. Through exploitation of this effect, aspirin can be a useful agent in the management of arterial thrombosis and prevention of adverse cardiovascular events.
- *Kidney.* NSAIDs may change renal blood flow and ultimately may cause renal impairment, which leads to retention of salt and fluid and resulting hypertension. The risk of renal impairment may increase in instances where NSAIDs are used in combination with other nephrotoxic agents or under some medical conditions such as chronic renal diseases and congestive heart failure.
- *Influence on pregnant women.* Similar to other drugs, there is a concern over possible teratogenic effects of NSAIDs. Moreover, NSAIDs may cause premature closure of the fetal ductus arteriosus and renal impairments in the fetus.⁶² Consequently, use of NSAIDs is not recommended during pregnancy, particularly during the first and third trimesters.
- *Liver.* Epidemiological evidence indicates that the use of salicylates and some members of NSAIDs are associated with the occurrence of severe hepatic injury and encephalopathy observed in Reye's syndrome.^{63,64} This syndrome is rare, but is often fatal, especially when associated with infection with varicella zoster virus and various other viruses, e.g. influenza viruses. The use of these agents in children or adolescents with chickenpox or influenza is contraindicated, but paracetamol may be a useful alternative.
- *NSAID intolerance.* The prevalence of intolerance for aspirin and most NSAIDs is 5–6%.⁶⁵ NSAID intolerance manifests as asthmatic attacks or urticaria/angioedema. Asthmatic attacks are often

severe and life-threatening. Although rare in children, up to 20% of the asthmatic population is sensitive to aspirin and other NSAIDs.⁶⁵ Patients who have, or have ever had asthma, frequent incidences of “stuffed” or “runny” noses, or nasal polyps, have a higher risk of hypersensitive attack following use of NSAIDs.

Paracetamol (acetaminophen)

Paracetamol has similar analgesic and antipyretic effects to NSAIDs. However, its anti-inflammatory effects are very weak. The mechanism by which paracetamol reduces fever and pain remains unclear. Since its mechanism of action differs from those of other NSAIDs, paracetamol lacks certain side-effects exhibited by other NSAIDs, such as gastrointestinal tract damage and blockade of platelet aggregation. Although paracetamol is regarded as safe and well tolerated during pregnancy, it should be noted that acute overdosage can cause fatal hepatic damage.

Principles of diagnosis of oral and maxillofacial infections

All patients are evaluated based on careful collection of history, physical examination, results of laboratory tests, and imaging studies. Infections, especially bacterial infections, can cause life-threatening complications. Therefore the emergency level must be evaluated initially.

Assessment of emergency level

The consciousness level and airway status of the patient are first determined. If any indication of significant complication or suspected serious condition is evident, the patient must be referred to an emergency clinic or specialist immediately following checking the vital signs.

Recording of medical history

Once the patient has been confirmed as not requiring critical care, an infection history is obtained by interview or self-administrated questionnaire. The patient's primary complaint and history of the present infection are carefully obtained. Necessary information includes the onset, nature, location, and duration of each symptom, history of recurrence, including its frequency, and responses to previous treatment.

A medical history of the patient between birth and the time of assessment is necessary for adequate diagnosis and management. Information on any medication previously prescribed must be fully obtained. The patient's defense level must be assessed together with the medical information. Common immunocompromising factors are listed in Table 29.9. Although any component of the host defense system can be suppressed, the majority of listed factors com-

Table 29.9 Immunocompromising factors associated with increased occurrence and poor prognosis of oral and maxillofacial infections.

Diabetes mellitus
Systemic use of corticosteroids
Systemic use of immunosuppressants
Malignant tumors
Leukemia, lymphoma, myeloma
Antineoplastic chemotherapy
Radiation therapy
Alcoholism
Cirrhosis
Chronic renal failure
Splenectomy
Organ transplantation
Collagen vascular disease
HIV/AIDS
Malnutrition

promise the functions and production of phagocytes and cytokines. Assessment of the hydration and nutritional status of the patient is also important. Patients may not take in sufficient water and food because of pain, poor appetite, trismus, and difficulty with chewing and swallowing. Furthermore, fever also increases fluid loss and calorific needs. As a consequence, a degree of dehydration and malnutrition may occur. The patient's fluid balance and nutrition level can often be anticipated through interview, although actual status should be evaluated based on blood and urine tests.

The experience and details of previous hypersensitive reactions and suspected side-effects of medications must be recorded.

It should be noted that patients often have undiagnosed diseases (especially diabetes, chronic hepatitis, infection with a hepatitis virus, HIV, and tubercle bacillus carriage), or in the case of some women, be unaware of a pregnancy. As a result, information obtained from interview may not be completely reliable.

Assessment of the patient's present status

Recording of symptoms

All symptoms of the patient must be determined. The nature, location, and duration of pain have to be recorded. Likely symptoms may include headache, chills, sweating, loss of appetite, and insomnia. Any difficulty in swallowing, breathing, eating, chewing, and opening of the mouth must be recorded to evaluate oral dysfunction.

Infections in the maxilla might involve the maxillary sinus. Rhinorrhea, epistaxis, problems breathing through the nose, nasal congestion, and changes in

sense of smell indicate possible sinusitis. In the case of the mandible, an abnormality of sensation in the area innervated by the mandibular nerve would suggest a significant inflammation of the bone marrow.

Physical examination

Physical examination includes assessment of the patient's general condition and also incorporates a comprehensive regional examination.

The general appearance of the patient, especially whether or not they have an appearance of fatigue, feverishness or malaise (so-called '*toxic appearance*') and the skin colour of the face needs to be inspected.

Vital signs must also be checked. These include the respiratory rate, pulse rate, blood pressure, and body temperature. Normally, the respiratory rate of healthy individuals is 14–16 breaths per minute, but it may increase as infection progresses. A decrease in blood oxygen level detected by pulse oximetry might indicate a possible airway obstruction. Pulse rate may also increase as the body temperature rises. In severe infection, the pulse rate often exceeds 100 beats per minute. Although infection itself has little effect on blood pressure, significant pain and anxiety might indirectly elevate blood pressure, whilst dehydration and shock may cause a reduction in blood pressure. Fever is often evident even in mild infection. It should be noted that various non-pathogenic factors affect body temperature. Fevers in children tend to be exaggerated, whereas elderly patients may experience little temperature change, even in severe infection. It is important to appreciate that non-infective diseases (e.g. collagen disease, cancer) can also induce fever, so it is essential to determine initially whether a fever actually involves an oral infection.

Regional examination starts with checking the symmetry of face. The area and nature of swellings, erythema, warmth, and tenderness are examined by careful observation and palpation. If the swelling is subtle, the clinician should ask the patient to describe any areas of swelling that they are aware of. The nature of the swelling varies from feeling very soft and almost normal through to a firmer or hard swelling, not dissimilar to a tightened muscle. Swelling due to the accumulation of pus has a different feel to it from that due to edema, and the former is described as fluctuant. Fluctuance has the feeling of a fluid-filled balloon, and can be determined by placing the fingers at the sides of the swelling and detecting fluid movement (Fig. 29.8). In the case of a deep-seated abscess, however, detection of fluctuance may be difficult by palpation.

Regional lymph nodes, and especially the submandibular and submental lymph nodes, should be inspected for enlargement, tenderness, redness, and warmth of overlying skin.

Dental and periodontal status is recorded together with any local inflammatory signs. If infection is suspected as being of dental origin, the probable source

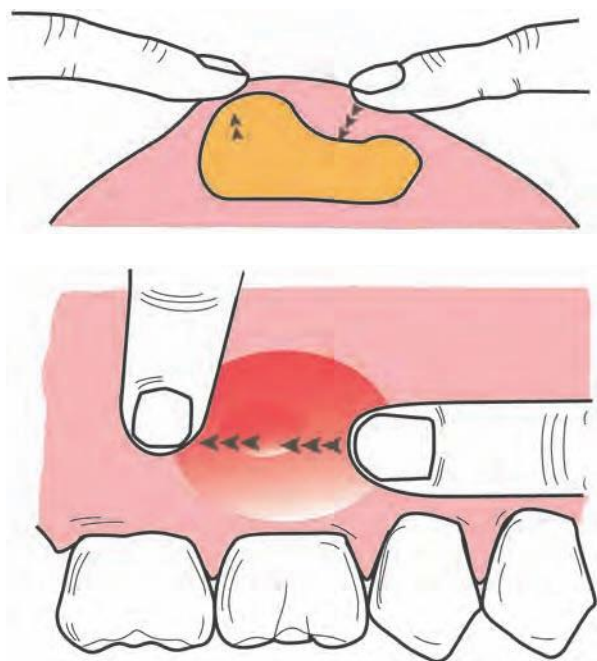


Fig. 29.8 Fluctuance. As one finger applies pressure, another detects the fluid movement.

of infection should be determined in the oral examination (e.g. inspection, palpation, and percussion). The maximum vertical range of jaw motion is recorded for determining the degree of trismus.

Imaging studies

Radiographic examinations are important in determining the possible cause of infection and the extent and status of the lesion. The clinician must be aware of the normal radiographic anatomic structures. An overview of infection-related radiographic methods is presented here. For a more detailed overview see Chapter 2.

Plain films

Radiographic examinations often start with a plain film study. Standard dental (intraoral) X-ray film, especially for a periapical radiograph, is suitable for examining localized dental and alveolar diseases because of its fine and clear image quality, cost-effectiveness, and low radiation exposure. As a panoramic radiograph (pantomogram) takes a much wider area of image than intraoral films, it is particularly useful for making a precise localization of infection and to demonstrate the extent of infection and any anatomic relationship between the lesion and surrounding structures. This approach is also available for the patients with trismus or other extenuating conditions, such as severe gag reflex caused by placement of film inside the mouth. The individual structures are somewhat unclear and fine detail cannot be seen, since the panoramic film provides a lower resolution picture than that obtained with the intraoral films.

A Waters' projection and posterior-anterior view may demonstrate inflammation within paranasal sinuses.

Computed tomography and magnetic resonance imaging

Computed tomography (CT) is available in medical imaging to supplement radiographs and medical ultrasonography. CT is particularly useful in examining fascial space infections, osteomyelitis of the jaw, and paranasal sinusitis. The high cost, possible side-effects of iodinated contrast agents (allergic reaction and kidney damage), and the relatively high radiation exposure are limitations of CT. Imaging artefacts, especially those caused by metallic materials (e.g. crown and reconstruction plate) may also reduce the effectiveness of CT.

Magnetic resonance imaging (MRI) is available for examining a variety of infections. An MRI study is more sensitive for the diagnosis of some types of infections, such as osteomyelitis of the jaw and suppurative arthritis of the temporomandibular joint, compared with CT scans. However, the cost of MRI examinations is generally higher than that of CT. Artefacts related to the use of metallic instructions may also disturb the MRI study. Use of MRI should be avoided in patients who have implanted medical devices that are strongly affected by magnetism, as evident with artificial heart pacemakers.

Radionuclide scans

A radionuclide scan is a way of imaging parts of the body by using a small dose of a radioactive chemical. A tiny amount of radionuclide, which is a chemical that emits gamma rays, is introduced into the body, usually by an injection into a vein. The radionuclide tends to collect or concentrate in specific organs and tissues with certain pathogenic conditions, including inflammation. The gamma rays that are emitted from inside the body are detected by a gamma camera and are converted into an electrical signal and subsequently the computer builds a picture. There are different types of radionuclides, and the radionuclide employed depends on which part of the body is to be scanned. Although radionuclide scanning may be unnecessary in making diagnosis of most forms of infection in the oral and maxillofacial region, it is a particularly useful imaging technique for the diagnosis of acute osteomyelitis (see the later section, Osteomyelitis of the jaw).

Blood and urine tests

Results of blood and urine tests can provide significant information relating to infection. The data are available not only for diagnostic purposes but also in the assessment of the patient's general condition and evaluation of the response of treatment through data monitoring.

Systemic effects of infection may result in the abnormal fluctuation of various factors measured by a blood test. Certain data are closely associated with progression of infection. These include leukocyte count and differential C-reactive protein (CRP) level, and erythrocyte sedimentation rate (ESR).

Leukocyte count and the leukocyte differential

Although the normal leukocyte count in peripheral blood ranges between 4000 and 11 000 cells/ml,³ it generally increases during the active phase of microbial infections (leukocytosis). In addition, the percentage of neutrophils in the blood may also be elevated. In the case of severe infection, immature neutrophils are often evident (this is referred to as a left shift). After the infection has reached its peak, the leukocyte count and the percentage of neutrophils will gradually decline to their normal range. Therefore the stage of infection can be determined by monitoring the fluctuation of these factors.

C-reactive protein

The CRP level in peripheral blood is frequently measured in the assessment of the activity of infection. The CRP value can reflect the intensity of inflammation with greater sensitivity than other blood factors including leukocyte count. However, the range of CRP fluctuation varies between patients. The monitoring of CRP level in the same patient is extremely useful in evaluating the outcome of therapy.

Erythrocyte sedimentation rate

An elevated ESR indicates the presence and intensity of infection. However, the normal ESR range can vary with age, ethnicity, gender, and with certain underlying diseases.²¹ Moreover, it reacts slowly to changes of inflammatory activity. The sensitivity and specificity of ESR on diagnostic usage for infection is not high.

Other tests

Blood and urine tests may also demonstrate anemia and abnormalities of hepatic and renal function, fluid balance, and nutrition level. Moreover, urinalysis and/or a blood sugar test may detect previously undiagnosed diabetes. It should be noted that severe infection itself tends to raise the blood sugar level, and any recent use of corticosteroids, alcohol or drug abuse or consumption of food and drink containing glucose may affect the data.

Microbiologic examination

The purpose of microbiologic examination is to identify any causative microorganisms and to determine

their susceptibility to antibiotics or antifungals. Microbiological examination is of great value for clinical practice because case-specific antimicrobial regimens can be made based on the results. However, data obtained from improper sample collection, poor technique, and inappropriate transport and management of specimen may prove misleading (Table 29.10).

Microbial examination generally comprises a microscopic analysis of the specimen and its subsequent culture. Culture is necessary for identification of organisms and any subsequent susceptibility testing (Fig. 29.9). Aerobic and facultative bacteria and fungi may grow relatively rapidly on agar media, whilst sufficient growth of strictly anaerobic organisms requires at least a few days. Therefore microbiologic examination, particularly involving the culture of anaerobic organisms, usually takes several days.

When collecting specimens, the clinician should record the site of sampling, visual features (for example, the presence of any yellow thick, blood-like or thin liquids), the odor of the specimen, and status of the abscess (e.g. whether it has already drained). A rotten or gangrene-like odor of the specimen indicates an involvement of anaerobes.

Large numbers of microorganisms reside in the human body, especially within the mouth. Therefore specimens may often be contaminated with indigenous microorganisms that are not involved in the infection. To minimize this risk, the material should be taken from sites that are representative of the infection, and any contact with surrounding tissues

Table 29.10 Summary of specimen collection guidelines for microbiologic examination.

Avoid contamination from indigenous flora
Aspiration is preferred to swab for sample collection
Collect a sample from correct site by proper technique
Collect adequate volumes
Place the specimen in adequate container or transport medium
Send the specimen to laboratory as soon as possible
Do not hesitate to re-examine if unreasonable results are obtained

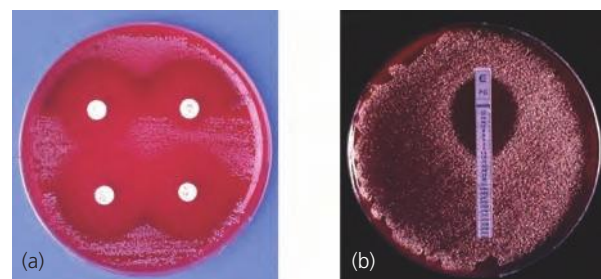


Fig. 29.9 (a) Picture of an agar plate for antimicrobial susceptibility. A wide area of no bacterial growth around antibiotic disks is evident, suggesting that the strain is susceptible to the antibiotic. (b) E-test provides the minimal inhibitory concentration of an antibiotic.

and fluids during collection must be avoided. Pus obtained by aspiration is less likely to be contaminated, whilst sampling with swabs tends to result in a high likelihood of contamination. However, pus specimens from abscesses that have already been opened are likely to be contaminated even when sampled by aspiration.

Some microorganisms are sensitive to *in vitro* environments. For example, oxygen kills strictly anaerobic microorganisms and drying kills most bacteria when left exposed to the air. Conversely, some types of aerobic organisms can multiply when stored for several hours at room temperature. Therefore, inadequate management of specimens after collection and delayed transport to the laboratory may result in deterioration and overgrowth of microorganisms within specimens and may produce incorrect results. An insufficient sample volume may also provide inaccurate results.

Purulent infection of dental origin usually involves strictly anaerobic bacteria. Aspiration is recommended for sampling because of the likely collection of a sufficient amount of material and the reduced risk of exposure to oxygen. The optimal time for specimen transport to the laboratory is 2 hours or less if an adequate volume of material is obtained; however if only small volumes of material are sampled, then the specimen should be processed within 15–30 minutes of collection.⁹ These processing times may be achievable when the diagnostic microbiology laboratory is located in close proximity to the clinic. However, if laboratory facilities are not locally available, specimens need to be stored under adequate transport conditions suitable for the suspected microorganisms (Fig. 29.10), and sent to an appropriate laboratory within 24 hours. In the case of swabs, the specimen must be kept in an appropriate transport medium and immediately sent to the microbiology laboratory.

Pieces of infected soft tissue or bone obtained surgically can also provide suitable specimens for microbiologic analysis. Such material should be placed in sterile Petri dishes or screw-top bottles with a small

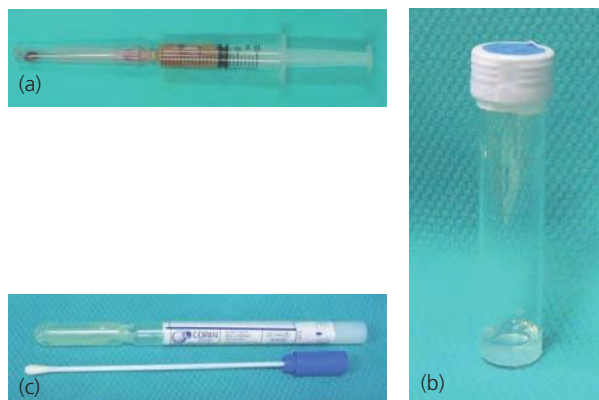


Fig. 29.10 Pus specimen obtained by aspiration (a) should be held in an anaerobic transporter (b), which contains a substance that eliminates oxygen. Swab sample (c) must be handled to avoid drying and sent to the laboratory immediately.

volume of saline solution to prevent desiccation and again must be transported to the laboratory as soon as possible.

A positive blood culture demonstrates that an infection is spreading through the bloodstream. Blood culture is important to determine if sepsis is occurring in the patient. It is also an option for assessing microbial flora involved in orofacial infection if an adequate pus specimen is not available. A minimum of 10 ml of blood is collected through venipuncture and inoculated into blood bottles (Fig. 29.11), which contain specific culture media in accordance with the suspected microorganisms present.

It is sometimes difficult to interpret microbiologic results. Blood-like or thin pus often yields extremely low bacterial counts compared with thick, yellow pus, and the former is therefore likely to produce less accurate results. Sampling pus in the central part of the abscess, where bacteria might have lost their viability, may also be a cause of false-negative culture. In patients receiving systemic antibiotics, the pus or blood (in the case of blood culture) may also contain traces of antibiotic. As a consequence, culture results may often be incorrect due to the selection of certain organisms by the antibiotic. In such circumstances the clinician should not hesitate in retesting the sample site.

Principles of management of bacterial infections

This section specifically focuses on the principles of management of bacterial infection, with fungal and viral infections reviewed independently (see later sections, Oral fungal infections and Oral and perioral viral infections). The management of bacterial infection consists primarily of surgery, antimicrobial therapy, and medical supportive care, with the exception of emergency treatment.



Fig. 29.11 Blood culture bottles. Left bottle is for aerobic culture and right one contains culture medium for strict anaerobes.

Surgical treatment

Surgery is an essential part of the treatment of suppurative infections. It ranges from simple and relatively non-invasive procedures, such as pulpectomy and tooth extraction, to wide incision of abscesses in the head and neck. Surgery may facilitate the removal of the cause of infection and promote an improvement of the host defense through elimination of accumulated pus, debris, and necrotic tissues. Surgical drainage may obviate the use of antibiotics or may enhance the effectiveness of antibiotics through restoring vascular flow at the infection site.

Drainage by incision

In the case of pus-producing infections, incision of the abscess cavity and subsequent drainage of pus are critical to the resolution of infection. Pus contains dead leukocytes (neutrophils) together with the causative bacteria. An adequate drainage of pus also provides a reduction in tissue tension, improvement of the local blood supply, and change in the local environment (increased oxygenation of the infected area). Even if antibiotics are administered, failure of drainage may result in further progression of infec-

tion and may delay resolution. Clinicians should carefully palpate swollen areas to detect fluctuance, which indicates abscess formation. Any abscess should be incised, even if other surgical procedures including tooth extraction and endodontic therapy are undertaken.

The standard process of incision and drainage is presented in Fig. 29.12. The notable principles are as follows:

- *Optimal timing.* The optimal timing of incision varies. In general, abscesses should be incised when pus has accumulated within the cavity. Most infections start as a phase of cellulitis that may occur as a soft, doughy, and diffuse swelling. In this stage, limited amounts of pus are available, and infection may not respond to surgery. However, in cases of cellulitis that spread widely, especially fascial space infections, an early incision may be recommended because undetected abscesses may appear through incision and subsequent exploration, and the surgical procedure may provide a reduction of pressure in the area of cellulitis.
- *Anesthesia.* Conventional local infiltration anesthesia may be satisfactory for incision of superficially placed and well localized abscesses. The anesthetic

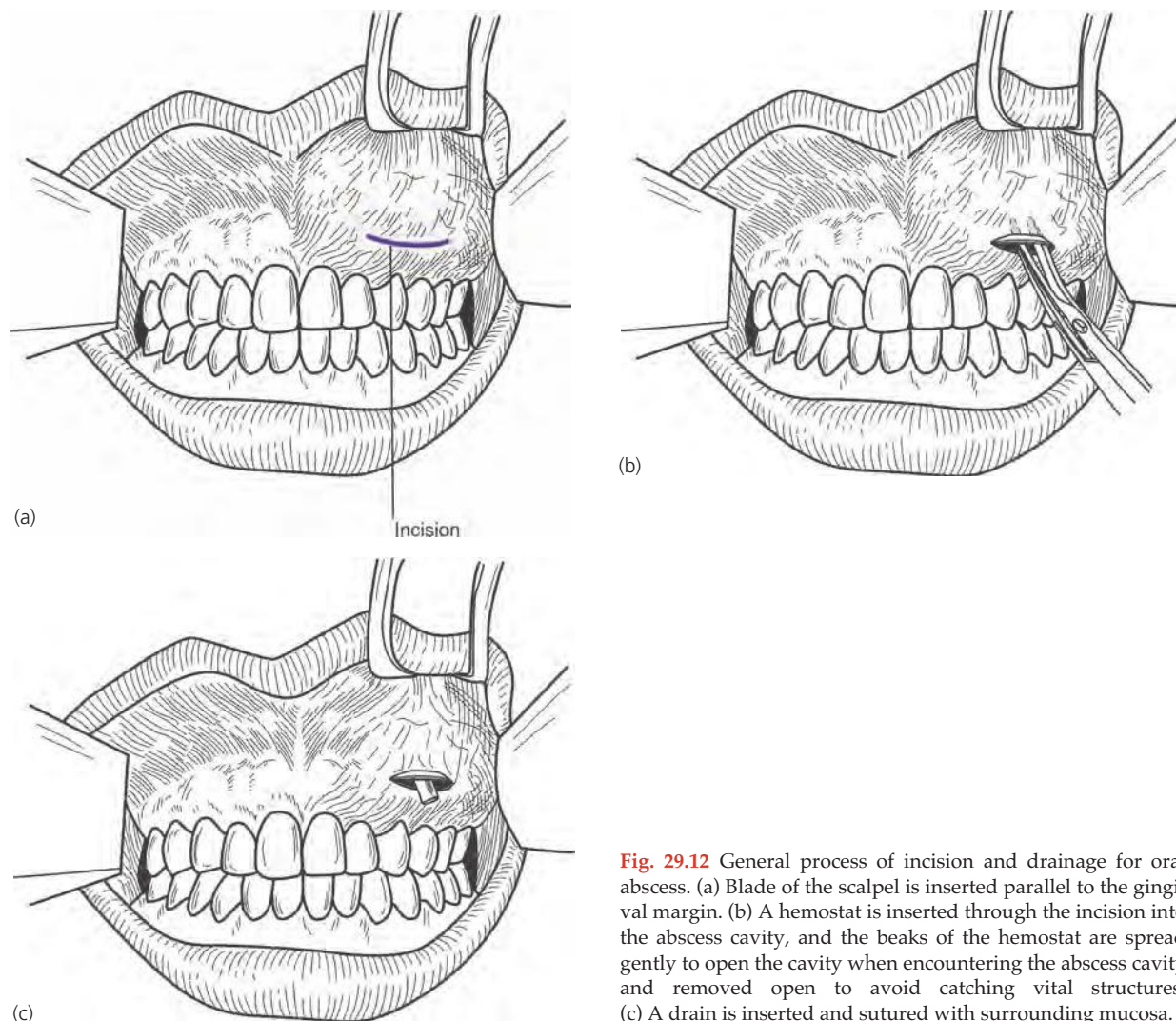


Fig. 29.12 General process of incision and drainage for oral abscess. (a) Blade of the scalpel is inserted parallel to the gingival margin. (b) A hemostat is inserted through the incision into the abscess cavity, and the beaks of the hemostat are spread gently to open the cavity when encountering the abscess cavity and removed open to avoid catching vital structures. (c) A drain is inserted and sutured with surrounding mucosa.

is injected into the gingiva, mucosa or skin surrounding the abscess, but should not be injected directly into the abscess cavity. Since inflammation creates an acidic environment, local anesthetics may be slower than normal in their onset of action and less effective in inflamed tissues.⁶⁶ Therefore, a greater volume of anesthetic is often required to be administered to achieve adequate anesthesia compared with that used in routine dental treatment. Infiltration anesthesia may be inadequate in cases of extensively spreading or intensive infection. Use of inferior alveolar nerve block, sedation or general anesthesia should be considered.

- *Aspiration of pus prior to incision.* Aspiration of pus from the abscess cavity using a disposable sterile syringe with a large-gauge needle (usually 18 gauge) prior to actual incision of the abscess is recommended for the following reasons: (1) actual pus accumulation can be confirmed; (2) the procedure may provide clues as to the precise localization of the abscess; and (3) pus obtained by aspiration is more suitable for bacteriologic examination. The authors recommend the use of a small-volume syringe (2 ml), because aspiration using a large syringe often causes pain through the strong suction pressure. The surface of the mucosa or skin must be disinfected with an appropriate antiseptic (for example, povidone-iodine and ethanol) before aspiration to avoid contamination of the specimen.
- *Avoid damage to important structures.* An incision must be carefully placed to avoid any damage to important structures, such as salivary gland ducts and nerves (especially the facial nerve branches).

The incision should be made in a direction parallel to the branch of facial nerves near the infection site. As the marginal mandibular branch of the facial nerve dips below the lower border of the mandible, an incision line is recommended to be at least two fingers' width below the lower border (Fig. 29.13). Attention is also required to avoid injury of the mental nerve by incision of the abscess in the region of the mandibular premolars. As damage to the greater palatine artery and lingual artery may result in significant hemorrhaging, incision of palatal and pharyngeal abscesses and surgical drainage of sublingual spaces need to be performed carefully.

- *Incision in a cosmetically or functionally acceptable place.* Although an intraoral incision generally does not cause serious cosmetic problems, the incision should not be longer than necessary. Careless incision might result in a later functional problem. Incision at sites related to oral function, such as the frenums should be avoided. In the case of extraoral incision, the incision should be placed in an esthetically acceptable area, for example, in the shadow of the jaw, beyond the hairline, and parallel to the lines of skin tension.
- *Incision with deliberation to maximize efficient drainage.* An incision at a position that encourages drainage by gravity will obviously be desirable.
- *Use of blunt dissection.* A superficially placed abscess cavity may be opened easily by incision. Vestibular abscesses usually form between the bone surface and the periosteum. In these cases, the scalpel blade is inserted to the surface of the cortical plate in order to open the abscess beneath the periosteum. If pus does not discharge, the inci-

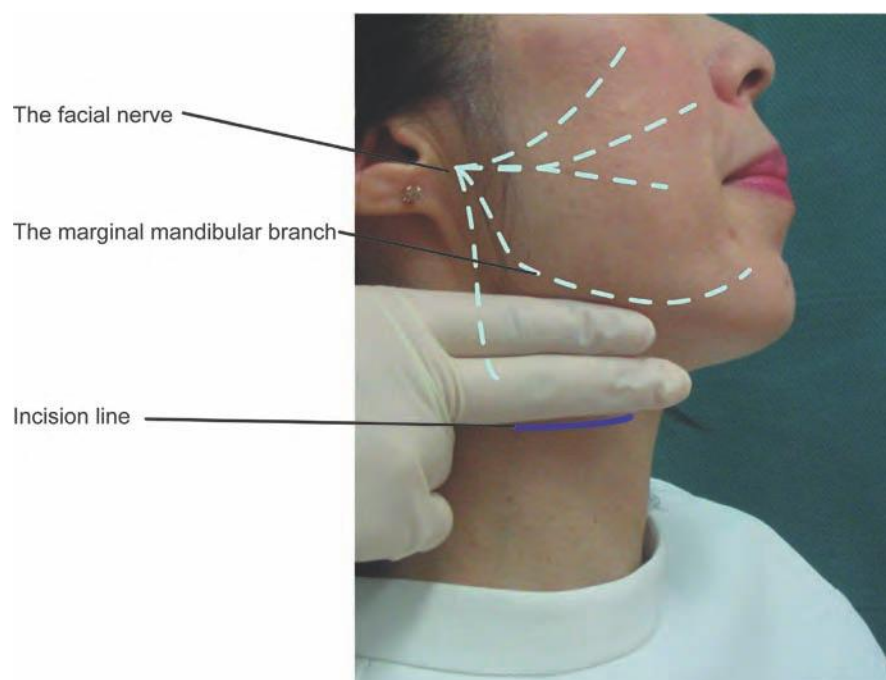


Fig. 29.13 An incision at submandibular region or mandibular angle should be placed two fingers' breadth below the lower border to avoid injury of the marginal mandibular branch of the facial nerve.

sion may not be deep enough. In the case of deeply placed abscesses, careless incision might cause serious damage and injury to vital structures, especially veins and nerves. A scalpel must not be inserted into deep tissues. Once the skin or mucous membrane is incised, the abscess must be approached by blunt dissection using a hemostat, as blunt dissection should minimize the risk of significant injury within tissues. A closed hemostat is inserted through the incision into the abscess cavity. Once the abscess cavity is encountered the beaks of the hemostat are spread to break up any small loculations or cavities of pus that have not been opened by the initial incision; this is done in a direction parallel to the vital structures in the region. The beaks of the hemostat should not be closed within the tissues, in case a vital structure is damaged.

- *Irrigation of the abscess cavity.* In the case of large abscesses, irrigation with saline may be effective in reducing residual contamination.
- *Placement of drain.* Abscesses tend to seal off shortly after incision. Therefore, a drain may be inserted to maintain the opening of the abscess cavity to encourage drainage of pus that is residual or forming. In the case of small abscesses the placement of

a drain may not always be required. A sterile Penrose drain (Fig. 29.14) is commonly used for intraoral abscess drainage. A drain of adequate length to allow drainage from the depth of abscess cavity is inserted using a hemostat, and it is then fixed by a suture to surrounding tissues that are not necrotic, to prevent unwanted movement and loss of the drain. The wound must not be closed.

- *Removal of the drain.* Drains should be removed when the drainage ceases or becomes minimal. Prolonged placement of drains might delay the normal wound healing process. Drains may be shortened over the next few days to ensure that drainage is completed.

Endodontic therapy (root canal opening)

The majority of orofacial bacterial infections are endodontic in origin. Drainage through the root canal is effective (Fig. 29.15), although infections of non-periapical origin will not respond to this procedure. Despite the fact that the extent of this drainage is usually smaller than that of an incision, it does contribute to prevention of further progression and may allow resolution of infection. Pus commonly accumulates in abscess cavities at the tip of the causative root apex, on the inside of the alveolar bone. Therefore, surgical opening of the root canal may be considered even in instances of cellulitis, in which a palpable abscess has yet to form. However, if the tooth has been restored



Fig. 29.14 A Penrose drain.

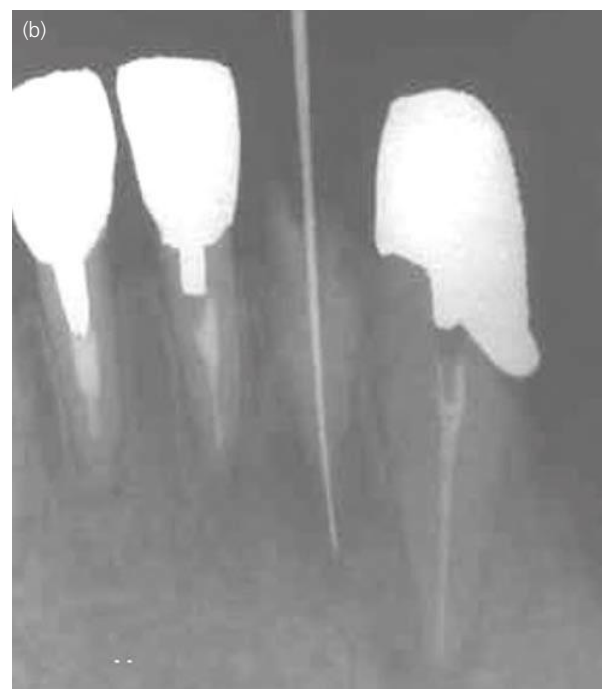


Fig. 29.15 The procedure of endodontic drainage. (a, b) An endodontic reamer is inserted as far as the abscess cavity at the tip of root apex. (c) Pus may be drained through the opened root canal.

with a post crown or if a root canal has been closed or constricted due to calcification or previous endodontic treatment, endodontic drainage may be very difficult. Severe root canal curvatures may compromise root canal treatment. Severe trismus also limits the application of endodontic drainage.

Although the actual effect is controversial, trephination techniques can be employed in cases of periapical abscess where there is extensive pain, when there is no swelling, and/or drainage through conventional endodontic therapy is not feasible (Fig. 29.16).

Tooth extraction

Extraction of teeth involved in infection results not only in the removal of the cause but also drainage of accumulated pus through the socket. Early tooth extraction is generally recommended, although it might cause additional swelling. A loose tooth often causes pain caused by contact with opposite teeth, food, tongue, and lip during chewing and swallowing, and therefore should be removed as soon as possible. It should be noted that the offending tooth is sometimes able to be salvaged by adequate dental treatment after resolution of the acute infection. Mild infections therefore do not always require tooth removal.

Surgical removal of foreign bodies involved in infection

As with tooth extraction, surgical removal of any infected foreign bodies aims to remove the cause of infection, eliminate harmful stimuli, and encourage drainage. Loose implants, screws, plates, and other materials should be removed as soon as possible.

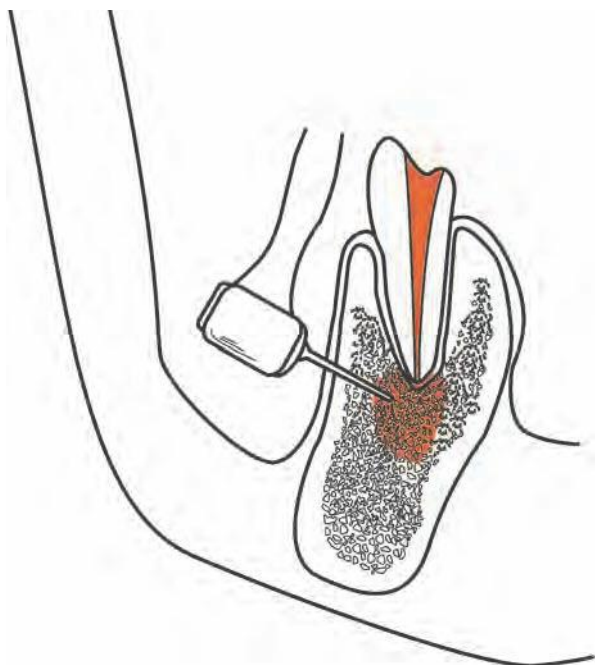


Fig. 29.16 Trephination involves artificial penetration into the abscess cavity, which is made through the mucosa and cortical plate to make a pathway for drainage.

Inappropriate surgical procedures

In some Middle Eastern countries, application of hot metal instruments (Wassam) by non-medical “doctors” has been used in an attempt to relieve the pain of dental infections. This totally inappropriate approach results in acute superficial skin infection and extensive ulceration that heals with scarring (Fig. 29.17).

Antimicrobial therapy

Prevalence of antibiotic-resistant organisms

Systemic antibiotic therapy is undoubtedly a significant part of the management of bacterial infections. However, recent years have seen a dramatic increase in the prevalence of antibiotic-resistant organisms and emergence of multidrug-resistant bacteria. Bacteria may become resistant to antibiotics through continued exposure to an antibiotic, and in the case of systemic antibiotic therapy, resistant bacteria at other body sites might develop. It is of great concern that many clinicians still use antibiotics inadequately, with poor understanding of the consequences.^{67–69} Antibiotics are an essential weapon in the combat of bacterial infections and are the result of a great deal of research and development over many years at a significant financial cost. Proper use of antibiotics is therefore an ethical duty for all clinicians.

The role of antibiotic therapy

Undoubtedly, antibiotics are overprescribed in clinical practices of dentists and oral surgeons. It is critical to keep in mind that the priority in the management of oral and maxillofacial infections is to improve host defenses through surgical procedures. Even if more enhanced and improved antibiotics are developed, this principle will not change. In circumstances where drainage is unavailable (for example in the case of



Fig. 29.17 A patient with a superficial skin infection of the face caused through intentional burning with application of a hot iron to an area of the face that was swollen due to an odontogenic infection (this “procedure” is referred to as Wassam).

cellulitis), the role of antibiotics is important. However, the role of systemic antibiotic therapy is as an adjunct to surgery, and the prime purpose of such therapy is to prevent spread of infection and onset of serious complications.

Clinicians must recognize the potential negative effects of any antibiotic therapy. All antibiotics may induce a variety of side-effects or interact with other systemically administered medicines, and some of these effects are potentially serious and fatal. Antibiotics distribute throughout the body, and may disrupt microbial homeostasis at a variety of body sites, with resulting clinical problems including diarrhea and mucosal candidosis.⁷⁰ Obviously, broad-spectrum antibiotics are more likely to disrupt such microbial homeostasis. As described earlier, antibiotic therapy increases the chances of the occurrence of bacterial resistance. The cost of the antibiotic might also be an important matter for consideration.

Indications for antibiotic use

Indications for antibiotic therapy are presented in Table 29.11. Systemic antibiotics may be unnecessary to treat a minor, chronic or well localized abscess. Management of dry socket, mild acute infection, and chronic infection do not require antibiotic treatment. However, in immunocompromised patients, use of systemic antibiotics may be advocated even if the infection is not severe.

Table 29.11 Indications and non-indications for systemic antibiotic therapy.

Situation in which antibiotic therapy is necessary or recommended

Acute orofacial suppurative infections
Osteomyelitis
Acute maxillary sinusitis
Suspected actinomycosis
Acute necrotizing ulcerative gingivitis
Acute bacterial infection in an immunocompromised patient
Fascial space infections
Acute peritonsillar abscess
Acute traumatic and surgical wound infections

Situation in which antibiotic therapy may be unnecessary

Pulpitis
Chronic periapical abscess or disease
Gingivitis and periodontitis
Minor vestibular abscess
Chronic or mild acute pericoronitis
Peri-implantitis
Postoperative swelling and pain
Alveolar osteitis (dry socket)
Chronic wound infection
Fungal or viral infection

Table 29.12 Conditions where microbiologic examination is recommended. Microbiologic examination includes identification of microbes by culture and antibiotic susceptibility testing.

Severe infections including fascial space infections
Rapidly spreading infection
Infection that exhibits little or no response to the present therapy
Recurrent infection
Possible infection of non-oral origin
Osteomyelitis
Maxillary sinusitis
Suspected actinomycosis
Surgical wound or trauma infection
Infection in immunocompromised patients

Empiric therapy and the necessity of microbiologic examination

The choice of antibiotic therapy should ideally be based on laboratory data. However, such results are often not available for several days. The cost of examination also limits such an approach in routine practice.

Orofacial bacterial infections, especially odontogenic infections, are most often caused by predictable organisms. As a consequence, routine microbiologic tests are not necessary and the initial choice of antibiotic is largely done on an empiric basis. However, there are circumstances where laboratory tests are strongly recommended. These include severe cases, rapidly spreading infection, and infection in immunocompromised patients (Table 29.12). Microbiologic examination is of great value when treating osteomyelitis, sinusitis, suspected actinomycosis, surgical site infections (surgical wound infections), and traumatic wound infections because of the uncommon organisms that might be involved.

Principles of antibiotic choice

Empiric choice of an antibiotic is based on the following principles (Table 29.13):

- *Predicted microbial flora involved in the infection.* Most orofacial infections involve predictable organisms. Clinicians should therefore have contemporary knowledge of the microbiology of orofacial infections.
- *Choice of the specificity and spectrum of the antibiotic.* Ideally, antibiotics will target only the causative organisms without an effect on the other commensal microbes. Often the most suitable candidate antibiotics are those of the penicillin class. Antibiotics that exhibit a broader antimicrobial spectrum should be reserved for situations where narrower-spectrum alternative antibiotics are unsuitable.
- *Use of a regimen with a single antibiotic.* Use of two or more antibiotics in combination may be

Table 29.13 The principles of empiric antibiotic therapy.

Understand the role of antibiotic therapy and indication of antibiotic use
Predict microbial flora involved in infection
Choose antibiotic with adequate spectra for predicted microbes
Generally single antibiotic use
Use an antibiotic with low toxicity and a reduced likelihood of side-effects
Choose antibiotic in accordance with patient's physical condition
Bactericidal antibiotic is recommended if the patient's immunity is impaired
Consider cost of antibiotic therapy

advantageous in certain clinical situations, providing a synergistic activity against specific organisms or enhancement of antibacterial spectrum. Therefore, a multidrug regimen of antibiotics is advocated for management of patients with severe infection or a high likelihood of a serious complication. However, this kind of regimen may increase the possibility of disrupted microbial homeostasis. Moreover, antagonistic antimicrobial effects can occur depending on the combination and the bacteria. A multidrug regimen of antibiotics is generally more expensive compared with that of a single antibiotic. Most infections can be treated with a single antibiotic, thereby reserving the multidrug regimen as a later option if required.

- *Use of safe antibiotics.* Although all antibiotics have various degrees of toxicity and a range of side-effects, β -lactam antibiotics, especially penicillins, are generally considered safe. Macrolides have a low incidence of side-effects, and quinolones (fluoroquinolone) and metronidazole have minor toxicity. In contrast, aminoglycosides and vancomycin exhibit high renal toxicity. Antibiotics may provide an undesirable enhancement or reduction of the effect of medicines that are administered systemically, such as anticoagulants, theophylline (drug for respiratory diseases), and the birth control pill. In particular, macrolides are more likely to cause such interactions.
- *Consideration of the patient's condition.* The systemic use of antibiotics in pregnant women involves an evaluation of risk versus benefit. Penicillin may be the most suitable candidate in pregnancy and for infants and children in the tooth- and skeletal-development stages. Cephalosporins, erythromycin, and clindamycin may be acceptable alternatives, whilst clinical use of tetracyclines (intrinsic dental staining), aminoglycosides (nephrotoxicity and ototoxicity), and fluoroquinolones (chondrotoxicity in growing cartilage) is not advocated for these patients. Antibiotics with significant nephrotoxicity should not be used in individuals with kidney disease.

- *Choice of bactericidal antibiotics.* Use of bactericidal agents is generally recommended in the management of acute infection and also for the treatment of immunocompromised patients. It is worth highlighting that bacteriostatic agents are no less effective than bacteriocidal equivalents, and most patients can be adequately managed by the former.
- *Consideration of antibiotic therapy cost.* The cost of therapy may be an important factor in determining the antibiotic regimen. Newer drugs, such as broad-spectrum cephalosporins and carbapenems, tend to be more expensive, whilst older antibiotics, including penicillin and agents that are available as generic drugs, may be cheaper. Intravenous administration is generally more expensive than oral antibiotics.

Principles of antibiotic treatment regimen

Although the treatment regimen should be made in accordance with the manufacturer's recommendation on the proper administration route, dosage, and interval, the following factors should be considered:

- *Dose.* Most infections can be managed with a standard dosage of antibiotic. "High dose" (maximum dose recommended by manufacturer) may be advocated if an infection is severe or progressing rapidly, if the involved site has insufficient blood supply or if a patient is immunocompromised.
- *Time intervals.* Strict adherence to the dosing interval recommended by the manufacturer is critical. However, patients with pre-existing renal disease and subsequent decreased antibiotic clearance might require longer intervals between doses to avoid possible excessive plasma levels and a resultant increase in toxicity reactions.
- *Route of administration.* Antibiotics are commonly administered orally because it is easy, non-painful and cost effective. However, the level of drug absorption in the gastrointestinal tract varies widely between individuals. In addition, various factors can affect drug absorption. Intravenous administration may ensure sufficient and consistent level of antibiotic in plasma and infection site. In the case of severe infection or infection with a high risk of serious complication, antibiotics should be administered intravenously. An intravenous antibiotic is also recommended in patients with trismus or difficulty in swallowing.
- *Duration.* Once antibiotic therapy starts, the antibiotic should be administered for an adequate period. Antibiotic treatment is recommended for an additional 2–3 days after clinical resolution of an infection has occurred to avoid recurrence. Osteomyelitis, sinusitis, and suspected actinomycosis may require longer administration, even after clinical signs and symptoms have disappeared.

Consideration of side-effects

Likely side-effects

If an indication of a side-effect appears, change of the antibiotic should be considered. Hypersensitivity (skin rash) and abdominal symptoms (nausea, vomiting, abdominal cramping, loose stool, and diarrhea) are common side-effects. These might occur even if the patient has previous experience of the same antibiotic without side-effects. A loose stool and the occurrence of diarrhea are usually associated with disrupted microbial homeostasis in the gastrointestinal tract, and once administration of the antibiotic ceases, these side-effects may resolve spontaneously. Serum enzyme concentrations often fluctuate to unacceptable levels, indicating deterioration in hepatic or renal function.

Anaphylaxis

Despite a very low incidence, anaphylaxis (anaphylactic shock) is the most serious and life-threatening adverse effect of antibiotics. β -lactam antibiotics, in particular members of the penicillin group, primarily cause this reaction. The clinical signs include diffuse urticaria, stridor, and angioedema. Rapid pulse rate, reduced blood pressure, weakness, heart arrhythmias, mental confusion, and various abdominal symptoms (nausea, vomiting, diarrhea, and abdominal pain or cramping) may appear. Airway obstruction due to the glottic (laryngeal) edema and bronchospasm is an extremely important consideration due to potential fatal consequences. In the vast majority of patients, signs and symptoms appear within 60 minutes after administration. It is parenteral antibiotic administration that usually produces a rapid reaction (within 5 minutes) and therefore patients should be watched for at least 5 minutes after injection.

Anaphylaxis requires immediate professional treatment. The priority is to transfer the patient to hospital as an emergency. The patient's vital signs must be checked and emergency management started with the ABC (airway, breathing, circulation) of resuscitation. Epinephrine (1:1000, 0.5ml) should be injected intramuscularly without delay to open the airways and to raise blood pressure by constricting blood vessels until professional treatment starts. If available, corticosteroids, such as prednisone, should be given to further reduce symptoms after primary lifesaving treatment is achieved. Although it is difficult to predict the likelihood of anaphylaxis in advance, careful interview prior to use of antibiotic is important to reduce its incidence, as a history of previous anaphylaxis and poorly controlled asthma increases the risk.⁷¹ Penicillin skin testing is useful to predict the likelihood of allergic reactions to penicillin. It should be recognized that the result will not establish whether a patient will have an allergic reaction to another type of β -lactam agent.

Medical supportive care

The roles of supportive care are to improve the quality of the patient's life and to encourage resolution of the infection indirectly. These roles may include a wide range of professional and patient self-care interventions.

Inpatient management

The large majority of orofacial infections are usually managed by an outpatient practice. However, there are certain cases in which outpatient management is impracticable or unnecessarily risky. The conditions requiring hospitalisation are listed in Table 29.14.

Management of fluid balance and nutrition

Fever increases fluid loss and calorific needs. Moreover, patients may often have inadequate fluid and nutritional intake, because of poor appetite resulting from sickness and mental depression, oral dysfunction (trismus and difficulty swallowing), and pain. As a consequence, hyponutrition and dehydration may occur. It is worth highlighting that children are much more susceptible to fluid depletion as a result of dehydration and become toxic faster than adults. Patients should be encouraged to drink plenty of water and take sufficient nutrition from meals, high-calorie nutritional supplements or confectioneries. If a patient cannot take adequate fluid and nutrition, hospitalization and professional supportive care, including enteral nutrition and intravenous fluid supplementation, are required.

Pain control

A NSAID is commonly used for pain control. NSAIDs have significant toxicities and risk of side-effects. Careful prescription is required. Paracetamol has generally less toxicity, and therefore it should be administered first. Prescription of a NSAID (recommended drugs may vary between countries) should be considered for control of more severe pain. Some appropriate regimens are presented in Table 29.15. In odontogenic infections, occlusal equilibration may be

Table 29.14 Criteria for hospital admission.

Need for urgent life-saving care, high risk of fatal condition or suspected serious complication
Considerable systemic inflammatory reaction in terms of fever or malaise
Significant trismus, dysphagia or dyspnea
Severe, rapid progressive or extensively spreading infection
Need for intravenous antibiotics
Significant dehydration or malnutrition that may require professional supportive care
Suspected reduced resistance to infection
Need for surgery under general anesthesia

Table 29.15 Appropriate analgesic regimens for management of pain caused by infection.

The first choice regimen		
Paracetamol	2 tablets (500 mg per tablet)	Up to 4 times daily
For moderate pain that may not be managed with paracetamol		
Aspirin	1 tablet (300 mg per tablet)	Up to 4 times daily
Ibuprofen	1 tablet (400 mg per tablet)	Up to 3 times daily
For severe pain that requires a more potent NSAID for its management		
Diclofenac	1 tablet (50 mg per tablet)	Up to 3 times daily
For moderate to severe pain in patients in whom NSAIDs are contraindicated		
Co-codamol	1 tablet	Up to 3 times daily

Regimens are for adults.

A tablet of co-codamol contains 30 mg of codeine phosphate and 500 mg of paracetamol.

effective to reduce pain that may increase when biting or chewing.

Fever control

Mildly elevated body temperature may be of benefit to host defense mechanisms, as immune reactions may be enhanced with elevation of temperature and some pathogens with strict temperature preferences could be hindered by a small rise in temperature. Therefore if the body temperature does not exceed 38.5°C or if the patient has no complaint of fever, control of fever may be unnecessary. Paracetamol and NSAIDs are simple and effective for an unacceptable fever. Overly cold water could stimulate shivering and even increase heat production.

Patient monitoring and evaluation of response to treatment

Once treatment has started, the patient should be carefully and frequently monitored to assess the response to treatment and the occurrence of any complications, side-effects or toxic reactions to the administered medicine. This is achieved through monitoring clinical signs and symptoms (for example, changes in vital signs, extent of swelling and redness, nature of swelling and pain, body temperature, dysfunction level and the patient's subjective feelings of improvement, and blood test data). It should be noted that a patient's condition can become dramatically worse and be more serious in only a matter of hours. More careful monitoring and special attention is required in cases of severe, rapidly progressing infection and immunocompromised patients. In contrast, patients with mild

Table 29.16 Likely reasons for failure of treatment of oral and maxillofacial suppurative infection.

A substantial residual collection of pus (inadequate or insufficient surgical drainage)
Persistence of infection cause or involved foreign body
Impaired host defense mechanisms
Antibiotic factors:
No compliance of patient with prescription order/suggestion
Inadequate dose of antibiotic or insufficient level of antibiotic in infection site
Wrong bacterial diagnosis
Involvement of antibiotic-resistant microorganism

infections without any risk factors may need only self-evaluation at home and return to the clinic for review 2 or 3 days after the original therapy.

In infection without complication, provided that adequate surgical drainage has been achieved, improvement will begin by the second day of therapy. However, in the early stages of infection that appears as cellulitis, drainage of pus is usually not possible. In this case, an antibiotic may be the principle treatment, and it takes several days to determine the actual outcome of such antibiotic therapy. Therefore the treatment regimen should not be changed within short periods, unless a dramatic and rapid deterioration of the condition occurs or an indication of a side-effect or complication appears. Even though improvement may not be obtained in the first 1–2 days, the clinician should resist a temptation to change treatment regimen. Osteomyelitis, maxillary sinusitis, and actinomycosis tend to respond to treatment slower than common odontogenic infections.

If clinical improvement is not seen after an adequate period (usually 48–72 hours after start of treatment), failure of the treatment may be suspected. The most likely cause is inadequate surgical treatment (Table 29.16). Careful re-examination for any undetected abscesses is required. Extraction of the offending tooth should be reconsidered if salvage of the tooth was being attempted. Dental root fracture and fissure, and the presence of a foreign body may be a cause of delay in resolution, and therefore these should be removed.

Depression of the host defense mechanism can be a cause of treatment failure. Undiagnosed or overlooked diseases should be managed adequately. The patient's medical history should be reviewed again, and more careful and probing questioning should be undertaken. Patients are often unaware of pre-existing diabetes; if warranted, the blood sugar level should be checked. A fluid balance and the nutritional level should be reassessed because these factors may affect the outcome of treatment.

Factors associated with the antibiotic may be involved in failure of treatment. It is necessary to confirm if a patient has followed the prescription

order and whether the regimen is adequate. Another problem to consider is whether the antibiotic actually has reached the infection site. Inadequate antibiotic levels at the infection site due to an inadequate blood supply may result in treatment failure. In such instances, an increase in dosage or change in route of administration should be considered. The involvement of antibiotic-resistant bacteria may also be associated with failure. If the initial choice of antibiotic is suspected as being inadequate, the antibiotic should be changed. Alternative antibiotics should be chosen based on microbiologic examination. However, if adequate specimens are not available, a treatment that extends the antibacterial spectrum should be attempted. This could use an alternative antibiotic, a broad-spectrum antibiotic or therapy with a multi-drug combination.

Odontogenic infections

The majority of orofacial infections have an odontogenic origin. Strictly, the term odontogenic infection encompasses all forms of dental infectious diseases. However, odontogenic infections are primarily considered to be pus-producing infections associated with the teeth and the surrounding supportive structures and therefore dental caries and gingivitis do not fall into this category of diseases.

Odontogenic infections consist of three main types: (1) periapical abscesses, which involve necrosis of the dental pulp and subsequent infection of the root canal; (2) periodontal abscesses formed in association of periodontitis; and (3) pericoronitis, which is an infection of the pericoronal soft tissue overlying the crown of a tooth. The large majority of odontogenic infections are of periapical origin. Purulent infection associated with periapical origin is frequently described as a dentoalveolar abscess, although any pus-producing infections involving the teeth and surrounding supportive structures are often regarded as dentoalveolar abscesses.⁷²

A collection of pus constitutes an abscess which is a defensive reaction against the spread of infection. Cellulitis describes a condition where swelling occurs with no significant localization of pus and has a greater tendency for progressive spread. Almost all odontogenic infections show elements of both cellulitis and abscess formation. Infections commonly start as a cellulitis and tend to localize and develop into an abscess over a period of days.

In developed countries, the incidence of severe odontogenic infections has greatly decreased, primarily due to an improved quality and availability of dental care, widespread fluoridation of public water supplies, increased use of toothpastes containing fluoride, and the increased availability and use of antibiotics. However, it must be recognized that odontogenic infections may progress extensively and at an alarming speed and might cause serious and fatal complications.

Types of odontogenic infections

Endodontic diseases

The most common forms of endodontic diseases are pulpitis and periapical abscesses. Pulpitis is an inflammation localized only within the pulp of the offending tooth. In contrast, periapical abscesses are purulent lesions within the surrounding supportive structures around the root apex of teeth. Such abscesses are commonly formed through episodes of pulpitis and subsequent pulpal necrosis (Fig. 29.18).

Almost all endodontic diseases involve dental pulp. Healthy vital pulp is generally sterile and responds to various stimuli without an exaggerated response. Normal pulp responds to cold stimulation with slight pain that disappears within a few seconds. Although electric pulp testers for assessment of the status of pulp are available, the results are not highly reliable.

Whilst pulpitis is not an odontogenic infection, it can be misdiagnosed as a periapical disease; its discussion here is, therefore, warranted.

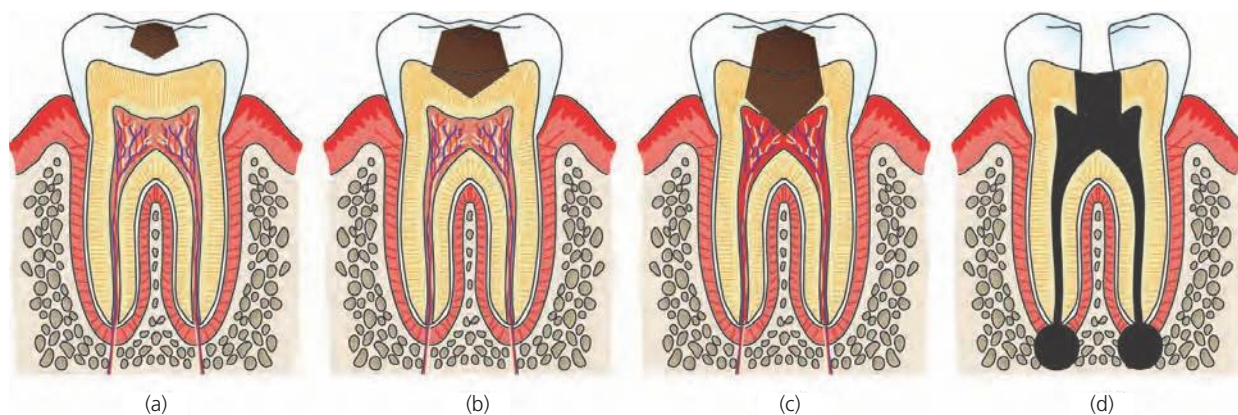


Fig. 29.18 The process from onset of dental caries to pulp necrosis. (a) Early stage of caries. (b) Advanced stage of caries, with possible reversible pulpitis. (c) Irreversible pulpitis with entry of bacteria into pulp cavity. (d) Pulp necrosis and formation of periapical abscess.

Pulpitis

Although various factors including physical, thermal, and chemical irritants are involved in pulpitis, bacterial factors, especially deep dental caries, are the most important in the etiology of pulpitis. Pulpal diseases range from reversible hyperemia to an irreversible pulpitis ending in necrosis. The clinical features of reversible pulpitis and irreversible pulpitis are summarized in Table 29.17. Reversible pulpitis generally produces no significant pain. On occasion, a mild painful response to stimulus (cold temperature) occurs, but this commonly subsides within a few seconds without additional pain or discomfort once the stimulus is removed.

Irreversible pulpitis is divided into acute and chronic forms. Acute pulpitis produces a throbbing and intensive pain. The pain involves an elevated pressure within the pulp cavity due to inflammation of the pulpal tissue. Since temperature increases pressure, an elevation in body temperature or application of a thermal stimulus (e.g. hot water) to the tooth may intensify the pain. Therefore patients often report that their pain becomes more intense when taking a bath, drinking alcohol or tea, or being in bed at night (body temperature is generally elevated in the evening and night). Although application of a cold liquid to the tooth sometimes reduces intensity of the pain, it may also provoke pain. Painful response to cold liquids usually lingers minutes even after the stimulus is removed. The pain is often difficult to control even with potent types of NSAIDs. As a troublesome feature, patients often misidentify the causative tooth, even confusing the maxillary tooth with mandibular one, but not the left and right sides of the mouth. This may lead to misdiagnosis in localizing the source of pain. Pulpal inflammation may affect the periodontal ligament, resulting in a painful response to mastication and the percussion test, although pulpitis does not always provide elevated sensitivity to the latter.

Chronic pulpitis usually does not produce a throbbing pain but does respond to cold and thermal

stimuli. Chronic pulpitis is often found in cases where the pulp has been exposed to the oral cavity. The disease process may occur for several months to several years, and also spontaneously transform into an acute infection.

Bacterial infection usually occurs in irreversible pulpitis. Viridans streptococci, lactobacilli, peptostreptococci, and anaerobic Gram-negative bacilli are often associated with these infections.^{73,74} Exposed dentinal tubules and direct pulpal exposure caused by operative procedures, trauma or deep dental caries can allow entry of bacteria into the pulp. Accessory and lateral canals can also provide direct channels for entry of bacteria. In the case of severe periodontitis, bacteria residing in the periodontal pocket may enter into the pulp through an accessory or lateral canal or the root apex. Infected regions of pulp will necrotize, and sooner or later necrosis will extend throughout the canal system. Once the whole pulpal system has necrotized, clinical symptoms of pulpitis will cease.

Diagnosis is based on medical history and physical examination of the reactions provoked with various stimuli (application of heat, cold, and percussion). Pulpitis does not provide a specific radiological change in the tooth and periodontal tissues. However, as pulpitis usually involves deep dental caries or severe periodontitis, radiographs can provide a precise localization of the source and determine whether inflammation has extended beyond the tooth apex.

The vitality of a pulp with reversible inflammation may be maintained by conventional and conservative dental treatment. In contrast, irreversible pulpitis requires endodontic therapy (pulpotomy or pulpectomy) or tooth extraction. As pulpitis does not respond to antibiotic therapy, systemic antibiotic is unnecessary.

Periapical abscess

Periapical abscesses are also described as periapical infections and periapical or apical periodontitis. Periapical abscesses commonly involve pulpal necro-

Table 29.17 Summary of the clinical features of reversible pulpitis, acute irreversible pulpitis, and acute periapical abscesses

	Reversible pulpitis	Irreversible pulpitis	Periapical abscess
Pulp vitality	Yes	Yes	No
Response to cold stimulation	Mild, disappearing within a few seconds	Often severe, lingering for minutes	No
Response to thermal stimulation	Little or no	Yes	No
Response to percussion test	Little or no	Sometimes yes	Yes
Pain at chewing	Little or no	Sometimes yes	Yes
Spontaneous pain	No	Yes	Yes
Enhanced pain intensity at night	No	Yes	No
Controllability of pain with NSAID	Not applicable	Often uncontrolled	Mostly controlled
Misidentification of the causative tooth	Usually no	Often yes	Usually no
Redness, tenderness, and swelling of the vestibule site	No	No	Yes
Response to antibiotic therapy	No	No	Yes

sis resulting from irreversible pulpitis, although failed endodontic treatment also induces the disease.

The necrotic pulp of the root canal serves as an important source of nutrition for microorganisms, and it is usually infected with a number of commensal oral microorganisms. Since the apical periodontal ligament is directly linked with the pulp system, the bacteria, bacterial products, and toxins released from the necrotic pulp may diffuse from the root canal into the periapical tissues with resultant development of a periapical inflammatory lesion. The host response to such an infection involves the building of a defensive barrier in an attempt to confine the pathogens and their toxins within the root canal system and periapical tissues. The most common sequelae of pulpitis is the formation of chronically inflamed periapical granulation tissue.³ Sometimes, the squamous epithelium of an apical granuloma proliferates, forming a cyst. An abscess may develop around the root of the tooth, either directly or after the formation of periapical granulomas and cysts. Chronic and asystematic episodes may continue whilst microbial homeostasis is maintained. However, once microbial homeostasis in the lesion is disrupted, the bacteria proliferate and invade the tissue, accompanied by acute inflamma-

tion. In acute periapical abscesses, the periodontal ligament is inflamed and the tooth often pushed slightly out of the tooth socket. Consequently, more intense pain during chewing and reaction to the percussion test with increased mobility of the tooth are evident. If any purulent exudate or pus penetrates the jawbone, infection may spread to intraoral or extraoral soft tissues. The natural history of progression and bacteriology of periapical abscesses is described later.

A periapical radiograph usually demonstrates radiolucency around the apex tip of the tooth. However, in cases with little destruction of periapical bone, significant change at the periapical region is not readily seen by radiographs.

Treatment options for an acute periapical abscess are discussed later. Once acute inflammation subsides, the disease is treated with endodontic therapy, apicectomy or extraction of the tooth.

Periodontal disease

Periodontal disease is a chronic infectious disorder that affects the soft tissue and bone supporting the teeth. This disease is initiated by a very complex microbial biofilm that develops on the tooth surface adjacent to the soft tissues of the periodontium. Periodontal disease often affects multiple teeth.

Periodontal diseases are generally divided into gingivitis and periodontitis. Gingivitis is the mildest form of periodontal disease, whilst periodontitis is the advanced form, and their clinical features are distinct (Fig. 29.19 and Table 29.18). In particular, occurrence of bone loss is the most important distinction between gingivitis and periodontitis (Fig. 29.20).

Periodontal disease is not necessarily a continuum from gingivitis. The duration of onset and the intensity of the periodontal inflammatory process vary considerably in individuals and also with tooth site



Fig. 29.19 Gingivitis is the mildest form of periodontal disease, with periodontitis being the advanced form. In contrast with gingivitis, periodontitis involves bone loss, which is the most important feature of the infection.

Table 29.18 Summary of clinical features of gingivitis and periodontitis.

	Gingivitis	Periodontitis*
Patient's age	Any age	Usually over 35
Gingival swelling	Yes (especially gingival margin and papilla)	Often yes, but sometimes slight swelling
Easy bleeding of gingiva	Yes	Often yes, but sometimes slight bleeding
Response to percussion test	No	Often yes
Bone loss	No	Yes
Nature of pocket	Gingival pocket due to gingival swelling	Periodontal pocket due to bone destruction
Halitosis	Generally no, but sometimes yes	Yes
Persistent metallic taste in the mouth	Generally no, but sometimes yes	Yes (depending on severity)
Gingival recession	No	Yes
Pulp vitality	Yes	Yes
Reversibility of tissue change	Yes	No
Possible cause of periodontal abscess	No	Yes
Possible cause of tooth loss	No	Yes

*Except mild cases.

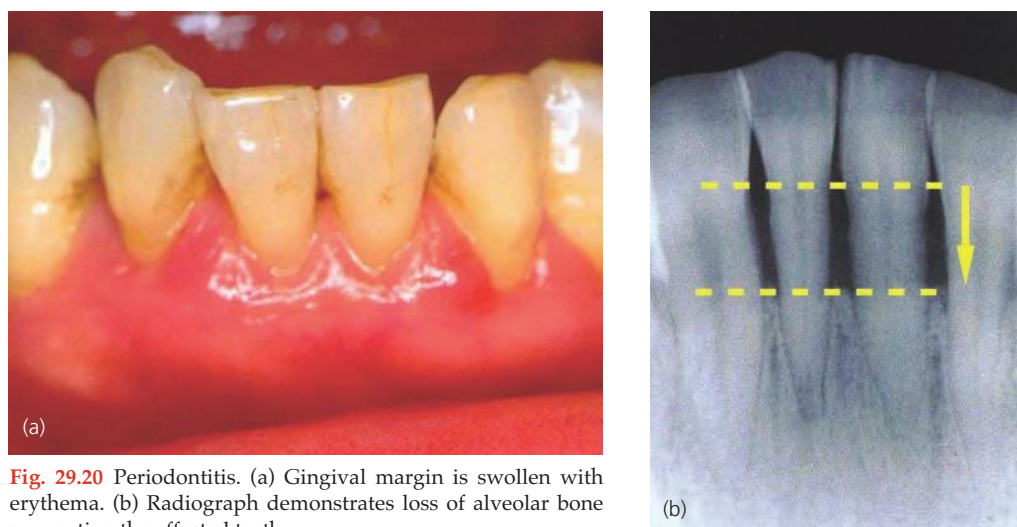


Fig. 29.20 Periodontitis. (a) Gingival margin is swollen with erythema. (b) Radiograph demonstrates loss of alveolar bone supporting the affected teeth.

within the same patient. Although gingivitis and periodontitis may not be regarded as odontogenic infections, their description here is necessary, as an understanding of their pathogenesis and clinical features are important for the diagnosis of odontogenic infections. Necrotizing ulcerative gingivitis, an uncommon form of periodontal disease, is described in a separate section.

Gingivitis

Gingivitis is inflammation of the gingiva, and involves plaque that accumulates in the gingival margins due to inadequate oral hygiene. Gingivitis causes little pain in its early stages and may go unnoticed. The gingivae appear red rather than a healthy pink and become movable instead of being firm and tight against the teeth. Due to the accumulated plaque, the gingiva may bleed readily, especially during tooth brushing or eating. Gingival swelling results in an increased depth of the gingival sulcus (gingival pocket formation), but gingivitis does not result in bone loss or destruction of periodontal tissues. It is important to recognize that gingivitis itself is unlikely to be a cause of orofacial purulent infections. Misaligned teeth, the rough edges of fillings, and ill-fitting or unclean dentures, bridges, and crowns increase the risk of gingivitis because of increased plaque retention. Pregnancy, stage in menstrual cycle, use of birth control pills, the onset of puberty, and diabetes mellitus are all predisposing factors to this condition.⁷⁵ Some drugs such as phenytoin, ciclosporin and calcium channel blockers (antihypertension agents) can cause an overgrowth of gingival tissue, which may disturb the maintenance of good oral hygiene. In cases of leukemia, the gingiva may also become engorged with a leukemic infiltrate.

Gingivitis can be prevented through maintaining good oral hygiene by the patients themselves or by professional care. Gingivitis may be completely reversible with appropriate oral hygiene practice,

although excess gingival tissue caused by intake of certain drugs may need to be removed surgically.

Periodontitis and periodontal abscess

Periodontitis is a severe form of periodontal disease in which the inflammation of the gingiva extends to the supporting structures of the tooth. As with gingivitis, periodontitis primarily involves dental plaque and calculus, but other factors may also be associated with its etiology. Suggested risk factors include old age, compromised host defense, heredity, history of previous periodontitis, male gender, inadequate dental habits, poor oral hygiene and dental care, inadequate margins on crowns and dental restorations, presence of specific bacteria including *Aggregatibacter actinomycetemcomitans*, *Tannerella forsythensis*, and *Porphyromonas gingivalis*, race and ethnicity, stress, diabetes, specific systemic diseases, and tobacco smoking.^{76,77}

The most significant hallmarks of periodontitis are bone loss and formation of a periodontal pocket. Destruction of the periodontal tissues and alveolar bone loss are primarily a result of the excessive and continued host inflammatory response to bacterial products. The most common symptoms include bleeding and gingival tenderness, pain during chewing, malodor, and bad taste with accompanying gingival recession. As infection erodes the bone holding the teeth in place, affected teeth become loose and shift in their position, and eventually they may fall out or need to be extracted.

Periodontitis is classified into several subtypes.^{75,78} Chronic periodontitis is generally characterized by a slow progression of the disease. However, there may be bursts of destruction, and the rate of disease progression may be modified by local, systemic, and extrinsic factors. Chronic periodontitis was previously termed “chronic adult periodontitis”, since it is most prevalent in adults (≥ 35 years of age). However, it can be also seen in adolescents, and consequently

the age-dependent terminology has been removed. Bacterial species involved in chronic periodontitis include *Porphyromonas gingivalis*, *Tannerella forsythensis*, and *Aggregatibacter actinomycetemcomitans*. In addition, other types of bacteria such as *Campylobacter rectus*, *Eubacterium nodatum*, *Fusobacterium nucleatum*, *Prevotella intermedia*, *Peptostreptococcus micros*, and spirochetes can also be associated.⁷⁷

Aggressive periodontitis is distinct from chronic periodontitis. This disease is characterized by rapid attachment loss and bone destruction, and possible familial aggregation of disease. Other likely features include periodontal tissue destruction that is greater than would be expected given the level of local factors (plaque and calculus), elevated levels of *A. actinomycetemcomitans* or *P. gingivalis*, phagocyte abnormalities, and increased production of PGE₂ and IL-1b.⁷⁸ It is important to recognize that patients are often otherwise systemically healthy. Since aggressive periodontitis often occurs in people under 35 years of age, it was previously called “juvenile periodontitis”. However, this is not exclusive and may also be seen in older patients.

Periodontitis as a manifestation of systemic disease often occurs at a young age. Systemic diseases that affect the immune function, inflammatory response, and tissue organization can modify the onset and progression of all forms of periodontal disease. Such diseases include hematological disorders (acquired neutropenia, leukemias, and others) and genetic disorders (familial and cyclic neutropenia, Down’s syndrome, leukocyte adhesion deficiency syndromes, Papillon–Lefevre syndrome, Chediak–Higashi syndrome, histiocytosis syndrome, glycogen storage disease, infantile genetic agranulocytosis, Cohen syndrome, Ehlers–Danlos syndrome types IV and VIII, and hypophosphatasia). Management of periodontal disease under such circumstances should be carried out in conjunction with management of the associated systemic disease.

In recent decades, epidemiologic association between periodontitis and specific systemic conditions, including coronary heart disease, atherosclerosis, diabetes, and a higher risk of preterm low birth-weight babies, has been recognized (Fig. 29.21).^{79–81} However, the mechanisms for the various relationships remain unclear, and there are debates over actual impact of periodontal therapy on improvement of the systemic condition.

Diagnosis of periodontitis is based on the inspection of the teeth and gingiva, combined with probing of the pockets, measurement of their depth, and radiographic examination. Although mild to moderate periodontitis may be managed by the patient themselves or professional treatment, severe cases usually require extraction of the tooth.

Periodontal abscesses are associated with periodontal breakdown and localized pus in the gingival wall of the periodontal pocket. Acute abscesses are painful, edematous, red, shiny, and ovoid elevations

of the gingival margin and/or attached gingiva (Fig. 29.22). A disturbance of microbial homeostasis, destruction of the epithelial barrier or random events may result in proliferation of bacteria residing in the periodontal pocket, with the generation of a suppurative lesion. In particular, an obstruction in the drainage of the periodontal pocket by debris is the most common trigger for the onset of abscess formation. It should be noted that obstruction of drainage may involve calculus and debris that are pushed into gingival tissue during professional periodontal treatment such as scaling and root planing. Although periodontal abscesses are closely associated with periodontitis, the bacteriology is more similar to periapical abscesses than periodontitis. A well localized, small abscess can easily be managed with surgical drainage without administration of systemic antibiotics.

Pericoronitis

Since the oral hygiene around third molars tends to decline due to anatomic reasons, the gingivae surrounding the crown of the third molar often become inflamed. This condition is called *pericoronitis* (Fig. 29.23). *Pericoronitis* commonly occurs with the mandibular wisdom tooth that has not fully emerged. The flap of gingiva over the partially emerged tooth can trap fluids, food debris, and bacteria. In cases where the maxillary third molar emerges before the mandibular one, the cusp of the former can damage the flap of the latter, causing irritation. Once the drainage of exudate from the gingival pocket is blocked by impaction of foreign bodies or by trauma-induced swelling of the operculum, an acute infection may occur. The bacteriology of *pericoronitis* is similar to that of other forms of odontogenic infections.

Pericoronitis typically occurs in teenagers and young adults, presenting shortly after the third molars start erupting. It presents as an erythematous, tender, sessile swelling of gingiva and the retromolar pad. *Pericoronitis* does not often progress into the development of an abscess. Acute *pericoronitis* provides a quite intense pain that may radiate to the external neck, the throat, the ear, and the oral floor. Since the third molar is adjacent to the muscles of mastication, infection often affects the muscles, especially the medial pterygoid muscle, resulting in trismus. *Pericoronitis* of the mandibular third molar can become serious since infection may readily spread to the opposing jaw, pharynx and deep fascial spaces.

Systemic antibiotic therapy is recommended unless infection is quite mild with no associated immunocompromising factors. In well localized *pericoronitis*, local antiseptic lavage and gentle curettage under the flap may be effective. Once the acute inflammation subsides, the offending tooth should be extracted or a wedge of hyperplastic pad tissue could be surgically removed.

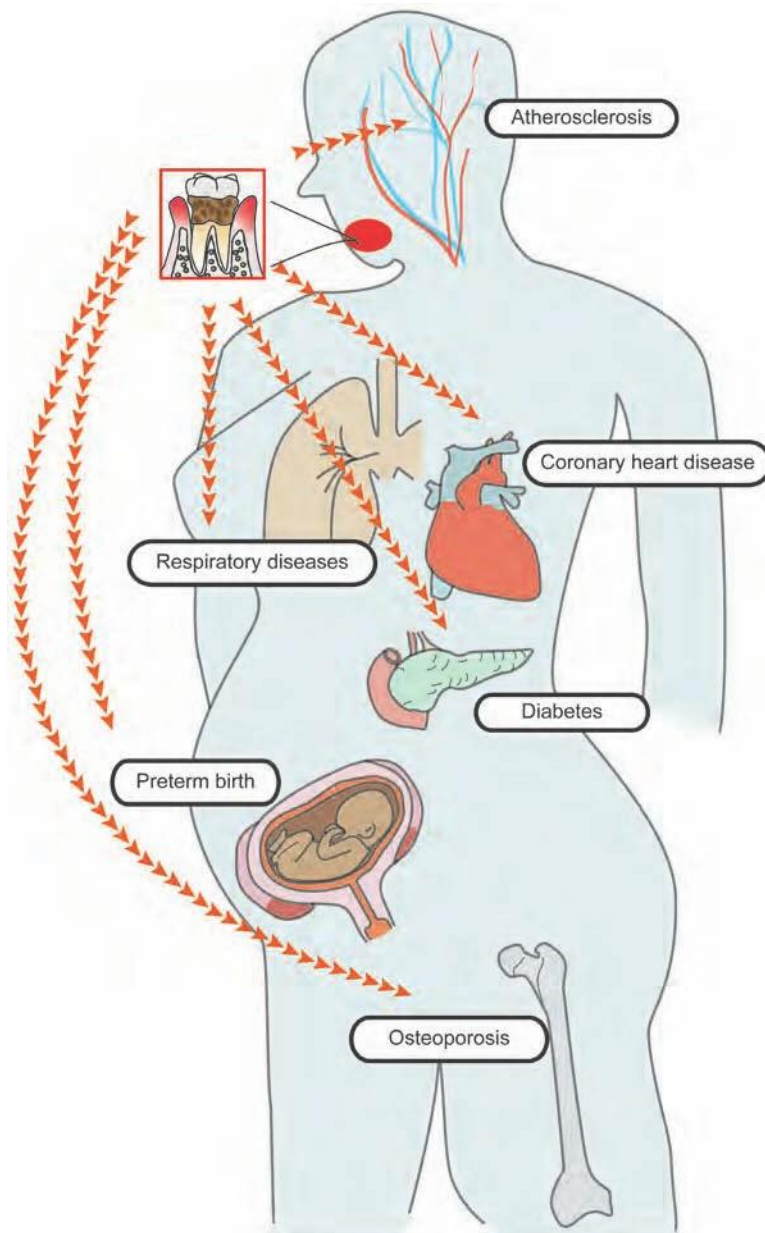


Fig. 29.21 Periodontitis has been considered to increase the risk of atherosclerosis, coronary heart disease, respiratory diseases (pneumonia), diabetes mellitus, osteoporosis, and preterm birth.



Fig. 29.22 Images of periodontal abscesses. Swelling around the gingival margin is evident. Spontaneous drainage of pus from periodontal pocket is often seen.

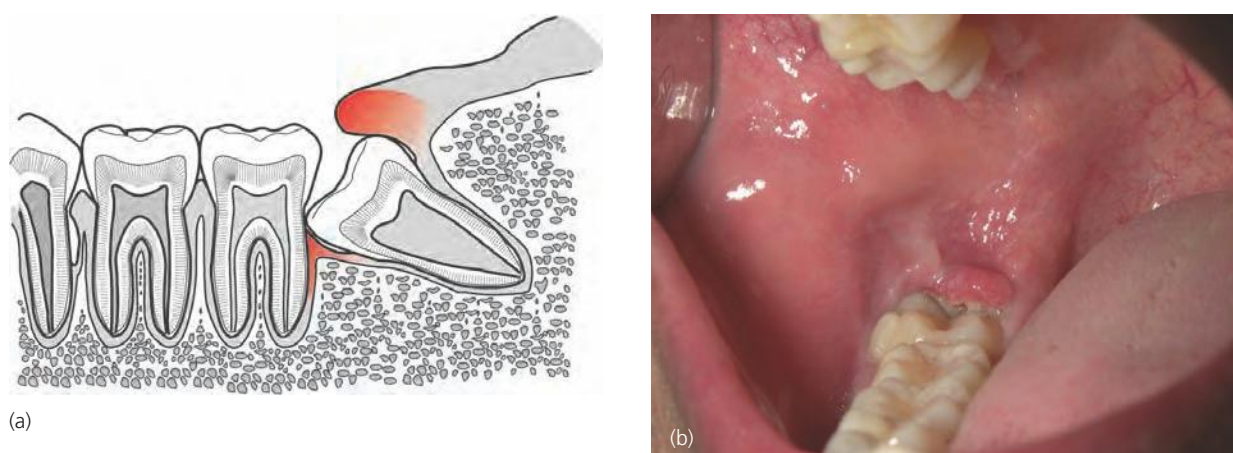


Fig. 29.23 Acute pericoronitis in the mandibular third molar. (a) The flap of gingiva over the partially emerged tooth can trap fluids, debris, bits of food, and bacteria, which are involved in the infection. (b) The erythema and swelling of gingiva and the retromolar pad are evident.

Bacteriology of odontogenic infections

Odontogenic infections are usually polymicrobial, with four to five or even more bacterial species isolated.³⁷ Most bacteria involved are of low virulence, so microbial synergism is believed to play an important role in development of infection.^{72,82}

There are no notable differences in the microbiology of periapical abscesses, periodontal abscesses, and pericoronitis.³⁷ The vast majority of species recovered from odontogenic infections are normal residents of the oral cavity. Although various organisms are detected, certain organisms are more frequently recovered than others (Table 29.19).^{37,72,82–84} These include viridians streptococci, peptostreptococci, and Gram-negative, strictly anaerobic bacilli. These organisms are considered to play a determinative role in the infection process and are regarded as major pathogens. Recent investigations have high-

lighted a predominance of strictly anaerobic bacteria. Strict anaerobes are reported to be recovered in more than 90% of the infections. Gram-negative anaerobic bacilli, including *Prevotella*, *Porphyromonas*, and *Fusobacterium* species are the most frequent isolates from odontogenic infections.^{37,82–84} Commonly recovered *Prevotella* species include *P. intermedia*, *P. nigrescens*, *P. melaninogenica*, *P. oralis*, *P. buccae*, and *P. oris*.⁸⁵ *Porphyromonas* species, especially *P. gingivalis*, are also often recovered from all forms of the disease. *Fusobacterium nucleatum* and its subspecies are the predominant form of fusobacteria involved in odontogenic infections. The involvement of *Tannerella forsythensis* (*Bacteroides forsythus*) and *Treponema* species may be overlooked because they are often not recovered by culture, but nevertheless are frequently detected by molecular analysis.^{82,86}

The predominant streptococcal bacteria belong to the *S. anginosus* group (*S. milleri* group), whilst *S. sanguinis* and *S. salivarius* are rarely recovered.

Table 29.19 Bacteriology of odontogenic infection. Identification of isolates from pus specimen from 87 patients, Kanazawa University Hospital, between September 2001 and March 2005.

Aerobic and facultative bacterial species	Percentage of isolates	Anaerobic bacterial species	Percentage of isolates
Viridans streptococci	6	<i>Prevotella</i> species	34.4
<i>Corynebacterium</i> species	3.1	<i>Fusobacterium</i> species	14.6
<i>Staphylococcus</i> species	2.2	<i>Peptostreptococcus</i> species	13.5
<i>Lactobacillus</i> species	0.7	<i>Porphyromonas</i> species	4.9
<i>Capnocytophaga</i> species	0.4	<i>Veillonella</i> species	2.9
<i>Neisseria</i> species	0.4	<i>Bacteroides</i> species (bile-sensitive)	1.6
Unidentified aerobic Gram-negative bacilli	2.7	<i>Eubacterium</i> species	0.4
		<i>Actinomyces</i> species	0.4
		<i>Bifidobacterium</i> species	0.2
		<i>Propionibacterium</i> species	0.2
		Unidentified anaerobic Gram-negative bacilli	12.2
		Unidentified anaerobic Gram-positive cocci	0.7

Streptococcus mutans is rarely isolated despite being the prime pathogen of dental caries. There is debate over the potential patho-etiological importance of *Staphylococcus aureus*. Although isolation of this organism often arises through contamination of the specimen at the time of sampling or in the laboratory, *S. aureus* may be relevant in the development of infection in some cases.

Most of the major bacterial pathogens are generally susceptible to penicillin (Table 29.20), although some published studies suggest a high prevalence of penicillin-resistant isolates.⁸⁷ However, 30–60% of *Prevotella* isolates exhibit resistance to a wide range of β -lactam antibiotics, including penicillin and even broad-spectrum cephalosporins.^{31,88} This resistance involves bacterial production of β -lactamase.

Pathology of odontogenic infections

Odontogenic infections are opportunistic. Normally, the host defense systems maintain the homeostasis between host and bacteria. However, if the homeostasis is disrupted, bacteria may start to proliferate and invade the tissues. Various local and general factors may be involved in the disruption of homeostasis. The transient decline of host defense activity might trigger the disruption of microbial homeostasis. Acute viral nasopharyngitis (common cold), fatigue, insomnia, and poor appetite often act as triggers. In addition an inadequate use of the root canal file beyond the apex, which creates bleeding or increased exudation into the canal, during endodontic treatment for chronic periapical disease, might provide a stimulus for the overproliferation of microorganisms.

Invasion of pathogens into the tissue causes a rapid and non-specific inflammatory response, which may be expressed as local inflammatory signs, including pain, swelling, redness, heat, and dysfunction, and systemic symptoms, such as general discomfort, fever, and balance disorders. Systemic conditions that compromise the host defense mechanisms, such as diabetes mellitus, and receipt of corticosteroids, anti-cancer agents, and immunosuppressants, increase the likelihood of occurrence of acute infection and risk of extensive progression.

The natural history of progression and clinical features

Bacterial infections of dental origin have a characteristic natural history and clinical features. In acute infection of periapical origin, the periodontal ligament is initially inflamed. In the case of periodontal abscess and pericoronitis, acute inflammation occurs first in the involved gingiva and subsequently in the periodontal ligament. Infection may readily spread to the alveolar bone surrounding the offending tooth and produce considerable pain when exudate and pus are under pressure within bone. Pain also occurs in the gingiva and mucosa around the tooth, usually accompanied by redness, tenderness, and edematous swelling. Intense inflammation of the periodontal ligament results in a slight lift of the tooth from the socket with resultant pain during chewing and increase in the tooth mobility. Regional lymph nodes may be enlarged and tender. An increase in the leukocyte count in the peripheral blood is evident, together with elevation in the percentage of neutrophils. The CRP level is usually elevated even in the early stage of infection. Fever and malaise may appear as systemic signs and symptoms.

The infection spreads equally in all directions but preferentially along the lines of least resistance. In cases of periodontal abscesses, drainage is likely to occur through the periodontal pocket. This may allow spontaneous and constant drainage of purulent material through the periodontal pocket. This is the reason why periodontal abscesses seldom undergo extensive spread. In the case of periapical abscess, the spread is primarily dictated by the thickness of the bone overlying the apex of the tooth (Fig. 29.24). The infection spreads through the cancellous bone and penetrates the thinnest part of the cortical plate. After the infection erodes through the alveolar bone and overlying periosteum, the location where infection appears in the soft tissue depends on the relationship of the perforation of bone to muscle attachments of the maxillary and mandibular bones (Fig. 29.25).

In the case of maxillary teeth, infection commonly erodes through the labiobuccocortical plate (Fig. 29.26 and Table 29.21). If infection perforates through the bone below the attachment of the muscle to maxilla,

Table 29.20 *In vitro* susceptibility of major pathogens of odontogenic infections. University Dental Hospital of Wales, 1999–2003.

	Susceptibility (%)				
	Penicillin	Erythromycin	Tetracycline	Clindamycin	Metronidazole
<i>Prevotella</i> species	70	94	80	100	100
<i>Peptostreptococcus</i> species	100	95	100	100	100
Viridans streptococci	100	98	100	100	0
<i>Fusobacterium</i> species	96	13	98	100	100
<i>Porphyromonas</i> species	100	100	100	100	100

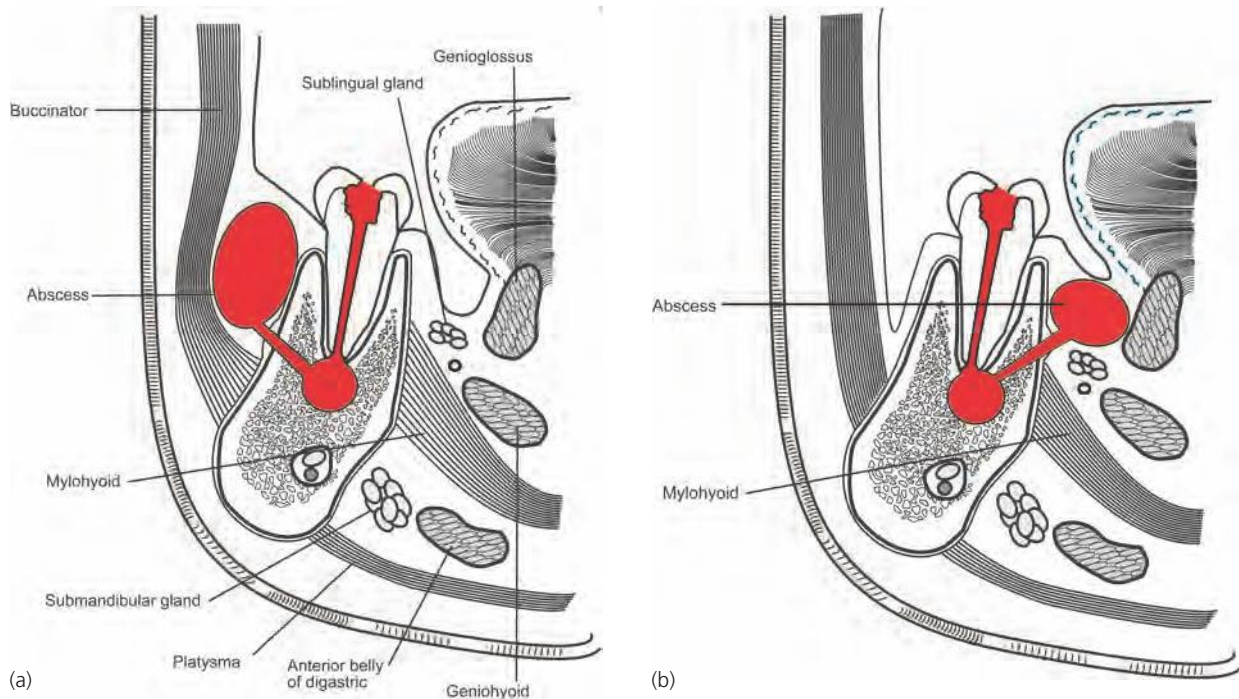


Fig. 29.24 The modes of infection spread from alveolar bone to the overlying soft tissue through penetrating the cortical plate in the case of the mandibular molar tooth. (a) If the buccal bone overlying the apex of the tooth is thinner than the bone on the lingual aspect of the tooth, infection is likely to erode buccal cortical bone and spread buccally. (b) If the buccal bone overlying the apex of the tooth is thicker than the bone on the lingual aspect of the tooth, infection may go into the lingual soft tissue (sublingual space).

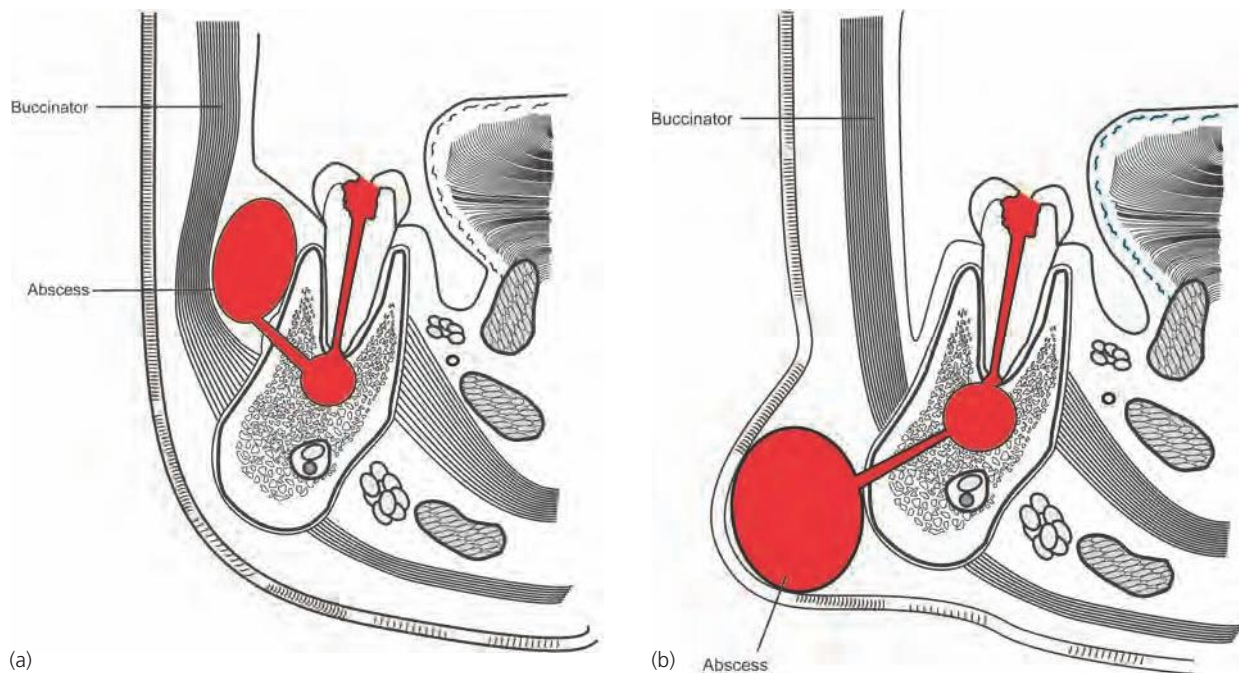


Fig. 29.25 The modes of infection spread to soft tissue after penetrating alveolar bone, when purulent exudate or pus erodes buccal cortical bone. Once the infection has penetrated the alveolar bone, the precise location of abscess formation is determined by the position of the perforation relative to the muscle attachments. (a) If the infection has eroded through the bone superior to the attachment of the buccinator muscle, it will appear as a vestibular abscess. (b) If infection has eroded through the bone inferior to the attachment of the buccinator muscle, it will be expressed as an infection of the buccal space.

vestibular swelling (abscess) will appear (Fig. 29.27). However, in the case of a long maxillary canine, infection will erode through the bone superior to the insertion of the levator anguli oris and will cause a canine space infection. In premolars and molars, if the infection erodes through the bone superior to the insertion

of the buccinator muscle, it may result in a buccal space infection (Fig. 29.28). A palatal swelling (abscess) may occur in the case of a severely inclined incisor (especially lateral incisor) or the palatal root of a molar. As the apices of the second premolar and the first and second molar teeth are located in close

Table 29.21 Possible mode of spread of odontogenic infection.

Involved teeth	Usual direction of spread	Site of localization
Maxillary central incisor	Labial	Labial vestibule
Maxillary lateral incisor	Labial	Labial vestibule
	Palatal	The palate
Maxillary canine	Labial	Labial vestibule or canine space
Maxillary premolars	Buccal	Buccal vestibule or buccal space
	Palatal	The palate
Maxillary molars*	Buccal	Buccal vestibule or buccal space
	Palatal	The palate
Mandibular central and incisor	Lateral	Labial vestibule or submental space
Mandibular canine	Lateral	Labial vestibule
Mandibular premolar	Buccal	Buccal vestibule
Mandibular first molar	Buccal	Buccal vestibule
	Lingual	Sublingual space
Mandibular second molar	Buccal	Buccal vestibule
	Lingual	Sublingual space or submandibular space
Mandibular third molar	Lingual	Submandibular space or pterygomandibular space

* Infection of buccal roots of maxillary molars commonly spreads buccally, whilst the palatal root may be involved in palate abscess.

relation to the floor of the sinus, infection sometimes spreads to the maxillary sinus.

In the mandible, infections of the incisors, canines, and premolars commonly erode through the labio-buccocortical plate of bone and above the associated musculature. Therefore, vestibular abscesses are commonly formed. In contrast, infections of molar teeth can spread either buccally or lingually. Infection from the first molar is likely to perforate buccally, whilst infection of the third molars usually erodes lingually. Infections spreading buccally will produce vestibular abscesses or buccal space abscesses. In the case of infection spreading lingually, the mylohyoid muscle determines if infection erodes to the sublingual or submandibular space.

An early stage of infection commonly takes the form of cellulitis, which is characterized by diffused swelling, no significant localization of pus, and a tendency of extensive spread. The swelling largely results from edema, and the degree of its hardness depends on the intensity of edema. A significant edema may produce a firmer or hardened (indurated) swelling, accompanied by a shiny appearance, warmth, and redness of the skin or mucosal surface. As the amount of interstitial fluid in the soft tissue reduces, swelling becomes softer and reduced.

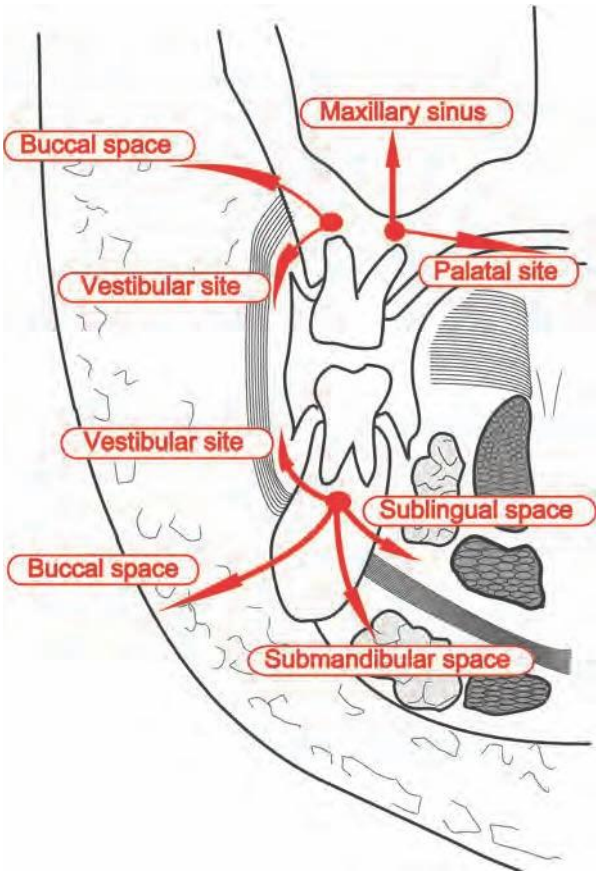


Fig. 29.26 Infection of the maxillary molar may spread to vestibular site, buccal space, palatal site or the maxillary sinus. Infection of mandibular molar will spread to vestibular site, buccal space, sublingual space or submandibular space.



Fig. 29.27 Vestibular abscess.

However, if the induration involves the cellular infiltrate of inflammation (and not fibrosis), the time required for resolution may be prolonged.

Once infection has peaked, pus may accumulate centrally within the swelling. Pus tends to move under influences of pressure, gravity, local heat, and/or action of muscle layers. When pus reaches an internal or external surface, it can discharge spontaneously. At periodontal disease sites, the periapical infection may drain through the deep periodontal pocket and form a periodontal–endodontic lesion. In general, the symptoms and signs of patients may improve dramatically after the pus has discharged.



Fig. 29.28 Buccal space infection.

Management

The principles of management are presented in the earlier section, Principles of management of bacterial infections. Surgical drainage is the most important part of management. If pus has been drained adequately, clinical signs and symptoms will dramatically improve. Failure of surgical drainage results in further progression of infection or prolonged period for resolution.

Use of systemic antibiotics does contribute to preventing the spread of infection and onset of serious complications, whilst most odontogenic infections are mild and can be managed with surgical procedure alone. An antibiotic regimen is usually determined empirically (Table 29.22). A penicillin class of antibiotic remains the first choice drug for treatment of odontogenic infections due to their suitable antimicrobial activity, low incidence of adverse effects, their bactericidal properties, and cost effectiveness. The actual choice of antibiotic and the dose may differ between countries due to different guidelines in therapeutic management. For example, in the UK and Japan, amoxicillin is recommended because of its better absorption from the gastrointestinal tract and high peak concentration in the serum.⁸⁹ In North America, penicillin V is often preferred, probably due to its narrow spectrum of activity and its cost effectiveness.⁹⁰ Although cephalosporins generally have a broader spectrum of activity compared with penicillin, their antimicrobial activity level against the major pathogens is not notably superior to that of penicillins.³¹ In the case of patients who have possible

Table 29.22 Antibiotics that may be used for treatment of odontogenic infections.

Very effective
Penicillin
Clindamycin
Metronidazole (alone or in combination with a β -lactam)
Cephalosporins
Carbapenems (e.g. imipenem)
β -lactam antibiotics combined with β -lactamase inhibitors (e.g. co-amoxiclav)
Effective
Macrolides (e.g. erythromycin or azithromycin)
Tetracyclines
Quinolones
Less effective
Sulfa drugs
Aminoglycosides

penicillin hypersensitivity, use of erythromycin, clindamycin, and metronidazole is recommended. These agents are also suitable alternatives when the causative bacteria are suspected as being penicillin resistant.

Fascial space infections

Orofacial infection can extensively spread superiorly to the skull base and inferiorly to the diaphragm. The spread of infection is largely determined by location and direction of fasciae. Infection generally spreads through the path of least resistance in subcutaneous connective tissues and along fascial planes, with separation of the layers of fascia. Purulent exudates, including pus and gas, tend to collect at specific locations, forming a space (termed fascial space). Fascial spaces commonly form in loose connective tissue between muscles or skin and bone, and have real and potential paths to communicate with other spaces.^{91,92} The communications between spaces are presented in Fig. 29.29. Infections can progress extensively and rapidly, affecting several spaces. This type of infection is called fascial space infection.

Each fascial space infection produces characteristic clinical signs and symptoms, which are associated with the anatomy. There are vital structures around deep fascial spaces in the head and neck. Involvement of these spaces may result in occurrence of serious complications, such as airway obstruction and mediastinitis. In the past, these infections resulted in a high mortality rate. Although the prognosis has now been much improved due to the establishment of modern therapy, fascial space infection remains a significant health problem with links to fatal complications.

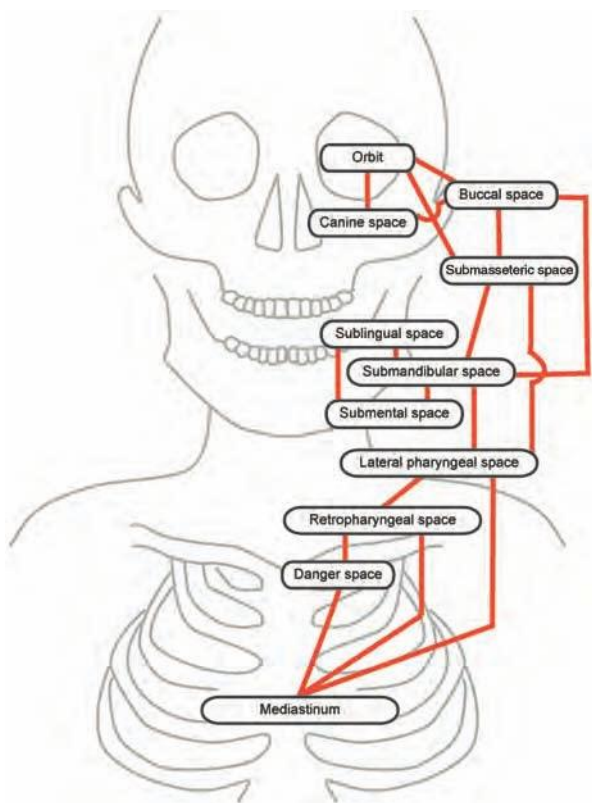


Fig. 29.29 The relation and communication between fascial spaces in oral and maxillofacial regions.

Etiology and microbiology

Fascial space infections can arise from many causes, the principle ones being diseases of the mouth, pharynx, tonsil and salivary glands, and surgical wound and trauma infections. Odontogenic infection is generally the most common cause.

As the majority of fascial space infections begin as odontogenic infections, the causative organisms of both diseases are similar (Table 29.23). The infections usually yield facultative and strictly anaerobic bacteria that reside within the oral cavity.^{93–95} However, in infections involving deep cervical spaces, such as the lateral pharyngeal and retropharyngeal spaces, *Staphylococcus aureus*, *Streptococcus pyogenes*, and *Klebsiella pneumoniae* are often recovered.

Table 29.23 Microorganisms recovered frequently from fascial space infection.

Aerobic and facultative bacterial species	Anaerobic bacterial species
Viridans streptococci	<i>Prevotella</i> species
<i>Staphylococcus aureus</i>	<i>Fusobacterium nucleatum</i>
<i>Streptococcus pyogenes</i>	<i>Peptostreptococcus</i> species
<i>Streptococcus pneumoniae</i>	<i>Veillonella</i> species
<i>Corynebacterium</i> species	<i>Bacteroides</i> species (bile-sensitive)
<i>Escherichia coli</i>	<i>Eubacterium</i> species
<i>Haemophilus influenzae</i>	
<i>Pseudomonas</i> species	

Principles of diagnosis and management

Diagnosis

It should be highlighted that infections of some fascial spaces involve airway obstruction. Therefore confirmation of an adequate airway is the first priority of diagnosis and treatment. It is also necessary to determine if there is indication of other complications, such as sepsis.

Observation and palpation can provide diagnostic information on the involvement of superficial fascial spaces. In contrast, deep fascial spaces have a complex anatomy and are covered with a substantial amount of unaffected superficial soft tissues. As a consequence such infections may be difficult to palpate and to visualize externally, and therefore there is a greater risk of overlooking the involved compartments. Although some clinical findings may offer valuable clues in determining the site and extent of infection, diagnosis should be made with a CT scan and MRI study.

Another important consideration is the host defense level of the patient, because poor host defense is strongly associated with rapid progression and development of serious complications and affects response to the treatment.

Management

Although the principles of management are similar to those for odontogenic infections, a more aggressive attitude is required (Table 29.24). As stated earlier, the first priority is the management of an adequate airway. In emergency situations, tracheostomy or cricothyroidotomy should be performed before any therapeutic measures are attempted. It should be noted that oral or nasal endotracheal intubation can be difficult because of swollen pharyngeal walls and laryngeal displacement, and a failure of instrumentation may result in additional swelling.

Surgical drainage has a critical role in the management of these infections. Early incision may be of great value even when pus has not collected in the abscess, because it may provide a reduction of pres-

Table 29.24 The principles of management for fascial space infections.

The first priority is airway management
Adequate diagnosis using CT scan or MRI
Careful and constant evaluation of patient’s condition
Early incision and drainage even in the case of no apparent evidence of abscess formation
Aggressive antibiotic therapy via intravenous route
Adequate supportive care with hospitalization
Assess and correct any immunocompromising factors and others that may delay recovery

sure in the tissues and allow exploration of undetected abscesses. Therefore the surgeon should not wait for apparent evidence of pus accumulation. However, aggressive surgical intervention may cause injury to vascular and neural structures with resulting sequelae and scarring. Therefore actual treatment will vary depending on circumstances, with consideration of the status of the individual patient and the relative merits of aggressive surgery. Some less severe infections with an absence of any immunocompromising factors may be managed with simple intravenous antibiotics and supportive care (plus extraction of the inciting tooth), therefore avoiding aggressive surgical treatment. In such cases, careful and constant monitoring of the patient is necessary. It should again be highlighted that these infections may progress very rapidly and the clinician should not hesitate to perform surgery if the situation deteriorates.

Surgical drainage

Surgical drainage is the most effective and reliable means of treatment (Table 29.25). A generous incision and aggressive but careful exploration of the involved spaces are required. A secure airway must be established before initiating any surgical procedure, as edema and hematoma associated with surgery may cause additional compromising of the airway. If necessary, a surgical airway should be established. Use of general anesthesia is recommended in cases of extensively spreading or deep infection. A properly and carefully placed incision and blunt dissection with hemostat or finger to avoid surgical injury of important structures are essential. All involved compartments must be opened, and multiple drains should be placed to provide adequate drainage and decompression of the infected area. If an adequate outcome is not obtained, undetected or unexposed abscesses might be involved. Repeat of surgical intervention should then be undertaken without delay.

Antibiotic therapy

Since microbiologic data are necessary to determine each case-specific choice of antibiotic, clinical samples should be obtained whenever possible. Nevertheless, the choice of antibiotic is usually empiric during the first visit of the patient. A penicillin class antibiotic is

the recommended first-choice drug for treatment of fascial space infections (Table 29.26). It should be noted that the diffusion of antibiotic into closed fascial spaces might be limited because of poor vascularity. In addition, the host defense level can often deteriorate even in patients with normal immunity because of fatigue, dehydration, and malnutrition. Therefore a standard dose of antibiotic may prove inadequate. Administration of a high dose (usually maximum) of antibiotic or the use of two or more types of antibiotics in combination that provide a synergistic effect should be considered. Administration of antibiotic through the intravenous route is strongly recommended because it ensures adequate plasma level of the drug. In cases of penicillin allergy, clindamycin and metronidazole may be the best alternatives, although erythromycin is an adequate candidate for mild to moderate infections. If involvement of penicillin-resistant organisms is suspected, metronidazole, clindamycin, co-amoxiclav, and carbapenem may also be recommended. Intravenous antibiotic therapy should continue until resolution of infection is confirmed, and then an additional course of oral antibiotics should be given for an adequate period to prevent recurrence.

Supportive care

The principles of medical supportive care are described in the earlier section, Principles of management of bacterial infections. Constant and careful

Table 29.25 Principles of surgical drainage.

Establish a secure airway before procedure
A generous incision and aggressive exploration of the involved spaces
Properly placed incision and blunt dissection without damage to important structures
Open all involved compartments without overlooking any potential tissue space
Place multiple drains
Should not hesitate to repeat surgical intervention if adequate outcome is not evident

Table 29.26 Instances of suggested antibiotic regimens for fascial space infections.

Antibiotic regimens for less severe cases*	
Penicillin (e.g. penicillin V, amoxicillin)	Maximum dose, orally
Erythromycin	Maximum dose, orally
Clindamycin	Maximum dose, orally
Metronidazole	Maximum dose, orally
Co-amoxiclav	Maximum dose, orally
Penicillin + metronidazole	Maximum dose, orally
Antibiotic regimens for moderate to severe cases	
Penicillin (e.g. penicillin G, ampicillin)	Maximum dose, intravenously
Co-amoxiclav	Maximum dose, intravenously
Ampicillin-sulbactam	Maximum dose, intravenously
Clindamycin	Maximum dose, intravenously
Imipenem	Maximum dose, intravenously
Penicillin + metronidazole	Maximum dose, intravenously
Penicillin + clindamycin	Maximum dose, intravenously
Co-amoxiclav or ampicillin-sulbactam + Clindamycin or metronidazole	Maximum dose, intravenously

* For example, the infection of buccal or canine space in patients who are not immunocompromised.

monitoring of the patient with intervention at the earliest sign of complication, especially a compromised airway, is necessary. Severe cases may require management by appropriate specialists in an intensive care unit.

Patients may have an underlying compromise of their normal immune defenses. Such factors that may delay recovery should be assessed and corrected. Patients usually suffer severe pain, dysphagia (difficulty swallowing), and trismus, and often have a poor appetite due to depression, malaise, and pain. As a result, patients may be exhausted with a degree of dehydration and malnutrition. It should be remembered that infection itself produces a compromised host.

Unless infection is mild with little risk of progression, patients should be hospitalized for intravenous antibiotic therapy, constant monitoring of their condition, adequate pain control, and management of fluid balance and nutrition.

Specific fascial space infections

Anatomy, clinical features, and surgical intervention of facial space infections that may be particularly important for dentists are discussed here. Bacterial infection in the mouth primarily spreads to involve contiguous fascial spaces. These spaces include sublingual, submandibular, submental, buccal, and canine spaces. Although most infections do not extend further, infection occasionally progresses to the posterior fascial spaces, passing through the primary spaces. The masticatory space and deep cervical fascial spaces are of importance for dentists because infection of these spaces can develop into serious conditions.

Sublingual space infection

The sublingual space is defined superiorly by the mucosa in the floor of the mouth and inferiorly by the

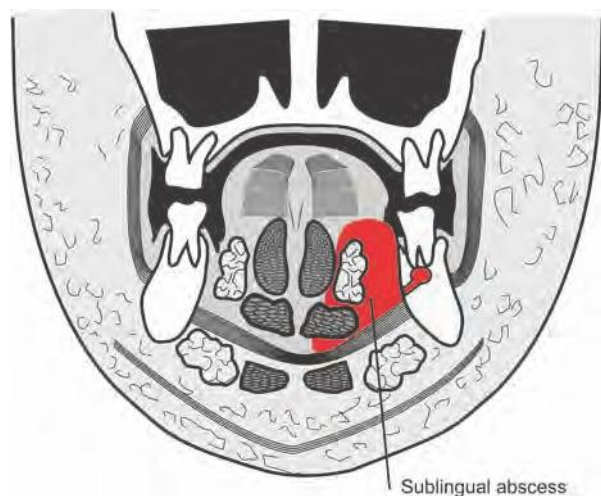


Fig. 29.30 Sublingual space infection occurs when odontogenic infection of the mandible perforates the lingual aspect of cortical bone above the attachment of mylohyoid muscle.

mylohyoid muscles (Fig. 29.30). Anterior and lateral borders are the lingual surface of mandibular bone. The sublingual space contains the sublingual gland, submandibular duct, lingual nerve, and sublingual artery and vein.

Odontogenic infection is the most common cause of a sublingual abscess, although sialolith and sublingual gland infection can also be included in the etiology. The mylohyoid muscle separates the sublingual space above from the submandibular space below (Fig. 29.31). This muscle attaches to the lingual surface of the mandible in an obliquely downward line from posterior to anterior. Since the anterior teeth and first molars commonly attach superior to the mylohyoid insertion, sublingual abscesses usually result from an infection arising from the roots of these teeth. As the sublingual space is divided into right and left by only loose connective tissue, sublingual space infections often expand bilaterally.

Infection of the sublingual space produces erythema and swelling of the floor of the mouth, accompanied by pain and tenderness, but creates little or no extraoral swelling. An elevated tongue is an important hallmark of this type of infection and it may result in a difficulty to freely move the tongue. As the sublingual space communicates with the submandibular space in the posterior aspects, infection may readily spread to the latter.

Surgical drainage can be performed through the intraoral approach with an incision of the mucosa, parallel to Wharton's duct (Fig. 29.32). If infection extends to the submandibular space, surgical drainage via a percutaneous approach may be required.

Submandibular space infection

The submandibular space lies between the mylohyoid muscle, superficial fascia, platysma muscle, and the submandibular skin (Fig. 29.33). The contents of this space include the submandibular salivary gland, its duct portions, and the submandibular

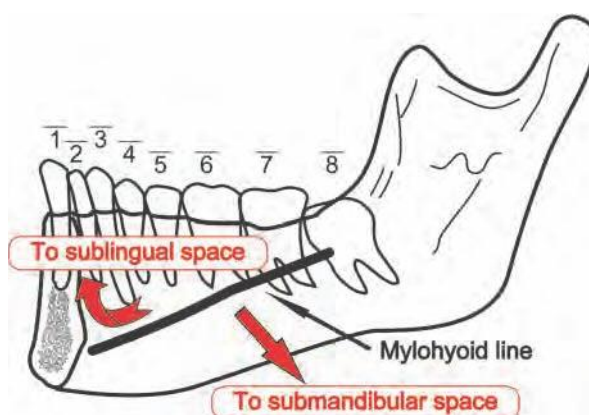


Fig. 29.31 The mylohyoid muscle determines if infection erodes to the sublingual or submandibular space. If the infection perforates the lingual cortical surface of mandibular bone above the attachment, the sublingual space will be involved. Conversely, if infection erodes inferior to the mylohyoid line, it will spread to the submandibular space directly.

lymph nodes. The posterior border of the submandibular space communicates with the masticatory spaces and lateral pharyngeal spaces. As discussed earlier, the attachment of the mylohyoid muscle on the lingual surface of mandibular bone determines if odontogenic infection spreads into the submandibular or sublingual space. As the second and third molar roots are commonly below the mylohyoid ridge, infection presenting on the lingual surface tends to enter the submandibular space.

A submandibular space infection produces pain, erythema, and a brawny or soft swelling of the submandibular triangle (the region of the neck immediately beneath the mandibular body) (Fig. 29.34).

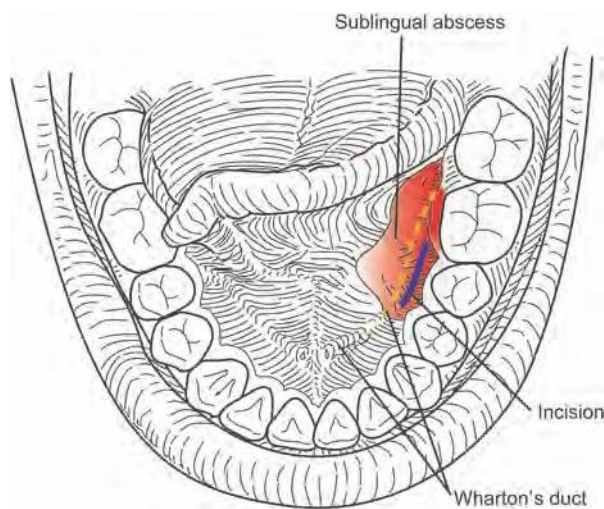


Fig. 29.32 For surgical drainage for sublingual abscess, an intraoral incision is employed. As shown in the figure, the incision should be placed in the mucosa of the floor of mouth, parallel to Wharton's duct. The edge of the knife should not be inserted deeply to avoid injury of lingual artery and nerve and duct of submandibular gland. After incision of mucosa, the abscess cavity is encountered by blunt dissection of underlying connective tissues.

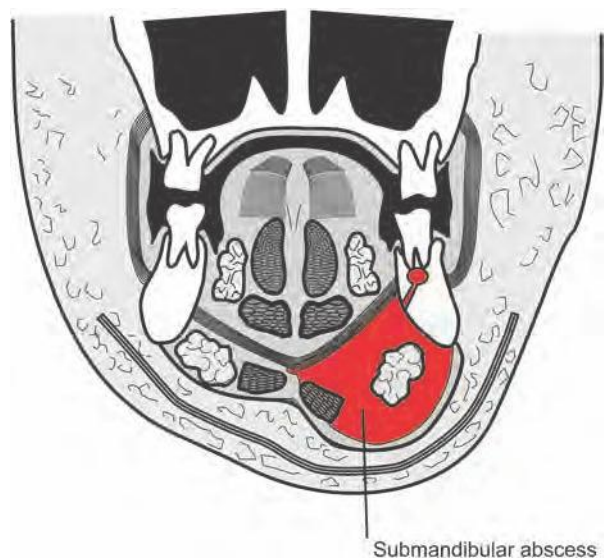


Fig. 29.33 Submandibular space infection usually originates from infection of mandibular molar tooth that erodes the lingual cortical surface below the attachment of mylohyoid muscle.

Infection often affects muscles of mastication, resulting in trismus. Infection can spread to involve the masticator and lateral pharyngeal spaces.

Surgical drainage via a transoral approach is usually difficult. Drainage is generally achieved by incision of the skin below and parallel to the mandible, with subsequent blunt dissection to the depths of the space, with attention to avoid any surgical damage to significant structures, including the submandibular gland, the facial artery, and the lingual nerve (Fig. 29.35).

Submental space infection

The submental space exists below the chin. It lies between the submental skin, the anterior bellies of



Fig. 29.34 Submandibular space infection involved in pericoronitis of mandibular third molar. Edematous swelling with erythema is evident in the submandibular region. Due to swelling, the boundary between the mandibular angle and neck becomes indistinct. Extension of swelling to the buccal space is also evident.



Fig. 29.35 Adequate place of incision is at least two fingers' width below the lower border of mandible to minimize the risk of damage to the marginal mandibular branch of the facial nerve. The incision line should be parallel to the lower border of mandible. The platysma muscle will appear following incision of skin and subsequent blunt dissection of subcutaneous tissue. The platysma muscle and underlying fascia are dissected using a finger or hemostat. The abscess cavity may be encountered around the submandibular gland that normally exists under the fascia.

the digastrics muscle, and the mylohyoid muscles (Fig. 29.36). The submental space contains areolar connective tissue and submental lymph nodes. A submental abscess commonly involves an infected mandibular incisor, although infection of cutaneous wounds and anterior mandibular fractures can also be the cause. A submental space infection produces a gross swelling with erythema and warmth in the chin so that the chin may appear to protrude anteriorly. Infection often affects the submandibular spaces. In contrast, as the tight attachment of superficial cervical fasciae to the hyoid bone prevents extension of infection inferiorly to the lower neck, submental infection seldom extends directly to the lower cervical regions.

Percutaneous surgical drainage is an effective approach to management, with an incision placed below the chin, parallel to the lower border of mandible (Fig. 29.37).

Ludwig's angina

The floor of the mouth contains the sublingual space, submandibular space, and submental space, and there is ready communication across the midline through to the opposite site. Due to this anatomy, infection may readily spread from the sites initially involved to most or all of the spaces in the floor of the mouth. This condition is called Ludwig's angina, named after the German physician, Wilhelm Frederick von Ludwig, who described the condition fully in 1836.⁹⁶ Since most cases of Ludwig's angina are of dental origin, bacterial cultures may mirror the oral flora. These infections usually involve viridans streptococci and anaerobes.⁹⁷

Ludwig's angina is characterized by a firm swelling of the floor of the mouth with elevation of the tongue, a relatively spreading cellulitis with no ten-

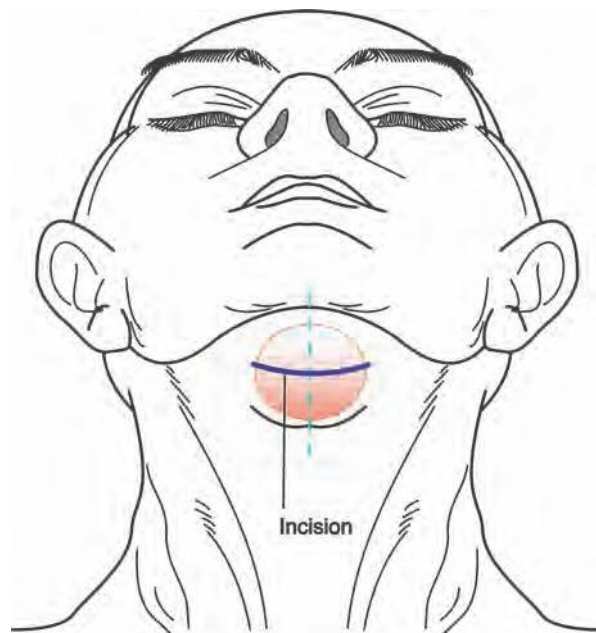


Fig. 29.37 Surgical drainage for submental abscess is usually performed through a cutaneous incision. A horizontal incision should be placed 1–3 cm below the lower border of the mandible rather than the top of the swelling (abscess) to exploit gravity to encourage drainage.

dency to form abscesses and involvement of bilateral submandibular and sublingual spaces (Fig. 29.38). The majority of infections begin in the submandibular space, often resulting from an infection of a mandibular molar tooth, and then rapidly spread to involve the sublingual space, usually on a bilateral basis. Although the initial symptoms are unilateral, they will progress to involve both sides. The submandibular and submental regions become tense, swollen, and tender. There is increasing neck rigidity, trismus, odynophagia (painful swallowing), and drooling. The floor of the mouth will become tense and indu-

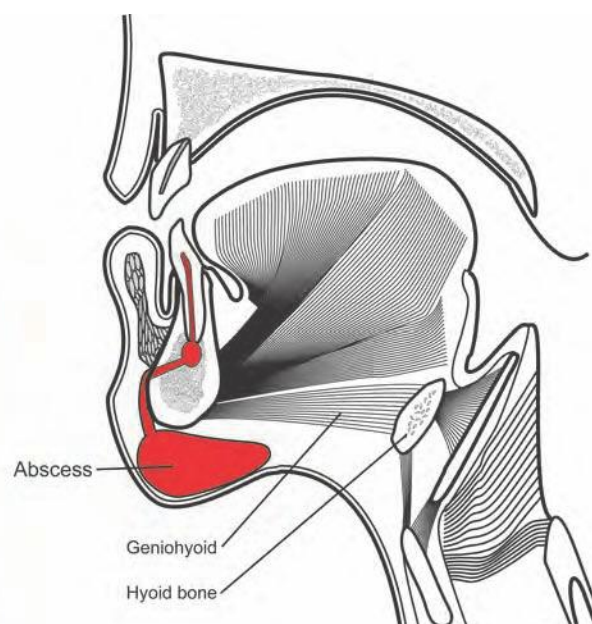
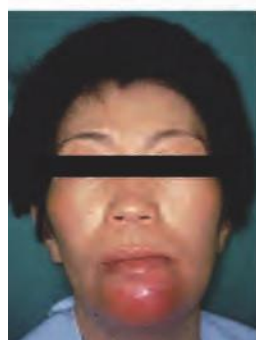


Fig. 29.36 Submental space infection caused by periapical abscess in the mandibular incisor.

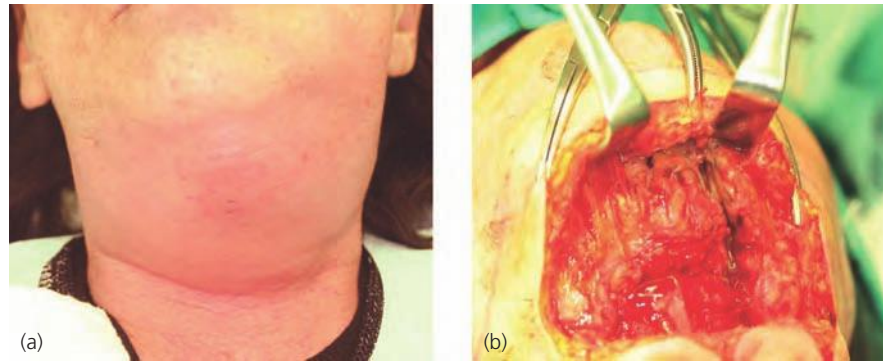


Fig. 29.38 (a) Ludwig's angina involved in odontogenic infection. A swelling with redness started in the right submandible and then extended to involve the floor of mouth, chin and opposite site. (b) Vigorous incision is required. Photographs were obtained from Dr T. Aoki (Tokai University).

rated with extensive mucosal swelling. Intense pain is usually present, but fluctuance is unusual. The tongue is pushed superiorly and its movements become stiff. The patient can develop a toxic condition, with high fever, tachycardia, and malaise often observed. As the swelling progresses, there is increasing encroachment upon the airway, resulting from elevation of the tongue and extension to the lateral pharyngeal space. It should be noted that progression from the onset of symptoms to respiratory obstruction often occurs within 12–24 hours.

The infection may spread to involve posterior fascial spaces, in particular the lateral pharyngeal space. Ludwig's angina has historically been associated with a high mortality rate, primarily due to inadequate diagnosis and management of airway obstruction. Although the prognosis has improved, Ludwig's angina remains a serious and life-threatening disease.

With regard to treatment, special attention must be given to maintenance of the airway. Early and vigorous incision and aggressive opening of all involved spaces are recommended to provide decompression of the area involved. Drainage through generous incision on the skin in the submandibular and submental regions is required. Although the length of the cutaneous incision may be variable, incision generally crosses to the submandibular region bilaterally. In addition to extraoral drainage, transoral drainage should be undertaken if instruments can be inserted into the oral cavity.

Buccal space infections

The buccal space occupies the portion of the subcutaneous space between the facial skin and the buccinator muscle. It contains the buccal fat pad, parotid duct and the facial artery. Common sources of buccal abscess are through involvement of the maxillary or mandibular premolar and molar teeth (Fig. 29.39).

Involvement of the buccal space usually results in a remarkable cheek swelling in the region between the zygomatic arch and the inferior border of mandible (Fig. 29.40). Abscesses may be palpable (fluctuance) intraorally and/or subcutaneously. Buccal

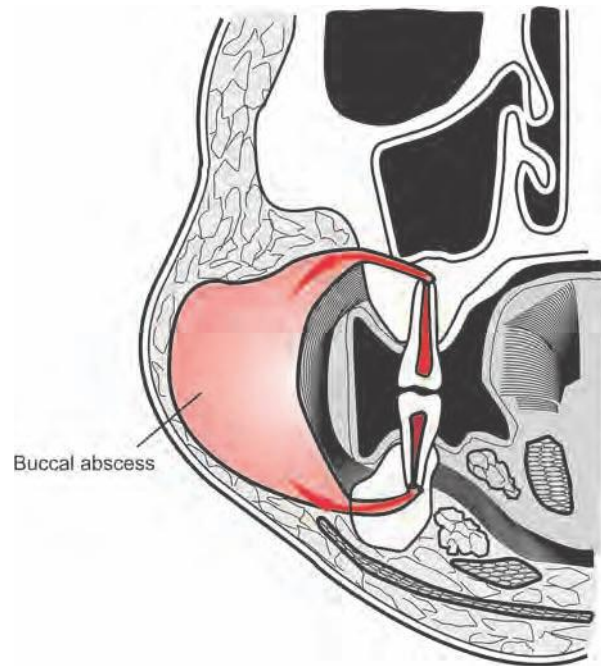


Fig. 29.39 Buccal abscess results from entry of purulent materials from odontogenic infections eroding the cortical plate superiorly to the maxillary attachment of the buccinator muscle or inferiorly to the mandibular origin of the muscle. (Illustration was redrawn with permission from Cummings CW, *et al.*, editors, *Otolaryngology: Head and Neck Surgery*, vol 2. St Louis, MO: Mosby, 1986.⁹⁸ Copyright © 1986 Elsevier.)

infections can extend into the periorbital and masticator spaces. Drainage is usually achieved by cutaneous incision.

Canine space infections

The canine space is a potential space that exists between the levator anguli oris and the levator labii superioris muscles. If the infection perforates the lateral cortex of the maxillary bone superior to the attachment of either of these muscles of the upper lip, the canine space is potentially involved. It is only the canine tooth that has a sufficiently long root through which infection may enter the canine space. Therefore canine space infections primarily result from infections of the maxillary canine tooth.



Fig. 29.40 Two cases of buccal abscess.

When the canine space is infected, there is a swelling of the anterior face that may obliterate the nasolabial fold. Canine space infection may also cause edematous swelling in the eyelids (Fig. 29.41). In later stages, infections may spread to involve the orbit.

Drainage through an intraoral approach with incision high in the maxillary labial vestibule is recommended. Although drainage with extraoral incision is also an option, it may result in scarring.

Infections of the masticatory spaces

Masticatory spaces is a clinical term used to describe the entire area of the mandible and muscles of mastication. It contains the masseter muscle, the ramus and posterior part of the mandibular body, the tendinous insertion of the temporalis muscle, the medial and lateral pterygoid muscles, and the inferior alveolar nerve and vessels. The spaces comprise three major subgroups, the temporal space, the pterygo-mandibular space, and the submasseteric space (Fig. 29.42). Since these spaces communicate with

one another, once one portion becomes infected the others may also be affected. Moreover, these spaces communicate with the buccal, submandibular, and lateral pharyngeal spaces so that infection of the masticatory spaces may spread to these spaces and the converse can also occur.

Trismus is commonly seen and might be a relatively long-term sequela. Due to significant trismus and dysphagia, the fluid balance and nutrition level of the patient often deteriorates.

Since severe trismus hinders the insertion of surgical instruments and prevents intraoral surgical approach, drainage usually requires cutaneous incision in the submandibular region and subsequent dissection of underlying soft tissue to the depth of the abscess cavity (Fig. 29.43).

Submasseteric space infection

The submasseteric space lies between the lateral aspect of the mandible and the medial boundary of the masseter muscle. The space is commonly affected by infection of the mandibular molar, especially the



Fig. 29.41 Two cases of canine space infection involved in periapical abscess in the maxillary canine. The swelling of anterior maxilla with erythema and edema of the eyelid is evident.

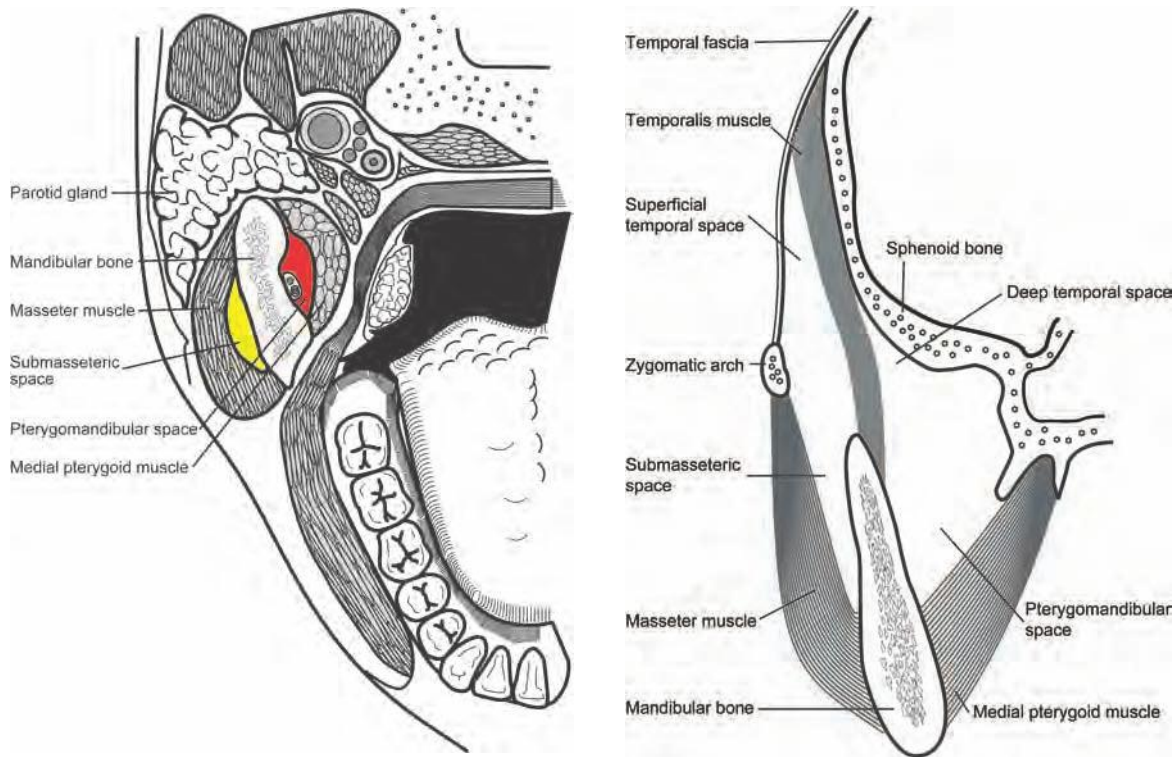


Fig. 29.42 Submasseteric space, temporal space, and pterygomandibular spaces lie around the mandibular ramus. Infection of the mandibular molars may spread to these spaces. The spaces are surrounded by masseter muscle, lateral and medial pterygoid muscles, temporal muscle, and skull. (Right illustration was redrawn with permission from Cummings CW, *et al.*, editors, *Otolaryngology: Head and Neck Surgery*, vol 2. St Louis, MO: Mosby, 1986.⁹⁸ Copyright © 1986 Elsevier.)

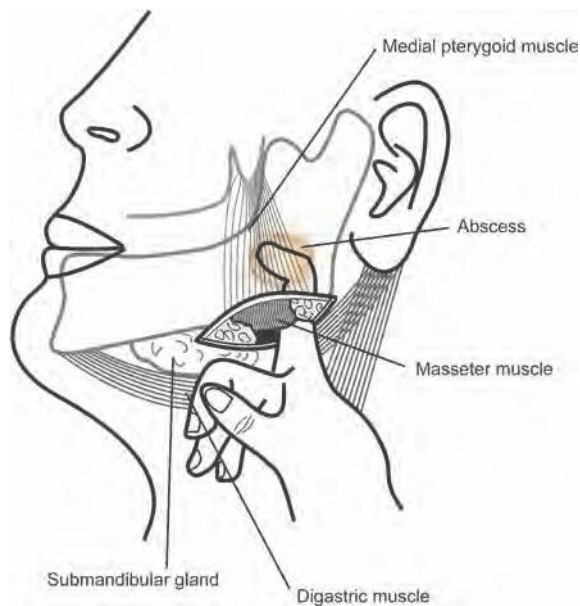


Fig. 29.43 Surgical drainage for submasseteric or pterygomandibular abscess involves cutaneous incision. Similar to the case of submandibular abscess, the incision should be placed at least two fingers' width below the lower border of mandible and subcutaneous tissue is dissected up to the inferior surface of the mandibular bone. When the mandibular bone is encountered, the attachment of masseter muscle or medial pterygoid muscle to the bone will be palpated. In the case of submasseteric abscess, once a part of attachment of masseter muscle to the mandible is exfoliated, the abscess cavity may be encountered. In the case of pterygomandibular abscess, the abscess cavity is explored through partial exfoliation of pterygoid muscle from the bone. Illustration was redrawn with permission from Kitano S, *et al.*, editors, *Korekarano Koku kansensho*, 1st edn. Tokyo: Gakken Shoin, 1993.⁶

third molar, but buccal space infection and fracture infection in the mandibular angle may also be involved. A submasseteric abscess produces pain, swelling in the area overlying the angle of jaw and ramus of the mandible, and trismus.

Pterygomandibular space infection

The pterygomandibular space exists between the mandibular ramus and the medial pterygoid muscle. The etiology of a pterygomandibular abscess is often infection of the sublingual and submandibular spaces, periocoronitis, and postextraction infection of mandibular third molar. It is the pterygomandibular space where a local anesthetic solution is injected for the inferior alveolar nerve block. Contamination following needle injection is therefore also a common cause of this infection. Infection of only the pterygomandibular space induces little or no facial swelling whilst an inflammation of the median pterygoid muscle results in significant trismus. The hallmark of this abscess is trismus without extraoral swelling. The infection usually affects the anterior tonsillar pillar and uvula. Sore throat and dysphagia are usual complaints of the patient. The deviation of the uvula to the opposite site may be observed if trismus is not severe.

Infection of the pterygomandibular space can spread to the lateral pharyngeal space. It is therefore necessary to determine whether the lateral pharyngeal space is also affected.

Temporal space infection

The temporal space is posterior and superior to the submasseteric and pterygomandibular spaces (Fig. 29.44). Temporal space infection may involve swelling in the temporal area that is superior to the zygomatic arch and posterior to the lateral orbital rim.

The temporal space can roughly be divided into two portions by the temporalis muscle: superficial and deep. The superficial temporal space lies between the temporal fascia and the temporalis muscle, and communicates with the submasseteric space. The extension of a buccal space infection can result in superficial temporal space infection. The deep temporal space is surrounded by the temporalis muscle, temporal bone, the skull base, and the lateral pterygoid muscle. Common etiologies of a temporal space infection involve extension of a buccal abscess, infection of maxillary and mandibular third molar, contaminated surgical wound of temporomandibular joint surgery, and arthroscopy.

Cervical fascial space infections

A deep neck infection (abscess) is synonymous with cervical fascial space infection. Despite being quite rare, oral bacterial infections can secondarily induce abscesses in the deep fascial spaces in the neck. Such infections are serious because of the propensity of dissemination hematogenously and contiguously along the fascial planes to involve the mediastinum, pleuropulmonary spaces, and other vital structures. Lateral pharyngeal and retropharyngeal spaces are most commonly involved, and in such circumstances an immediate life-threatening condition occurs through possible airway obstruction (Fig. 29.45).

The lateral pharyngeal space receives purulent material from the fascial spaces around the mandible and the floor of the mouth. Common sources of lateral pharyngeal space infection are from submandibular abscesses and the masticatory spaces. In a typical case, odontogenic infection of the mandible spreads

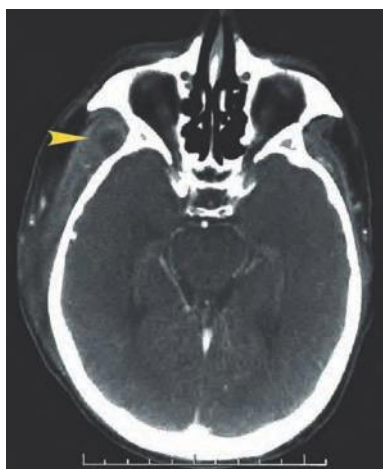


Fig. 29.44 CT scan demonstrating an abscess formation in the temporal space.

to the submandibular spaces and then extends to the lateral pharyngeal space through the pterygomandibular space. Therefore clinicians should determine whether the lateral pharyngeal space is involved during treatment of fascial space infection in the mandibular area or floor of the mouth.

CT scanning is the gold standard in the evaluation of cervical fascial space infections, as it may visualize the location, boundaries, and relation of the infection to the surrounding neurovascular structures. It is also effective in evaluating the degree of airway obstruction. The presence of gas in the affected soft tissue is often seen on CT scans. This sign strongly suggests the involvement of a necrotizing infectious form that requires immediate attention and aggressive therapy.

Special attention needs to be given to possible damage of vital structures during surgical intervention. Also, considerable hemorrhaging from blood capillaries in inflamed tissues can occur. As a consequence of these potential complications skilled specialists should be involved in the treatment of these infections. However, dentists should understand their clinical features and have sufficient knowledge for diagnosis.

Lateral pharyngeal space infection

The lateral pharyngeal (parapharyngeal) space extends from the base of the skull inferiorly to the level of the hyoid bone. The space contains the medial portion of the carotid sheath, internal carotid artery, internal jugular vein, cranial nerves IX, X, XI and XII, and the cervical sympathetic trunk (Fig. 29.46).^{98,99} There is potential communication with the retropharyngeal space.

Lateral pharyngeal infection causes medial displacement of the lateral pharyngeal wall and tonsil and the deviation of the uvula to the opposite side due to swelling. Trismus is caused by inflammation of the pterygoid muscles, and dysphagia occurs as a result of inflammation of the pharynx. The lateral aspect of the neck is also often swollen. Inflammation of the paraspinal muscles may result in torticollis with a resulting limitation of neck movement. Patients usually have a high temperature and become quite sick.

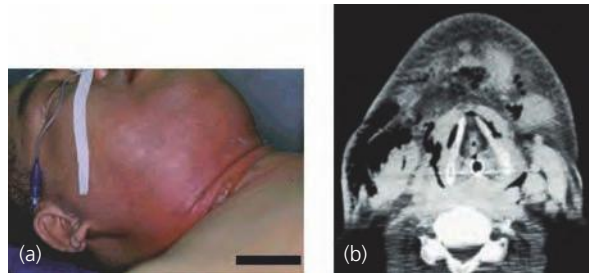


Fig. 29.45 Cervical fascial space infections. (a) Extensive swelling and redness in the neck and chest are evident. (b) CT scan demonstrates compromise of airway and the presence of gas in affected soft tissue, suggesting involvement of anaerobic bacteria. Photographs were obtained from Dr T. Aoki (Tokai University).

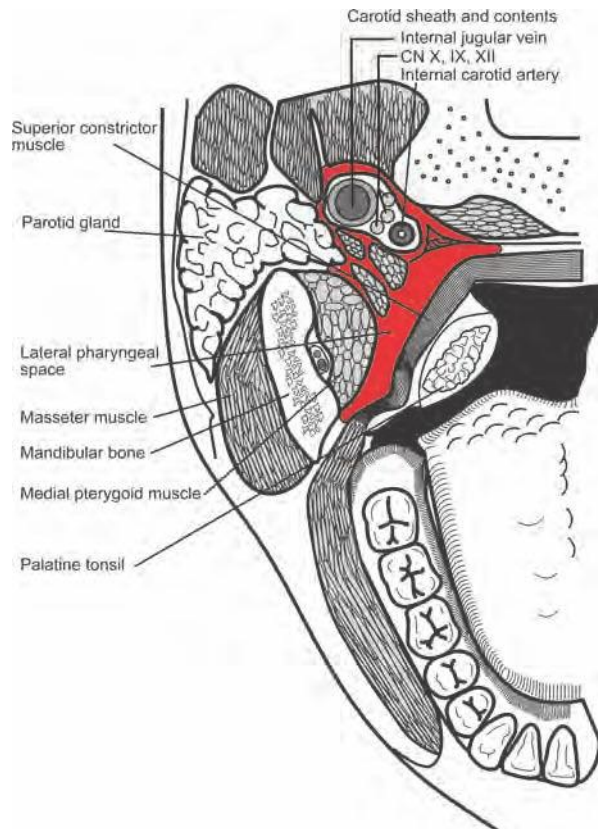


Fig. 29.46 Anatomy of lateral pharyngeal spaces.

It is important to recognize that infection has a potential to cause an airway obstruction. Other possible complications include thrombosis of the internal jugular vein, erosion of the carotid artery or its branches, neural defects, Horner syndrome from involvement of the cervical sympathetic chain, and meningitis resulting from extension of infection to the pterygopalatine fossa and skull base. Mediastinitis could occur as infections spread to the mediastinum through the connective tissue around the carotid sheath or via the retropharyngeal space.

Although drainage can be accomplished by either or both of the transoral and extraoral approaches, an extraoral approach may provide more adequate and reliable entry to the lateral pharyngeal space (Fig. 29.47). Attention is required to avoid injury to neurovascular structures.

Retropharyngeal space infection

The retropharyngeal space lies between the buccopharyngeal fascia (covering the posterior pharynx and esophagus) and the alar fascia (a division of the deep layer of deep cervical fascia), and occupies the space posterior to the pharynx and esophagus (Fig. 29.48). It extends from the skull base to the level of the fourth thoracic vertebra, where the above-mentioned layers fuse.

Infection may enter this space directly, as with traumatic perforations of the posterior pharyngeal wall or esophagus, or indirectly from the lateral pharyngeal space.

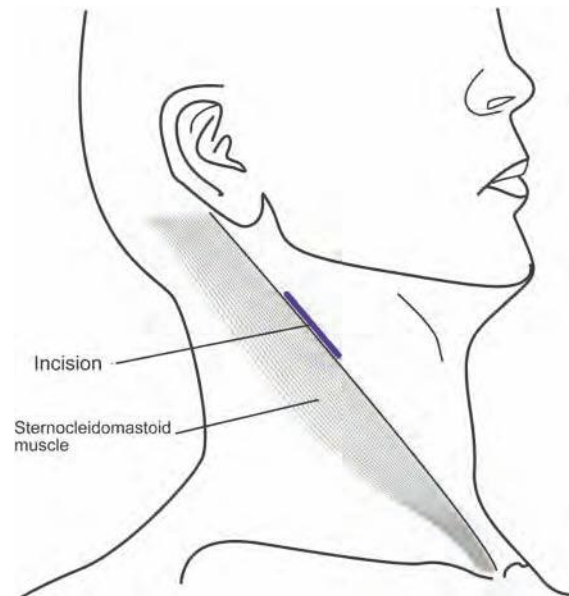


Fig. 29.47 Extraoral approach may provide adequate entry to the lateral pharyngeal space. Incision is placed on the skin over the anterior edge of the sternocleidomastoid muscle.

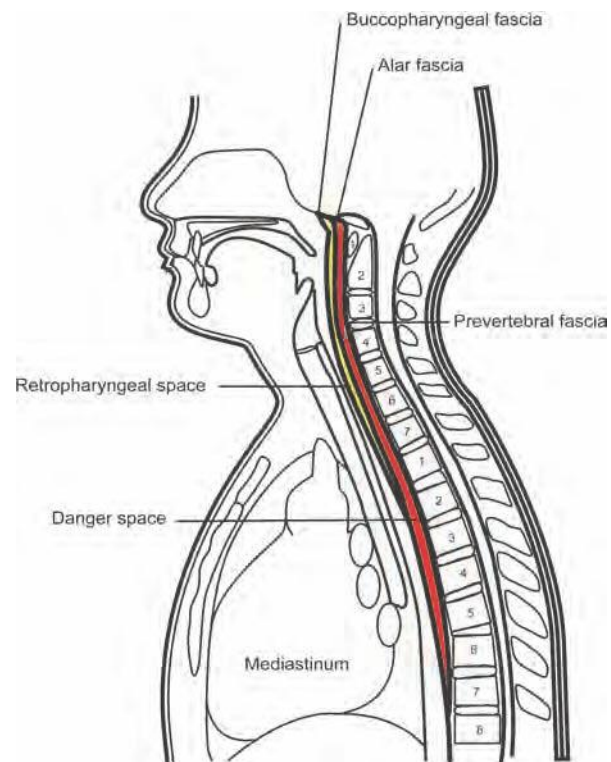


Fig. 29.48 Anatomy of retropharyngeal space and danger space. These spaces are close to the mediastinum and therefore infection of these spaces might spread to involve the mediastinum. (Illustration was redrawn with permission from Cummings CW, *et al.*, editors, *Otolaryngology: Head and Neck Surgery*, vol 2. St Louis, MO: Mosby, 1986.⁹⁸ Copyright © 1986 Elsevier.)

The common clinical findings of this infection include high fever, severe throat pain, dysphagia, and limited neck mobility. In extreme cases there may be breathing difficulty, stridor or tachypnea. Whilst lateral neck radiographs demonstrate a widening of the retropharyngeal soft tissues, a CT scan provides a more reliable diagnostic indicator.

Infection in the retropharyngeal space can spread into the mediastinum directly or via the “danger space”, causing mediastinitis. Moreover, it can cause airway obstruction and rupture of an abscess with subsequent aspiration of pus into the chest, which can lead to acute respiratory failure. Clinicians must remember that there is a significant risk of aspiration during intubation or intraoral incision due to injury to the pharyngeal wall and subsequent rupture of the abscess, so prudent and careful procedure is necessary. Needle aspiration preceding intraoral incision and drainage is effective in reducing the risk of pus entering the chest during the procedure.

Danger space

The “danger space” lies posterior to the retropharyngeal space between the alar and prevertebral fascias, and extends from the skull base down into the posterior mediastinum to the level of the diaphragm. The danger lies in the tendency for infections to spread inferiorly through the space and into the thorax because the loose areolar contents offer little resistance.⁹⁹ Danger space infection results in serious complications including mediastinitis, pleural empyema (pyothorax), and sepsis.

Gas gangrene and necrotizing fasciitis

Severe cervical space infections usually take a necrotizing infectious form. Cervical gas gangrene and necrotizing fasciitis may be used to describe these or overlapping conditions.^{100,101} Whereas a severe cervical space infection only relates to the location of the infection, gas gangrene describes the particular involvement of gas-producing bacteria. Necrotizing fasciitis is used to describe an inflammatory process dominated by tissue necrosis which is spreading rapidly along the fascia.

Gas gangrene in other body sites usually involves *Clostridium* species and necrotizing fasciitis in its classical form is caused by Group A β -haemolytic streptococci.⁹ However, in cervical space infections, non-*Clostridium* anaerobic bacteria, including *Prevotella*, *Fusobacterium*, and peptostreptococci, are

primarily involved and recovery of *Clostridium* species is very rare.

As with other less severe maxillofacial infections, common etiologies include traumatic or surgical wound infections (surgical site infections) and odontogenic infections. Many of these primarily involve the submandibular or the masticator space, and subsequently spread to the lateral pharyngeal or retropharyngeal spaces through the fascial plane with extensive destruction of tissues (Fig. 29.49). There is the potential for spreading downwards into the mediastinum, possibly leading to mediastinitis, pericarditis, lung infection, and sepsis. The infection may spread upwards to the skull base and to the meninges. Another scenario is the spreading of the infection via the blood vessels giving rise to suppurative thrombophlebitis, development of sepsis or disseminated intravascular coagulation.

Pre-existing immunosuppressive conditions, especially uncontrolled diabetes and alcohol abuse, are important predisposing factors, although the infections do occasionally affect otherwise healthy individuals.

Diagnosis is made based on clinical features, imaging study, operative findings, and the patient's progress. An imaging picture of gas formation may be an important diagnostic sign (Fig. 29.50). CT and MRI scans are the most useful imaging methods for diagnosis. It is important, however, that the clinician does not wait for apparent evidence from the imaging study since delayed treatment can lead to fatality. Presumptive surgery should be advocated and diagnosis should be made with surgical inspection.

Management consists of surgery, administration of antibiotics, and supportive medical care. In particular, extensive tissue debridement is most important. Resected tissue should be sent for microbiology culture and any recovered bacteria tested for their sensitivity to antibiotics. The wounds should be sufficiently washed and be left open. Repeated surgical intervention should be carried out without hesitation if deemed necessary. After wound healing, a skin graft may be required.

Administration of a high-dose and broad-spectrum antibiotic via the intravenous route is a



Fig. 29.49 Patients with cervical necrotizing fasciitis. Left photograph courtesy of Dr T. Sugiura (Yamanobe Hospital). Right photograph was obtained with permission from Aoki T *et al.* A case of descending necrotizing mediastinitis associated with odontogenic infection. *Japanese Journal of Oral and Maxillofacial Surgery* 2000.¹⁰²



Fig. 29.50 CT scan revealing gas formation in the affected tissue.

necessity. Adequate antibiotics as empirical choices include penicillin, broad-spectrum cephalosporin, imipenem, clindamycin, and metronidazole. To enhance the antimicrobial spectrum and activity, the use of more than one antibiotic is generally recommended. The antibiotic regimen should be modified on the basis of culture and sensitivity results. Hyperbaric oxygen (HBO) therapy is advocated as an adjunctive treatment, although the effect of HBO on patient mortality rate has yet to be proven.

Shock and multi-organ failure are relatively common as is the occurrence of a compromised airway. Resuscitation and general supportive measures are vital and most patients require monitoring in an intensive care unit.

Osteomyelitis of the jaw

Osteomyelitis is defined as an inflammation of bone marrow.³ However, in almost all cases, infection extends to involve the cortical bone and the periosteum of the affected area. Although this disease occurs much less commonly today than in the past, largely due to the widespread use of antibiotics, it remains an intractable disease and can induce serious cosmetic and functional sequelae.

Osteomyelitis of the jaw may be roughly divided into suppurative and non-suppurative, based on clinical features. Although there are several rare forms of the disease, such as syphilitic, tuberculous, and viral osteomyelitis, this section only discusses the relatively common types. Osteoradionecrosis and

bisphosphonate-associated osteonecrosis are also included in the discussion.

Suppurative osteomyelitis of the jaw

Suppurative osteomyelitis is the dominant form of the disease, which is primarily characterized by the production of pus and necrosis of bony tissues. Suppurative osteomyelitis includes two distinct clinical forms, acute and chronic. Acute osteomyelitis presents a clinical picture of an acute infection which usually includes systemic effects. In contrast, chronic osteomyelitis induces minimal systemic upset. Chronic suppurative osteomyelitis may be divided into two subforms, a primary chronic form that manifests with no acute episode and a secondary chronic form, which involves a prolonged inflammatory process.

Etiology

Odontogenic infection is the most common cause of osteomyelitis of the jaw. Additional etiologies include peri-implantitis, infected cyst or tumor, and surgical wound or trauma infection. Hematogenous seeding of bacteria from other body sites is very rare.

Pathogenesis

Inflammation triggered by bacterial invasion into the marrow space induces a compromised microcirculation and increased pressure in the intramedullary site. This may result in vascular collapse, venous stasis and ischemia, and eventually lead to bony necrosis. As ischemic and necrotic bony tissues do not have a sufficient blood supply, host defense mechanisms may not be able to cope effectively with bacterial invasion and proliferation. Multiplication of bacteria and the resultant inflammation induce further necrosis of surrounding bony tissues, resulting in extensive spread of infection.

The blood supply in the bone, the effectiveness of host defense mechanisms, and the virulence of the pathogens are the prime factors involved in the onset, severity, and persistence of osteomyelitis. In particular, restriction of the circulatory system in the bone is a critical factor. In such circumstances, the mandibular cancellous bone is more likely to become ischemic and more sensitive to infection compared with the maxillary bone. This is because the blood supply to the mandibular bone from its regional artery is less oxygenated compared to the blood supplying the maxilla. Furthermore, the dense overlying cortical bone of the mandible prevents penetration of periosteal blood vessels. Hence the body of the mandible is the most common site for disease and osteomyelitis at this site tends to be refractory.

Bone with chronically impaired blood flow and degenerated or fibrous marrow is sensitive to infection. Fibrous bone dysplasia, previous radiation

exposure of the bone, osteoporosis, osteopetrosis, Paget's disease, and the presence of bone tumors are all predisposing factors of osteomyelitis. Osteomyelitis in bone with these factors is usually intractable. As with other forms of infection, osteomyelitis is more likely to become established in immunocompromised patients.

Microbiology

The microbiology of osteomyelitis of the jaw is similar to that of odontogenic infections which is not surprising as the majority of osteomyelitis cases originate from odontogenic infections. Viridans streptococci and strict anaerobes such as *Prevotella*, *Fusobacterium*, and *Peptostreptococcus* species are the predominant isolates.^{103,104} It should be noted that osteomyelitis sometimes involves pathogens that are uncommonly recovered from odontogenic infection, such as *Staphylococcus aureus*.

The stages of disease and clinical features

As the vast majority of osteomyelitis occurs in the mandible, the progression from the acute to the chronic stage of mandibular osteomyelitis is primarily discussed here.

Acute stage

Acute suppurative osteomyelitis is a process that occurs shortly after the predisposing event. The initial phase is characterized by deep and intense pain, high intermittent fever (38–40°C), and an identifiable causative tooth. Other systemic symptoms include chills, malaise, headache, and a decreased appetite. At this juncture, teeth have not yet become loose. Swelling is minimal and fistulae are absent. Infection is localized only at the intramedullary site and adequate treatment at this phase may prevent further progression.

With the spread of infection, systemic toxic symptoms become more severe and sepsis might occur. Blood examination demonstrates a range of leukocytosis with a left shift and an elevated CRP level. Regional lymph nodes become enlarged and tender. Purulent exudate and pus erode the cortical bone and periosteum. As a result, facial or submandibular cellulitis with extensive firm swelling, warmth, and erythema is evident. If the masticatory muscle is affected,

trismus may occur. A range of cancellous bone is involved and multiple teeth adjacent to the infection source begin to loosen. A throbbing pain in the jaw, severe tenderness, and a feeling of extrusion of teeth occur. Teeth anterior to the causative tooth become highly sensitive to mastication and the percussion test. As infection affects the inferior alveolar nerve, areas of mucosa supplied by the trigeminal nerve, such as the lower lip and gingiva, become numb (this is called Vincent's symptom). Subsequently, pus discharge from the gingival sulcus, multiple mucosal fistulas, and cutaneous fistulae, become apparent. Since damaged bony tissues are not yet radiographically detectable, there is little or no radiographical change in the involved area. The disease will either resolve or progress to the more chronic stage, depending on treatment.

Subacute and chronic stage

The acute stage usually continues for 1–2 weeks and, if osteomyelitis is neglected or does not respond to treatment, the disease will develop into a subacute and chronic stage. However, some cases primarily have a chronic form of osteomyelitis, without an acute episode.

In the chronic stage, the body temperature falls into the normal range and most subjective symptoms disappear or become minimal. Teeth in the affected area show a range of mobility and are sensitive to palpation and percussion. Swelling becomes localized and multiple subperiosteal abscesses are usually evident.

The appearance of bony destruction ("moth-eaten" appearance) and a fragment of necrotic bone, (sequestrum) can be confirmed using a plain radiograph (Fig. 29.51). A sequestrum will subsequently separate from living bone. A layer of new bone may form around the necrotic bone, and this is called an involucrum. Portions of osteosclerosis relating to a hyper-inflammatory reaction may be found in the affected area.

In some cases, sequestra are resorbed completely in granulation tissue or are spontaneously expelled through the mucosa or skin, and in these instances are removable through less invasive surgical procedures. In such cases, full resolution of the infection is possible. In contrast, cases can arise where the necrotic bone persists for long periods. In extreme cases,

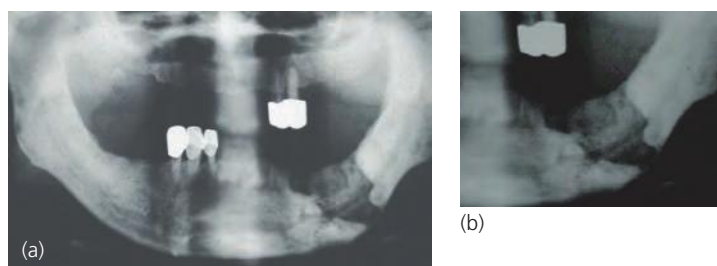


Fig. 29.51 (a) A panoramic radiograph of chronic suppurative osteomyelitis resulting from postextraction infection of the mandibular molar. (b) In a magnified view of the affected area, a sequestrum with a moth-eaten appearance is observed.

pathological fractures occur due to either a significant volume of bone loss from sequestration or because of reduced bone strength (Fig. 29.52).

Maxillary osteomyelitis

For anatomic reasons, maxillary osteomyelitis is uncommon and usually not aggressive. It tends to become localized in a short period, forming subperiosteal abscesses with a limited size of sequestrum. In contrast with adult cases, osteomyelitis in infants commonly occurs in the maxilla and tends to be severe (this is discussed later).

Diagnosis

Imaging

Conventional plain radiographs

Since radiographic bone imaging can generally detect bony lesions that have lost approximately 30–50% of the calcified constituent,¹⁰⁴ radiographic change becomes detectable 1–3 weeks after the onset of acute osteomyelitis. Consequently conventional radio-



Fig. 29.52 A pathological fracture of the mandible involved in severe chronic suppurative osteomyelitis.

graphs provide little information on the status and extent of infection, although they may be of value to assess the likely cause.

Once osteomyelitis has developed sufficiently, characteristic features will be seen. Affected bone has an increased radiolucency, which may be uniform in its pattern or patchy, with a moth-eaten appearance. Sequestra may be seen as the radiopaque areas like “islands of bone”. There may be an area of increased radiodensity surrounding the area of radiolucency as a result of an increase in bone production associated with the inflammatory reaction.

CT and MRI

Bony change may be detected earlier with a CT scan than with conventional radiographs, although CT may not be an effective diagnostic tool for the very early phase of osteomyelitis. Increased attenuation in the medullary cavity, destruction of cortical bone, proliferation of bony tissue, and formation of sequestra may be observed. A CT scan is particularly useful in visualizing the actual extent of the lesion.

MRI has been reported to be sensitive for the early detection of osteomyelitis, although it lacks specificity on the diagnosis. As the bone marrow is replaced by fluid and inflammatory cells, infected areas can be seen as regions of reduced signal intensity on T1-weighted images (Fig. 29.53) and as regions of increased signal intensity on T2-weighted images.

Radionuclide scans

Radionuclide scanning provides a diagnostic modality superior to conventional imaging techniques for acute osteomyelitis. Technetium and gallium bone scans are commonly used. In technetium bone scans, the lesion is detected by using the characteristics of ^{99m}technetium ions, which accumulate at bony tissue where there is significant osteoblastic or osteoclastic activity. Gallium scan images can depict the lesion



Fig. 29.53 A panoramic radiograph and MRI of chronic suppurative osteomyelitis in the mandible of the same patient. (a) The panoramic radiograph shows an osteomyelitis lesion ranging from the mandibular body to part of the ramus. (b) MRI (T1-weighted image) demonstrates a reduced signal intensity in the whole mandibular ramus, suggesting infection that erodes extensively in the mandibular ramus. Infection also affects the surrounding soft tissues.

since ⁶⁷Ga ions tend to accumulate at inflammatory sites.

Diagnostic principles

Diagnosis of acute osteomyelitis is generally based on the history, clinical findings, such as deep intense pain, paresthesia of the inferior alveolar nerve or mental nerve, and fever, and results of blood examination.

In chronic osteomyelitis, bony destruction can be confirmed with plain radiographs. Therefore, diagnosis is usually made based on a combination of medical history, clinical findings, and radiographic appearance. Differentiation from bone tumors and fibrous bone dysplasia is required as these often exhibit similar radiographic appearances.

Treatment

Treatment consists of antibiotics, surgery, and supportive care. Acute suppurative osteomyelitis of jaws is primarily managed by antibiotic therapy. Adequate antibiotic therapy is started during the early stage of the infection and surgical removal of the cause may provide complete resolution of the disease without the subsequent chronic stage. In the case of chronic osteomyelitis, surgery plays a key role in the management.

Antibiotic therapy

Osteomyelitis commonly occurs in bones with an inadequate blood supply or in immunocompromised patients. Therefore, use of high doses of antibiotics is generally recommended. Moreover, as an adequate antibiotic level in the serum must be maintained, the antibiotics should be administered intravenously. In general, the recommended antibiotics belong to the penicillin class because of their high activity against the bacteria commonly involved. Clindamycin and metronidazole can be excellent alternatives for patients who are hypersensitive to penicillin. Suggestions for an empirically selected antibiotic regimen are presented in Table 29.27. It is worth noting that because osteomyelitis can involve a variety of bacteria, clinical specimens should be obtained for microbiologic examination and, whenever possible, the chosen antibiotic regimen should be based on these findings.

In the case of acute osteomyelitis, the course of intravenous therapy should continue until clinical signs have completely disappeared, and an additional course of oral antibiotic should then be taken to ensure complete eradication of the infection, although the duration of the course will vary depending on the case.

In chronic osteomyelitis, intravenous antibiotic administration is recommended after surgery until evidence of wound sealing is seen. This is undertaken even in instances where there are no signs of infection recurrence. Additional courses of oral antibiotics to

Table 29.27 Suggested antibiotic regimens for suppurative osteomyelitis.

Antibiotic for initial therapy	
(1) Penicillin (e.g. penicillin G or ampicillin)	Maximum dose, intravenously
(2) Clindamycin	Maximum dose, intravenously
(3) Co-amoxiclav or ampicillin–sulbactam	Maximum dose, intravenously
(4) Imipenem	Maximum dose, intravenously
(5) Penicillin + metronidazole or clindamycin	Maximum dose, intravenously
After clinical signs have resolved (in the case of acute osteomyelitis) or initial healing of surgical wound is confirmed (in the case of chronic osteomyelitis), switch to one of the following regimens for an additional 4–6 weeks	
(1) Penicillin (penicillin V or amoxicillin)	Standard dose, orally
(2) Cephalexin	Standard dose, orally
(3) Erythromycin	Standard dose, orally

minimize the risk of recurrence following intravenous therapy are often advocated, although the duration of treatment may vary with each case. It should be noted that chronic osteomyelitis is difficult to manage with antibiotic therapy alone. If treatment failure is suspected (e.g. an uncommon persistence of postoperative pain, pus discharge, and inadequate wound healing despite performing aggressive antibiotic therapy in the postoperative period), a change of the antibiotic regimen could be of value, but commonly reoperation is required.

Surgery

The role of surgical treatment in acute suppurative osteomyelitis is generally limited. Loose teeth, sequestra, and involvement of a readily accessible foreign body should be surgically removed as soon as possible. In cases where there is extensive pain, trephination techniques can be employed for drainage of pus.

In contrast, surgery is important in the treatment of chronic osteomyelitis. In these infections, there are large areas of ischemic and necrotic bone that may prevent the effect of treatment and interfere with the natural healing mechanisms. The purposes of surgery are: (1) to improve the blood supply in the involved area thereby allowing adequate penetration of antibiotic; and (2) to maximize the host defense mechanisms and self-healing ability. Sequestra, non-vital bone, involved foreign bodies, inflammatory granulation tissue, sclerotic bone resulting from long inflammatory reaction, and any devitalized tissues with compromised vascularity must be thoroughly removed in surgery. During the operation, it may be difficult to distinguish healthy and devitalized

bony tissues and to determine the surgical margin. Generally, bone should be removed until vital bleeding areas are encountered in all directions. The wound is irrigated with copious amounts of saline solution to reduce contamination. The following surgical procedures are commonly used:

- *Sequestrectomy.* In some cases, once full formation of sequestrum has been confirmed, removal can be performed by simple incision and small surgical trauma.
- *Saucerization and decortication.* Both saucerization and decortication involve surgical removal of infected bone marrow and overlying cortical bone. However, the techniques are distinct:
 - Saucerization involves the surgical excavation of bony tissue to form a shallow depression to facilitate drainage from infected areas with improvement of blood supply to bone and to prevent formation of dead space (Fig. 29.54). After the cortical bone is unroofed to expose the involved medullary cavity, thorough debride-

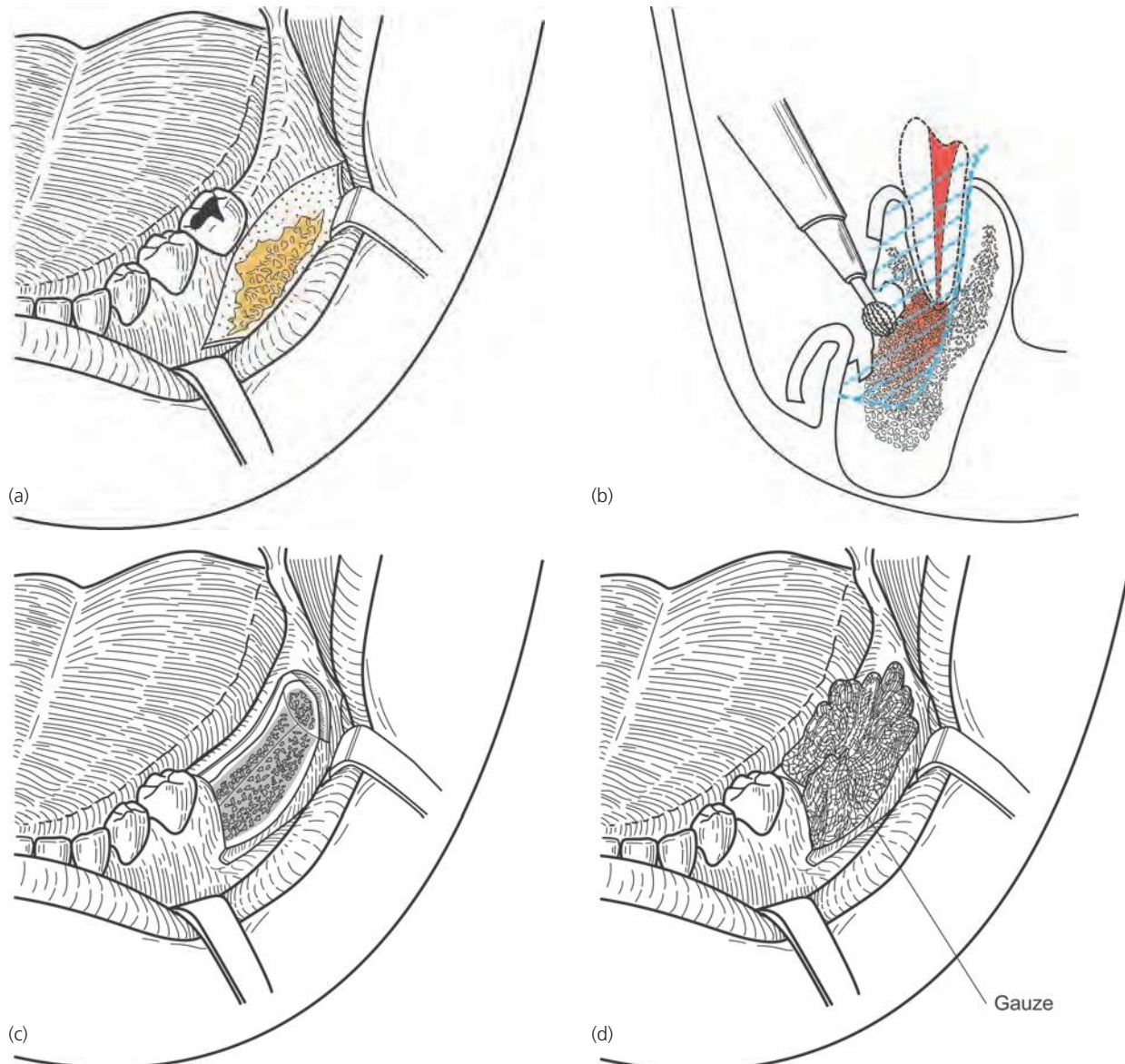


Fig. 29.54 Saucerization generally consists of the following steps. (a) After incision, the buccal mucoperiosteal flap is reflected to expose the infected bone. Extensive tissue reflection is unnecessary to preserve blood supply. Then, loose teeth, pus, sequestrum, necrotic tissue and granulation in the bone marrow and cancellous bone are removed with sharp curettes. The procedure should be performed carefully to avoid damage to the inferior alveolar nerve and vessel. (b, c) Bone in affected areas is reduced using burs or rongeurs until vital bleeding of the area is encountered at all margins. Any sharp bony portion and undercut portion are arranged using burs and bone files to produce a saucer-like depression of the bone. (d) After the surgical area is thoroughly irrigated with copious amounts of saline solution, the buccal flap is trimmed and a medicated gauze pack (for example, gauze covered with iodiform or antibiotic ointment) is inserted for hemostasis and to maintain the flap in a retracted position. The pack is placed firmly and slightly overfilling the defect but should not put pressure on the defect. If necessary, several sutures are tied over the pack to maintain its position. The sutures should be removed 5–7 days after surgery. As healing of the surgical wound proceeds, the bony defect is replaced by granulation tissue and therefore the size of defect will reduce. The gauze pack should be replaced every 7–10 days until signs of initial healing, such as presence of an epithelialized surface on the bed of granulation tissue, are confirmed.

ment is performed and then the margin of bony defect is arranged to eliminate sharp edges and undercut portions. The wound is closed either by primary or secondary intention. However, as primary closure of the wound involves a high risk of recurrence or wound infection, leaving the wound open for healing should be considered. This wound treatment is effective in the prevention of the recurrence of osteomyelitis and also in reducing the risk of wound infection because it decompresses the bone from exudate or pus and may allow subsequent exfoliation of necrotic tissues. Dressings for the wound should be changed frequently after surgery. If therapy is successful, the wound will be closed by regeneration of connective tissue without any wound-healing problems.

- Decortication is a surgical procedure that aims to encourage an increased blood supply to bone marrow in the infected area from the blood vessels of buccal periosteum. This is achieved through surgical removal of the buccal bony cortex and subsequent creation of direct contact of exposed bone marrow with the mucoperiosteal flap. The lateral and inferior cortex plates are removed to form a window after the mucoperiosteal flap is elevated (Fig. 29.55). Pus,

sequestrum, necrotic tissue, and granulation that exist under the cortical bone should be carefully removed and the surgical area is then irrigated. After irrigation and placement of suction drains, primary closure of the wound is performed.

- *Resection and reconstruction of the jaw.* Resection of infected areas and immediate or delayed reconstruction may be considered if an involved area is extremely extensive or less aggressive surgical procedures have failed.

Supportive care

Patients should be hospitalized for any aggressive surgery, and provided with intravenous antibiotic therapy and managed for correct fluid balance and nutrition. As mentioned previously, the patient is likely to have an underlying compromise of their host defenses. The factors that may delay recovery should be assessed and corrected.

Hyperbaric oxygen therapy

HBO therapy may be available in conjunction with surgery. It is a mode of treatment in which a patient breathes 100% oxygen at a pressure greater than normal atmospheric pressure. Because of the increased oxygen tension, large amounts of oxygen dissolve in

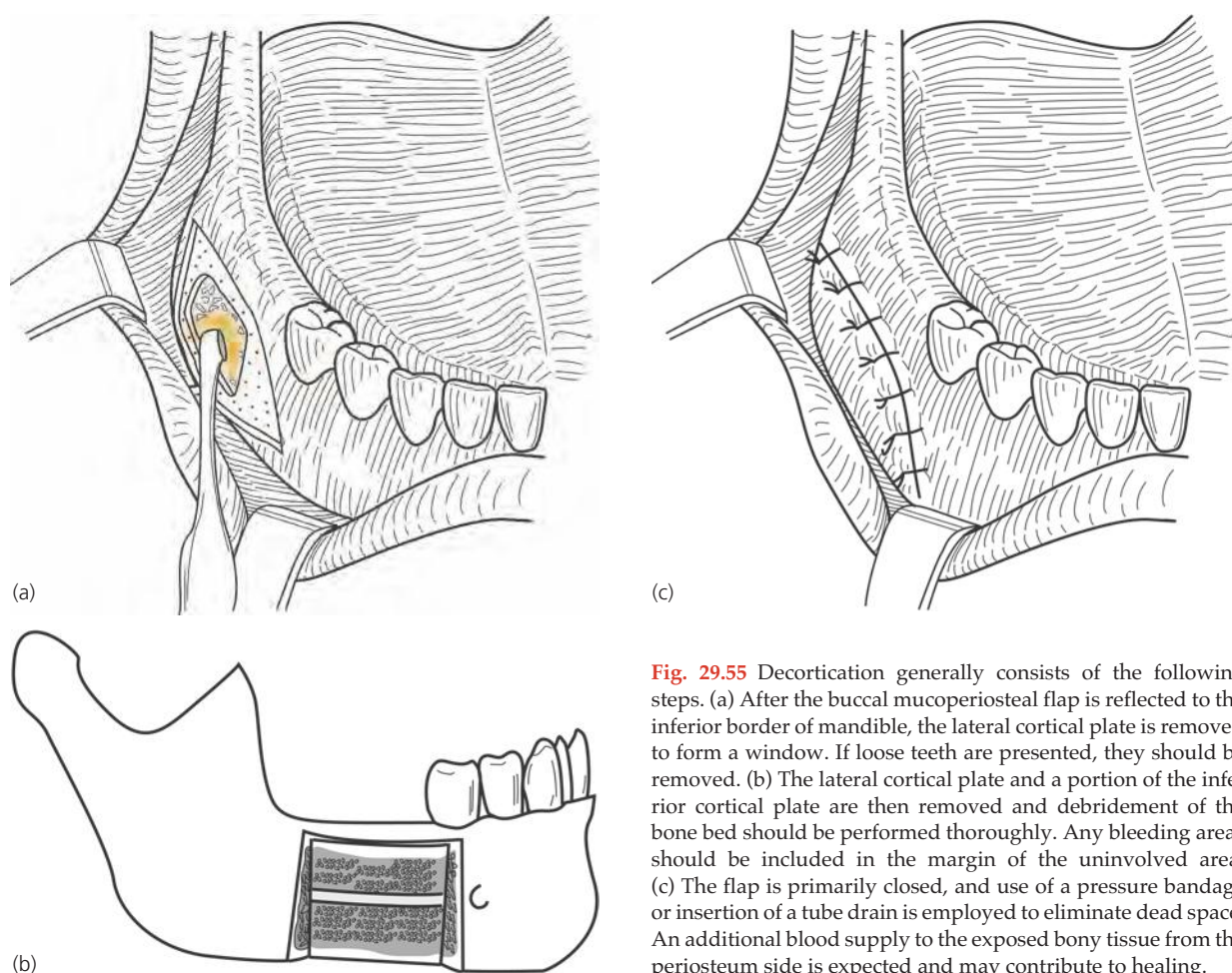


Fig. 29.55 Decortication generally consists of the following steps. (a) After the buccal mucoperiosteal flap is reflected to the inferior border of mandible, the lateral cortical plate is removed to form a window. If loose teeth are presented, they should be removed. (b) The lateral cortical plate and a portion of the inferior cortical plate are then removed and debridement of the bone bed should be performed thoroughly. Any bleeding areas should be included in the margin of the uninvolved area. (c) The flap is primarily closed, and use of a pressure bandage or insertion of a tube drain is employed to eliminate dead space. An additional blood supply to the exposed bony tissue from the periosteum side is expected and may contribute to healing.

the blood, and are then readily delivered to tissues even with poor blood supply. This therapy has neo-vascularization properties and stimulates the immune system. In addition, it may provide a direct kill of certain bacteria.

Infantile osteomyelitis

Infantile osteomyelitis usually occurs a few weeks after birth, and commonly involves the maxilla. Although the exact etiology remains unclear, infantile osteomyelitis is thought to involve perinatal trauma, infection of the maxillary sinus, contaminated nipples or hematogenous seeding of bacteria from other body sites.

This disease could spread to involve the eye and brain. Therefore there are potential risks of serious optic and cerebral sequelae, facial deformities, serious damage to jaw growth, and loss of teeth and bone. Despite a historically high mortality rate, largely due to spread to the brain and sepsis, death is today rare, and morbidity has been greatly diminished with the advent of antibiotics.

The local signs of this infection are swelling of the face with edema of the eyelids and subperiosteal abscesses on the alveolar mucosa and palate. Common systemic signs include high fever, rapid pulse rate, vomiting, delirium, and prostration.

As with acute suppurative osteomyelitis in adults, radiographs are usually of little value in making an early diagnosis. Treatment must be prompt and aggressive to prevent permanent optic damage, neurologic complications, and loss of teeth buds and bony tissues. Management commonly consists of the use of intravenous antibiotics and drainage of the abscess. Although *S. aureus* is the most common pathogen involved, involvement of other bacteria also occurs. Therefore the choice of antibiotic should again be based on microbiologic examination of spec-

imens, although penicillin and clindamycin are recommended for empiric use. Due to the risk of surgical damage to tooth buds, a conservative approach in sequestrectomy should be considered.

Chronic diffuse sclerosing osteomyelitis

Chronic diffuse sclerosing osteomyelitis is a non-suppurative form of osteomyelitis. It is usually found in the mandible and is characterized by recurrent pain and swelling, no suppuration or abscess formation, radiographic appearance of variable amounts of bone deposition and diffuse sclerosis with ill defined borders in the jaw, and insidious onset and persistence of disease.

The etiology and pathogenesis of the disease remain unclear. Possible etiologies include hyperactive immunological responses, chronic tendoperiostitis from muscle overuse, and a manifestation of synovitis, acne, pustulosis, hyperostosis or osteitis (SAPHO) syndrome.^{105–107}

A history of intermittent pain, swelling, trismus, pressure, and paresthesia are important clinical findings for diagnosis. Radiographic investigation demonstrates intermingled sclerotic and osteolytic lesions with a solid periosteal reaction and/or external bone resorption. In cases with a long-standing disease history, osteosclerosis becomes increasingly marked (Fig. 29.56).

There is generally no specific laboratory test for diagnosis. As with suppurative osteomyelitis, panoramic radiograph, radionuclide scanning, CT scans, and MRI techniques are useful for diagnosis and follow-up investigations.

Diffuse sclerosing osteomyelitis is difficult to eradicate and may persist for years. Treatment is usually aimed at addressing the symptoms rather than being curative, and includes NSAIDs, corticosteroid,

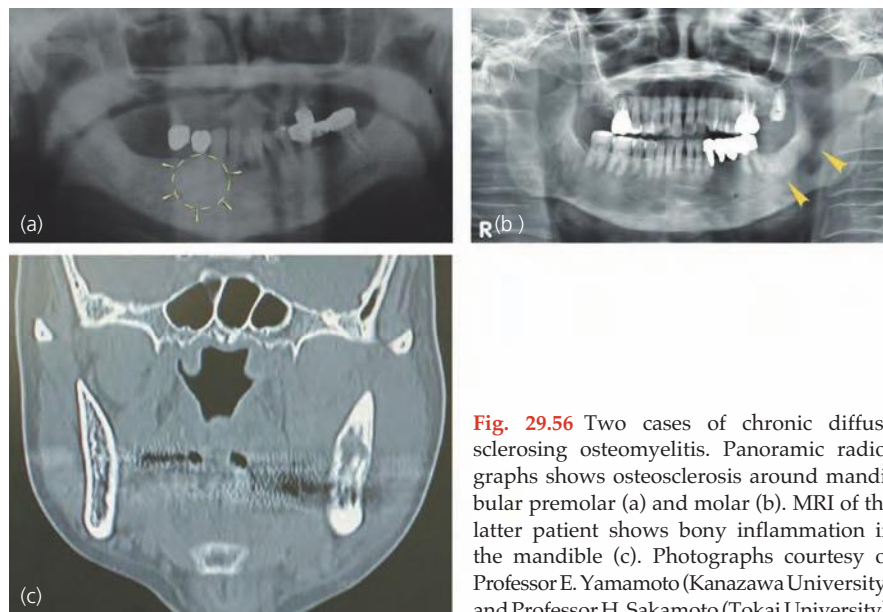


Fig. 29.56 Two cases of chronic diffuse sclerosing osteomyelitis. Panoramic radiographs shows osteosclerosis around mandibular premolar (a) and molar (b). MRI of the latter patient shows bony inflammation in the mandible (c). Photographs courtesy of Professor E. Yamamoto (Kanazawa University) and Professor H. Sakamoto (Tokai University).

HBO therapy, and muscle relaxants. Antimicrobial treatment is usually unsuccessful, and surgery, including debridement and decortication, is often of limited success.

Garré's sclerosing osteomyelitis

Garré's sclerosing osteomyelitis is another form of non-suppurative osteomyelitis. Its name originates from a Swiss surgeon, Dr Carl Garré, who first reported the clinical complications of acute osteomyelitis in 1893.¹⁰⁸ This disease is characterized by an active periosteum proliferation, and formation of subperiosteal bone with no purulent exudate or abscess formation (Fig. 29.57). Garré's sclerosing osteomyelitis is believed to result from an over-inflammatory reaction in the periosteum. It occurs commonly in children and young adults as these ages are the peak periods for periosteal osteoblastic activity. It usually affects the lateral surface of the body of the mandible. Periapical abscesses and post-extractive infections are common etiologies, although there are some cases with an undetectable origin of the disease.

Common clinical features include a localized, unilateral and hard mandibular swelling with little tenderness. The swelling may result in facial asymmetry, although the overlying skin commonly appears normal. Pain can be episodic with dormant periods. There is no apparent systemic sign such as fever, leukocytosis or lymphadenopathy. Radiological findings show a thickened cortical bone and "onion skin" appearance due to periosteal new bone formation.

Differential diagnosis includes Ewing's sarcoma, osteosarcoma, fibrous dysplasia, cherubism, syphilitic osteomyelitis, and healing fracture callus. Treatment involves elimination of the cause of infection, commonly done by tooth extraction or endodontic treatment; antibiotic therapy is usually unnecessary. Surgical remodeling can be considered in cases where there is no spontaneous regression.

Osteoradionecrosis

Osteoradionecrosis is bone necrosis in previously irradiated tissues and in the absence of tumor persistence or recurrence (Fig. 29.58). It is the most significant complication of radiation therapy for malignant



Fig. 29.58 Panoramic radiograph of a 60-year-old man with osteoradionecrosis of the left mandible. The disease became apparent 14 months after radiotherapy (62 Gy) for cancer of the floor of the mouth.

tumors of the head and neck, resulting from both the short- and long-term effects of radiation therapy on bone and the surrounding soft tissue. The incidence of osteoradionecrosis is between 5% and 15%,¹⁰⁹ and most cases occur in the mandible. The condition is irreversible and will not heal spontaneously. In the majority of cases, the disease is induced by secondary trauma, but it does occur spontaneously in 10–35% of cases.^{109,110} The most prominent etiological factor is the reduction in the vascularization potential of tissues.^{109–111} The resulting hypovascular and hypoxic conditions jeopardize cellular activity, collagen formation, and wound healing capacity. Risk factors for the condition include higher radiation doses, tooth extraction after radiotherapy, and poor oral hygiene.¹¹²

A variety of clinical presentations can be seen ranging from small asymptomatic regions of exposed bone that remain stable over time through to full-blown osteonecrosis characterized by severe pain, orofacial fistulas, a foul-smelling necrotic jaw bone with suppuration and pathological fracture.

Diagnosis is primarily based on the patient's medical history and clinical signs and symptoms such as severe pain, non-healing exposed bone within the treatment area after completion of radiotherapy, and incidents of recurrent infections. Plain radiographs may show decreased bone density and occasionally fractures.

Management of osteoradionecrosis remains extremely difficult and time-consuming. Early stages of the disease can be managed with less aggressive treatments such as frequent irrigation with saline,

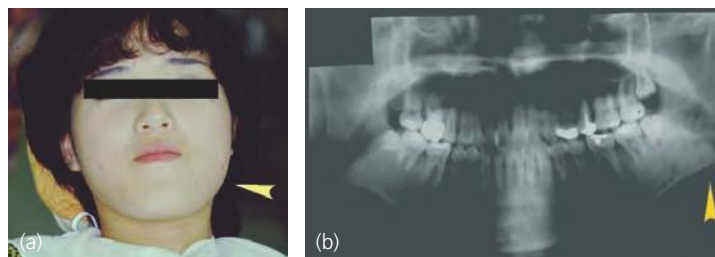


Fig. 29.57 Garré's sclerosing osteomyelitis in the mandibular angle of a young woman. Possible cause is pericoronitis in the mandibular third molar. (a) Facial asymmetry and unilateral and hard mandibular swelling with no inflammatory change in overlying skin are evident. (b) A panoramic radiograph shows thickened cortical bone and a bony lesion around the mandibular angle.

analgesics, antibiotics, local debridement, and sequestrectomy. However, patients who present with extensive lesions or in whom less invasive therapy has failed, often require aggressive resection, necessitating some form of reconstruction. Pathogenic fracture is unlikely to heal and therefore excision of the necrotic ends of both fragments and eventual replacement with a bone graft is required. HBO therapy is often used in conjunction with surgery.

Bisphosphonate-related osteonecrosis of the jaw (BRONJ)

Bisphosphonates are a class of drugs that inhibit osteoclast action and the resorption of bone. These agents are generally used in the prevention and treatment of osteoporosis, osteitis deformans, bone metastasis of malignant tumors with or without hypercalcemia, multiple myeloma and other conditions that feature bone fragility. Intravenous and oral preparations are available and the former is primarily used in the management of bone metastasis and the latter often prescribed to treat osteoporosis and osteopenia.

In 2003, the first case of a BRONJ patient, who exhibited non-healing, exposed necrotic bone in the jaw during bisphosphonates therapy, was published.¹¹³ Since then, similar cases have been reported from many countries. It has now been recognized that use of bisphosphonates can cause osteonecrosis of jaw (BRONJ) (Fig. 29.59). It is hypothesized that the pathogenesis of BRONJ might be primarily related to the drug-mediated inhibition of osteoclast function and bone remodeling.^{114,115}

Definition and epidemiology of BRONJ

Despite no diagnostic criteria having been globally accepted, the American Association of Oral and Maxillofacial Surgeons (AAOMS) proposed that involvement of BRONJ can be considered if all of the following three characteristics are present: (1) current

or previous treatment with a bisphosphonate; (2) exposed, necrotic bone in the maxillofacial region that has persisted for more than 8 weeks; and (3) no history of radiation therapy to the jaws.¹¹⁶

BRONJ is usually found in the mandible and the large majority of cases are related to high-dose intravenous administration. The actual incidence remains unclear because of a lack of published studies of sufficient sample size. Estimates of the cumulative incidence of BRONJ in patients treated with intravenous bisphosphonates range from 0.8–12%.¹¹⁶ The incidence of BRONJ associated with oral bisphosphonates is reported to be from 0.01–0.04%.¹¹⁷ With an increased recognition of the condition, an enhanced duration of drug exposure, and longer follow-up period, it is likely that the incidence will rise. The suggested predisposing factors for the development of BRONJ are extensive. These include intravenous bisphosphonate administration, an increased duration of drug exposure, involvement of the mandible, the presence of thin mucosa overlying bony prominences as seen with tori and bony exostoses, the occurrence of dentoalveolar surgery including tooth extraction, implant surgery, periodontal surgery, a history of inflammatory dental disease, old age, patients of Caucasian race, involvement of a tumor (multiple myeloma > breast cancer > other cancers), corticosteroid therapy, diabetes, smoking, alcohol use, poor oral hygiene, and use of chemotherapeutic drugs.^{116,118}

Prevention of BRONJ

Despite the fact that osteonecrosis can occur spontaneously in patients receiving bisphosphonates, the majority of affected patients experience this complication after dentoalveolar surgery. Therefore, patient education in maintaining good oral hygiene and dental care is of importance in preventing dental disease that may require dentoalveolar surgery.

Prior to treatment with an intravenous bisphosphonate, patients should have a thorough oral examination, any unsalvageable teeth should be removed,

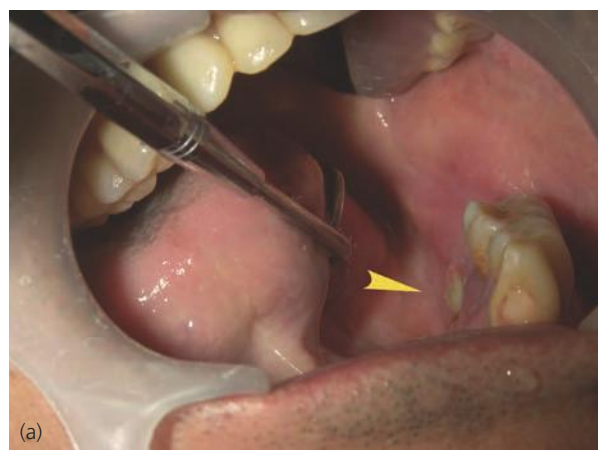


Fig. 29.59 Intraoral view of two patients with bisphosphonate-associated osteonecrosis. (a) An exposed and partly necrotic bone of mandible in the lingual aspect is evident (case 1). (b) Extensive exposure of necrotic bone (case 2).

all invasive dental procedures should be completed, and optimal periodontal health should be achieved. If systemic conditions permit, initiation of bisphosphonate therapy should be delayed until dental health is optimized. Once intravenous bisphosphonate therapy starts, procedures that involve direct osseous injury should be avoided.

In the case of oral bisphosphonates, elective dento-alveolar surgery is generally not contraindicated. The risk of developing BRONJ appears to increase when the duration of therapy exceeds 3 years, although this timeframe may be shortened in the presence of certain comorbidities, such as chronic corticosteroid use.¹¹⁶ For individuals who have taken an oral bisphosphonate for less than 3 years and have no clinical risk factors, no alteration or delay in the planned surgery is necessary. These patients should, however, be adequately informed of the risks of compromised bone healing. For those patients who have taken an oral bisphosphonate for less than 3 years and have also taken corticosteroids concomitantly, the prescriber should be contacted to consider discontinuation of the oral bisphosphonate (drug holiday) for at least 3 months prior to and 3 months after oral surgery, if systemic conditions permit, because it may lower the risk of BRONJ. A break from drug therapy should also be considered in patients who have taken an oral bisphosphonate for more than 3 years with or without any concomitant prednisone or other steroid medication.

The management principles of BRONJ

An effective treatment for BRONJ has not yet been established. In this section, management of BRONJ is discussed based on the guidance proposed by the AAOMS in 2007.¹¹⁶

Patients with established BRONJ generally exhibit a poor response to the established surgical treatment algorithms for osteomyelitis. Therefore, conservative debridement of necrotic bone, pain control, infection management, use of antimicrobial oral rinses, and withdrawal of bisphosphonates are preferable to aggressive surgical measures. Surgical debridement has been variably effective in eradicating necrotic bone, but overinvasive procedures might aggravate bony necrosis or expand the area of lesion. Clinicians should be aware that it is difficult to obtain a surgical margin with viable bleeding bone as the entire jawbone has been exposed to the pharmacologic influence of the bisphosphonate. Areas of necrotic bone that are a constant source of soft tissue irritation should be removed or recontoured without exposure of additional bone. Loose segments of bony sequestrum should be removed without exposing uninvolved bone. The extraction of symptomatic teeth within exposed, necrotic bone should be considered because it is unlikely that the extraction will exacerbate the established necrotic process. Symptomatic patients with pathologic mandibular fractures may

require segmental resection and reconstruction with a reconstruction plate. The potential for failure of the reconstruction plate because of the generalized effects of the bisphosphonate exposure needs to be recognized by the clinician and patient.

Discontinuation of intravenous bisphosphonate therapy shows no short-term benefit. However if systemic conditions permit, long-term discontinuation may be beneficial in stabilizing established sites of BRONJ, reducing the risk of new site development and relieving clinical symptoms. Discontinuation of oral bisphosphonate therapy in patients with BRONJ has been associated with gradual improvement in clinical disease. Discontinuation of oral bisphosphonates for 6–12 months might result in either spontaneous sequestration or resolution after debridement surgery. If systemic conditions permit, modification or cessation of oral bisphosphonate therapy should be done in consultation.

Staging categories and treatment strategies of BRONJ

The AAOMS has proposed the use of the staging categories and treatment strategies that are presented in Table 29.28:

- *Patients at risk.* Patients who have no apparent exposed or necrotic bone but have been treated with either intravenous or oral bisphosphonates are considered to be at risk of developing BRONJ. Although the patients of this group do not require any treatment, these patients should be informed of the risks of developing BRONJ.
- *Stage 1 patients.* Patients who show exposed, necrotic bone but are asymptomatic and have no evidence of infection. These patients benefit from the use of oral antimicrobial rinses, such as 0.12% chlorhexidine. No surgical treatment is indicated.
- *Stage 2 patients.* Patients who exhibit exposed, necrotic bone with pain and clinical evidence of infection. Pain should be controlled through the systemic use of analgesics. The use of oral antimicrobial rinses in combination with systemic antibiotic therapy is recommended. Preferred antibiotics include the members of penicillin group, quinolones, metronidazole, clindamycin, doxycycline, and erythromycin, although if microbiologic data is available, the antibiotic regimen should be adjusted accordingly. In some refractory cases, patients may require combination antibiotic therapy, long-term antibiotic maintenance, or a course of intravenous antibiotic therapy. Only superficial debridement to relieve soft tissue irritation is advocated.
- *Stage 3 patients.* Patients show exposed, necrotic bone with pain, infection, and one or more of the following: pathologic fracture, extraoral fistula, or osteolysis extending to the inferior border. Pain control is important for preservation of quality of

Table 29.28 Staging and treatment strategies for bisphosphonate-associated osteonecrosis.

Patient at risk	No apparent exposed/necrotic bone in patients who have been treated with either oral or IV bisphosphonates	No treatment indicated Patient education
Stage 1	Exposed/necrotic bone in patients who are asymptomatic and have no evidence of infection	Antibacterial mouthrinse Clinical follow-up on a quarterly basis Patient education and review of indications for continued bisphosphonate therapy
Stage 2	Exposed/necrotic bone associated with infection as evidenced by pain and erythema in the region of the exposed bone with or without purulent drainage	Symptomatic treatment with broad-spectrum oral antibiotics, e.g. penicillin, cephalexin, clindamycin, or first-generation fluoroquinolone Oral antibacterial mouthrinse Pain control Only superficial debridement to relieve soft tissue irritation
Stage 3	Exposed/necrotic bone in patients with pain, infection, and one or more of the following: pathologic fracture, extraoral fistula, or osteolysis extending to the inferior border	Antibacterial mouthrinse Antibiotic therapy and pain control Surgical debridement/resection for longer-term palliation of infection and pain

life. Surgical debridement/resection in combination with antibiotic therapy might offer long-term palliation with resolution of acute infection and pain, but deliberation is necessary to determine if an aggressive procedure is actually required.

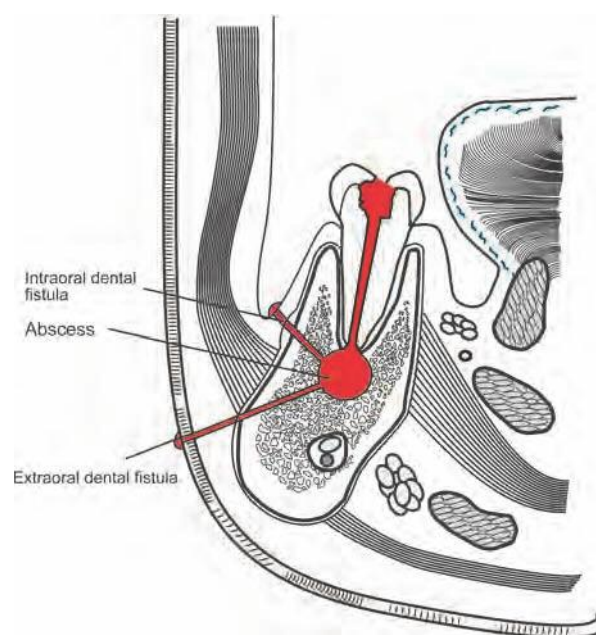
Dental fistulae

A fistula is an abnormal passage or communication between two internal organs or leading from an internal organ to the surface of the body. Various pathologic conditions are involved in the creation of a fistula. In the oral and maxillofacial region, odontogenic infections such as periapical abscesses, severe periodontitis, and pericoronitis are common causes of fistulae. Additional etiologies include peri-implantitis, osteomyelitis and osteoradionecrosis of the jaw, actinomycosis, postextraction infection, traumatic infection, and surgical wound infection (surgical site infection). A fistula can also involve non-infective entities such as salivary gland diseases, oral malignant tumor, and oroantral communication caused iatrogenically.

This section discusses the clinical features and management of those fistulae related to odontogenic infections (dental fistulae). Dental fistulae are often referred to as dental sinus tracts.¹¹⁹ Dental fistulae formed within the mouth are internal dental fistulae, and those created on the skin surface of the face are termed cutaneous dental fistulae or extraoral dental fistulae (cutaneous dental sinus tracts) (Fig. 29.60). In terms of pathological characteristics, a fistula consists of granulation and/or epithelial tissues.¹²⁰

Pathogenesis and clinical findings

The purulent by-products produced by odontogenic infection are initially collected within alveolar bone. The purulent by-products of pulpal infection will

**Fig. 29.60** Intraoral dental fistula and extraoral dental fistula.

seek the path of least resistance and move through bone and soft tissue. Once the cortical plate has been penetrated, the fistula's exit point is determined by the location of muscle attachments and fascial sheaths.

The series of paths (fistular tracts) commonly end within the connective tissue. However, occasionally they reach the surface of the oral mucosa or skin and establish the fistula. A fistula is usually created spontaneously but occasionally forms as a result of incision and drainage of an abscess. A small amount of pus and other purulent material can constantly drain through the fistular tract with the result that pus does not accumulate within the bone. This drainage releases the pressure inside the alveolar bone and prevents a transformation into an acute infection. Such drainage does, however, impede the healing of the fistula; as a result, once established, it may not close

spontaneously unless the focus is treated adequately or eliminated.

Intraoral dental fistulae

An intraoral dental fistula starts with an abscess in the alveolar bone and ends on the gingiva or on the surface of the palatal mucosa. A single fistula is common but one focus can produce multiple fistulae.

An intraoral dental fistula is usually seen as a small and well limited oval or round swelling with erythema, or as a polyp-like lesion (Fig. 29.61). Therefore, less experienced clinicians may sometimes regard the lesion as a neoplasm. This presentation results from inflammation and formation of granulation. If granulation is minimal, the fistula may develop as a 'crater-like' lesion without swelling. The fistula itself does not produce pain or tenderness.

Extraoral dental fistulae

An extraoral dental fistula is an abnormal passage or communication between lesions of chronic odontogenic infection within the alveolar bone and the cutaneous surface. An extraoral dental fistula commonly forms in the vicinity of the focus. In the majority of cases, the focus is a chronic infection of mandibular teeth and thus the fistula frequently appears on the chin, cheek, submental or submandibular region.



Fig. 29.61 Intraoral dental fistulae associated with periapical abscess (a) and pericoronitis (impacted tooth) (b).



Fig. 29.62 Extraoral dental fistula.

However, an extraoral dental fistula occasionally forms at a location away from the focus.

An extraoral dental fistula appears as a soft, slightly depressed nodule or papule on the skin, often fixed to underlying structures (Fig. 29.62). The lesion often has a central opening for discharge of fluid materials from the focus. Palpation of the surrounding tissue may produce pus or purulent materials, which could support the diagnosis.

Diagnosis

As intraoral and extraoral fistulae have similar clinical appearances to other types of lesions that can occur in the oral and maxillofacial region, they may be misdiagnosed, leading to inappropriate antibiotic therapy, aspiration or surgery.

Intraoral and dental examinations using radiographical analysis are necessary for fistula diagnosis (Fig. 29.63). The presence or absence of a periapical abscess, necrotic pulp tissue, periodontal disease, pericoronitis, crack or fracture of tooth, and foreign body in an extraction socket should carefully be determined. The fistular tract can be traced by gentle probing using an instrument with a blunt tip or by obtaining a periapical radiograph following insertion of a gutta-percha cone into the fistular tract (Fig. 29.64).

An extraoral dental fistula should be distinguished from a granuloma, benign and malignant tumor, actinomycosis, and furuncle. Tumor, mucocoele, and epulis need also to be differentiated from intraoral fistulae.

Treatment

Treatment of the focus is a necessity in the management of fistulae. Treatments include tooth extraction, endodontic therapy, apicectomy, professional periodontal treatment, and debridement of extraction socket. If the causative tooth is salvaged, then the risk of recurrence will generally increase. The persistence of a foreign body, infected granulation, dying bone or sequester in the socket after tooth extraction are also causes of recurrence.

Antibiotic therapy alone is not effective in completely resolving the fistula without recurrence. Similarly, surgical removal of the fistula without resolution of the focus also yields limited success.



Fig. 29.63 (a) Extraoral dental fistula involved in a periapical abscess in the vicinity of the mandibular first molar. (b, c) Careful oral inspection and radiographic examination illustrates the fistula and involved tooth.



Fig. 29.64 (a) Insertion of a gutta-percha cone into the fistular tract for tracing. (b) A radiograph indicates the involvement of an abscess at the root furcation of the mandibular molar.

In the case of an intraoral fistula, once the focus is treated adequately, the fistula will heal spontaneously without any additional treatment. However, in the case of an extraoral dental fistula, a combination of focus treatment and surgery for the fistula (Fig. 29.65) may be required because the fistula tract may harbor small foreign bodies or large amounts of infected granulations and epithelial tissue that may prevent complete healing and may cause recurrence. In addition, spontaneous closure of the fistula often causes a residual umbilication of the skin that may be esthetically unappealing (Fig. 29.66). If an esthetically unac-

ceptable scar persists after healing of the fistula, additional plastic surgery may be required.

Acute necrotizing ulcerative gingivitis

Acute necrotizing ulcerative gingivitis (ANUG) is unique in clinical presentation and course. Its clinical presentation is distinctive from all other periodontal diseases. ANUG is characterized by a rapidly progressive ulceration typically starting at the tips of the

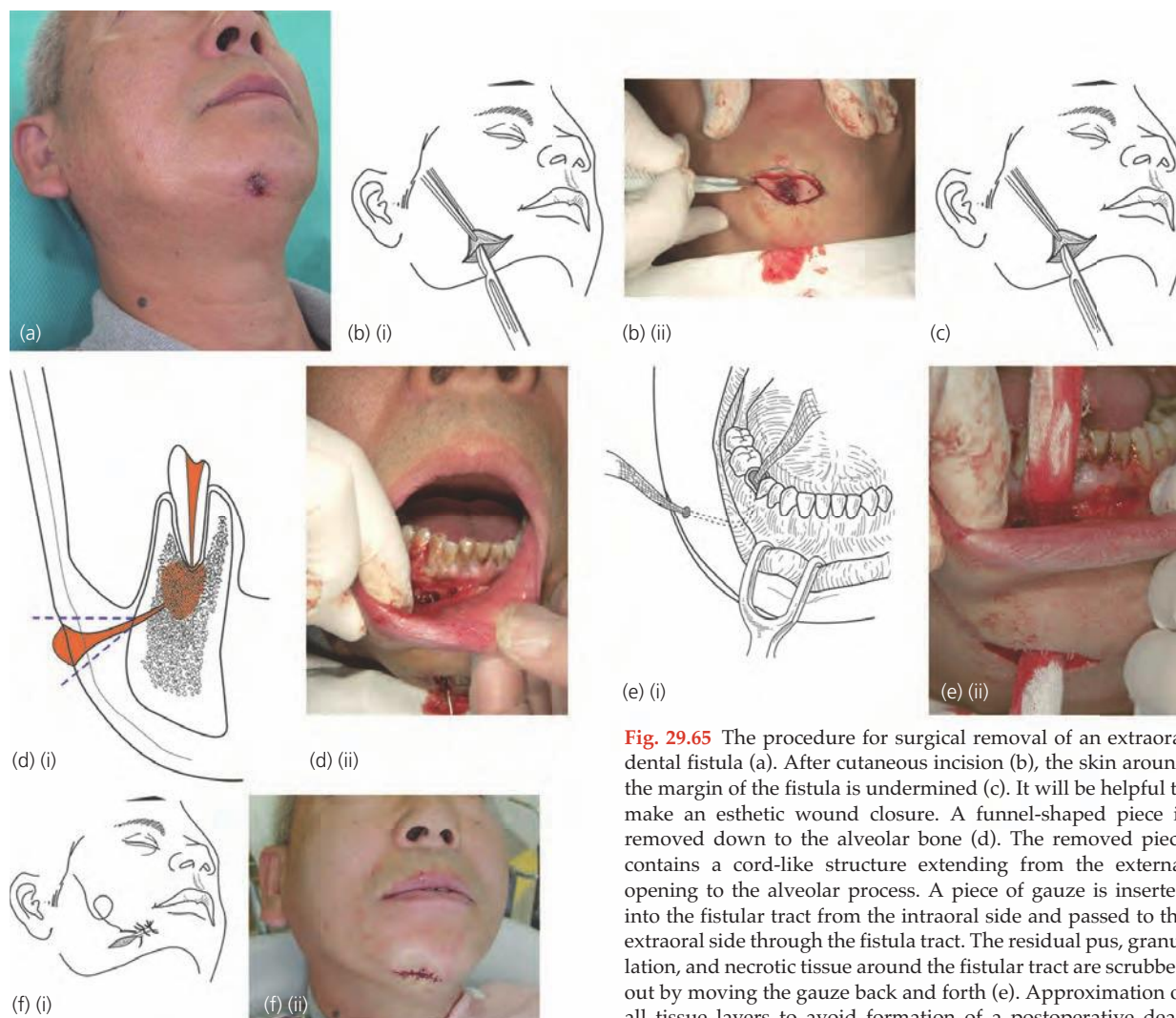


Fig. 29.65 The procedure for surgical removal of an extraoral dental fistula (a). After cutaneous incision (b), the skin around the margin of the fistula is undermined (c). It will be helpful to make an esthetic wound closure. A funnel-shaped piece is removed down to the alveolar bone (d). The removed piece contains a cord-like structure extending from the external opening to the alveolar process. A piece of gauze is inserted into the fistular tract from the intraoral side and passed to the extraoral side through the fistula tract. The residual pus, granulation, and necrotic tissue around the fistular tract are scrubbed out by moving the gauze back and forth (e). Approximation of all tissue layers to avoid formation of a postoperative dead space. Cutaneous surface is sutured esthetically (f). Illustrations were redrawn with permission from Ohtani T, *et al.*, editors, *Atlas of Oral and Maxillofacial Surgery*, vol 2. Tokyo: Ishiyaku Publishers, 1988.¹²¹



Fig. 29.66 Residual cutaneous umbilication associated with closure of an extraoral dental fistula.

interdental papilla, spreading along the gingival margins, and going on to acute destruction of the periodontal tissue with severe pain, interdental necrosis, and bleeding (Fig. 29.67).

Etiology and pathogenesis

The etiology of ANUG is not fully understood. However, spirochetes and *Fusobacterium* species are thought to be associated with the etiology. Recent reports suggest that other oral strict anaerobes, including *Prevotella* species, are also involved in the disease.¹²² Pre-existing gingivitis, nutritional deficiency, tobacco smoking, psychological stress, and immunosuppression have been implicated as predisposing factors.¹²³

Clinical features

The clinical presentation of ANUG comprises of a punched out ulcer, which is involved primarily with



Fig. 29.67 Acute necrotizing ulcerative gingivitis. Ulceration at the tips of the interdental papilla and destruction of the periodontal tissue are evident. In extreme cases, denudation of dental root occurs (right).

the tips of interdental papillae and the labial or lingual margin or both. Involvement may be a single tooth, a group of teeth or throughout the mouth, but the condition is rare in edentulous patients. The mandibular anterior region is most frequently affected.

The edge of the ulcer is usually defined by erythema and edema and often the lesion itself is covered by a grey pseudomembranous slough. The affected area often becomes highly sensitive to touch, and produces a constant radiating, gnawing pain, which is intensified by hot food or mastication. Spontaneous gingival bleeding upon exposure to the slightest stimuli can occur. In addition ANUG is often accompanied by marked halitosis and foul taste.

Mild to moderate cases may manifest as local lymphadenopathy with a slight elevation in body temperature. However, in its most severe form, high fever, increased pulse rate, leukocytosis, headache, loss of appetite, lassitude, and insomnia can all result.

ANUG may subside spontaneously, although the disease has the potential to cause progressive destruction of periodontium and denudation of dental root with systemic complications.

Diagnosis

The patient's clinical history and symptoms are often sufficient information to permit diagnosis. If there is uncertainty, ANUG can be confirmed rapidly by microscopic examination of a Gram-stained smear taken from an area of ulceration and should reveal numerous fusobacteria, medium-sized spirochetes and acute inflammatory cells.

Management

Initial management should involve a thorough mechanical cleaning and debridement of the teeth in the affected area. The use of hydrogen peroxide mouthwashes, both to provide mechanical cleaning and also to serve as an oxidizing agent, has been recommended, although the benefit of such treatment is not universally accepted. The importance of local

measures cannot be overemphasized, but symptoms will improve more rapidly if the patient is also given a systemic antimicrobial agent. Metronidazole (200mg 8-hourly or after meals) prescribed for 3 days will usually produce a dramatic improvement within 48 hours. In the long term, good oral hygiene therapy prevents further gingival damage.

Peri-implantitis

Dental implant treatment aims to restore oral function that has been disrupted through the loss of teeth. The surgical placement of an implant has become a widely accepted approach to replace missing teeth. Despite various types of implant being available, the large majority of them are designed to osseointegrate within the bone. Osseointegration is defined as the direct structural and functional connection that occurs between living bone and the surface of implant. Despite most implants exhibiting long-term success, complications may occur in a small percentage of cases. Any pathologic changes that occur in the peri-implant tissue are generally categorized as a form of peri-implant disease. Inflammatory changes confined to the soft tissue surrounding an implant are diagnosed as peri-implant mucositis (Fig. 29.68). Progressive peri-implant bone loss in conjunction with a soft tissue inflammatory lesion is termed peri-implantitis. Despite a limited incidence of peri-implantitis, the condition is frequently encountered by dentists and this simply reflects the high number of people who currently receive implant treatment. As with odontogenic infections, peri-implantitis has the potential to cause extensive maxillofacial cellulitis, osteomyelitis, and maxillary sinusitis.

Etiology and pathogenesis

Although the exact etiology of peri-implantitis remains unclear, the infection is thought to occur due to the presence of oral anaerobic bacteria in plaque biofilm adhering to the implant. Of course, host susceptibility to infection is a significant factor in

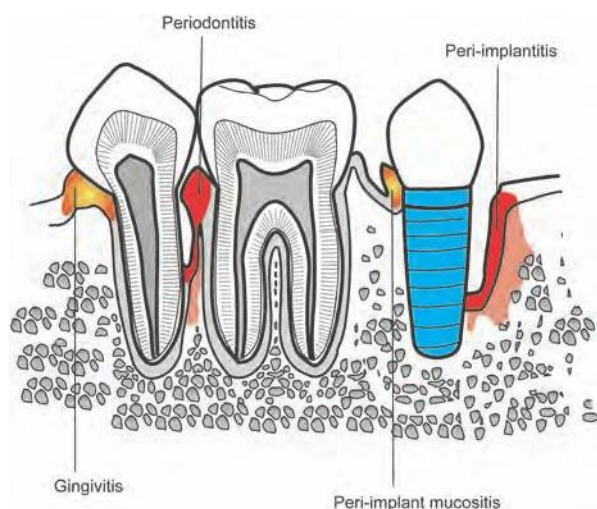


Fig. 29.68 Peri-implant diseases are divided into peri-implant mucositis and peri-implantitis. Peri-implantitis involves bone loss, whilst the bone is not significantly affected in the case of peri-implant mucositis. Peri-implant mucositis and peri-implantitis are analogous to gingivitis and periodontitis in natural teeth, respectively.

addition to the bacterial contamination. Unlike in natural teeth, there is no periodontal ligament between the implant and supporting bone. In addition, the arrangement of collagen fibers around implants is different from that of natural teeth. It is these differences in the nature of attachment of soft tissue to both the implant and natural tooth, which result in peri-implant tissues being more sensitive to bacterial infection and tissue breakdown.

Poor oral hygiene, smoking and a previous history of periodontitis predispose patients to peri-implantitis.¹²⁴ An immunocompromising condition, impaired bony metabolism, and inadequate implant surface are also suggested to be risk factors.

Microbiology

Implantitis with advanced pocket formation yields high levels of spirochetes and Gram-negative bacilli (*Fusobacterium* species, *Prevotella* species, *Porphyromonas* species, *Tannerella forsythensis*, and *Aggregatibacter actinomycetemcomitans*), whereas successful implants are characterized by very low numbers of cultivable organisms with a predominance of Gram-positive cocci.¹²⁵ These microbial shifts are very similar to those that occur around natural teeth.

Clinical features

Peri-implant mucositis is analogous to gingivitis and is primarily a disorder caused by plaque accumulation. It may be identified clinically by bleeding on probing, and redness and swelling of the soft tissue.

Peri-implantitis usually produces little subjective symptoms in its initial phase. As it progresses, gingival bleeding and tenderness, pain at chewing, purulent exudates from the pocket, malodor, bad



Fig. 29.69 (a) Intraoral photograph of a patient with severe peri-implantitis. Pus discharge from the pocket with erythema is evident. (b) A radiograph shows significant bone loss around the implants.

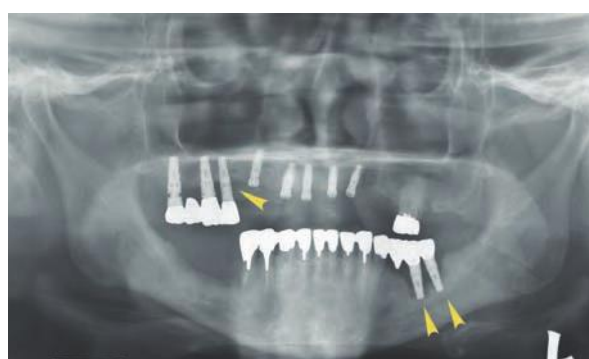


Fig. 29.70 A radiograph of a patient with various degrees of peri-implantitis.

taste, and gingival recession with gingival swelling and erythema will appear (Fig. 29.69). Bone levels decrease with the loss of integration (Fig. 29.70). Bone destruction may proceed without any notable signs of implant mobility until osseointegration is completely lost, because the bottom part of the implant may still retain a degree of osseointegration.

Diagnosis

The clinical diagnosis of implantitis is generally based on inspection of the implant and peri-implant tissues, the patient's subjective symptoms, probing of the pockets, and radiographic examination.

The bone supporting the implants might be absorbed from the alveolar crest side as a normal postoperative alteration (vertical bone loss, <0.2 mm, annually after first year of function)¹²⁶ and this should be taken into account in the radiographic study. Probing of the pockets is useful to measure pocket depth and to determine the presence or absence of bleeding on probing and suppuration. Due to the previously mentioned differences in attachment between implants and natural teeth, actual pocket depths are not used as determinative indices of the disease as they are for periodontitis. A significant increase in the pocket depth and bone loss in the follow-up period suggests peri-implantitis and its progression.

Treatment

Treatment for salvaging the implant consists of the patient's own self-care with respect to their oral hygiene and also professional treatment. The implant surface is usually contaminated with soft tissue cells, bacteria, and bacterial by-products. These contaminants will compromise therapeutic effects and induce further breakdown of the peri-implant tissue. Subgingival debridement, removal of calculus and cleaning, and decontaminating the implant surface are therefore necessary.

Mechanical cleansing and local irrigation can be effective in the treatment of peri-implant mucositis. Furthermore, the adjunctive use of antimicrobial mouthrinses may enhance the outcome of mechanical therapy of mucositis lesions.

Treatment of peri-implantitis is generally difficult, and there are no treatment principles that have been universally accepted. Non-surgical therapy alone may not be effective. It has been reported that guided bone regeneration (GBR) and bone graft techniques result in various degrees of success.¹²⁴ However, such techniques do not address disease resolution but rather merely attempt to fill the osseous defect. Implant removal should be considered in cases where osseointegration has been reduced severely and bone loss has extended into the apical half of the implant, when the implant demonstrates mobility, or if peri-implantitis causes significant suppuration. After the implants are removed, the ridge defects usually reconstruct spontaneously.

If peri-implantitis causes extensive infection, such as maxillofacial cellulitis, osteomyelitis, and maxillofacial sinusitis, appropriate treatment is required. Due to the similarity in microbiology and pathogenesis, the principles of treatment of implant diseases are the same as for diseases of odontogenic origin.

Infection of the maxillary sinus

Maxillary sinusitis is an inflammation of the maxillary sinuses. As described in the Biomedical sciences

section of this chapter, the maxillary sinuses are primarily lined by mucoperiosteum. The antral mucus glands produce mucus and this normally drains to the nose through the sinus opening or ostium. The secretion and drainage of mucus are parts of the local host defense system and play an important role in maintaining microbial homeostasis in the sinus. If infection or an allergic reaction affects the sinus, the antral mucosa may become thickened with pathogenic associated changes, including edema, degeneration, hyperplasia or hypertrophy. Mucosal thickening may induce obstruction of the ostium with an associated reduction in mucus drainage and subsequent overgrowth of pathogenic bacteria. If left untreated or inadequately treated, infection of the maxillary sinus may spread to involve other subgroups of the paranasal sinus.

Maxillary sinusitis can be divided into acute and chronic, based on duration of disease, and odontogenic or non-odontogenic based on the etiology. Non-odontogenic factors, especially upper respiratory tract infections and allergic reactions, are the most common etiologies, with approximately 10% of all maxillary sinusitis cases being of dental origin.¹²⁷

Maxillary sinusitis of odontogenic origin

Unlike non-odontogenic sinusitis, odontogenic maxillary sinusitis usually manifests unilaterally and its pathophysiology, microbiology, and management are different from those of non-odontogenic sinusitis.

Etiology and pathogenesis

Acute odontogenic maxillary sinusitis primarily results from multiplication of bacteria invading from the mouth or the focus of a dental infection. The distance between the dental root apices and the antral floor generally correlates with the likelihood of sinusitis. Since the maxillary premolar and molar teeth have the closest proximity to the antral floor, infection of these teeth, as seen with periapical abscesses, severe periodontitis, peri-implantitis, and postextraction infection, is the most common cause of the disease (Fig. 29.71). Additional etiologies include

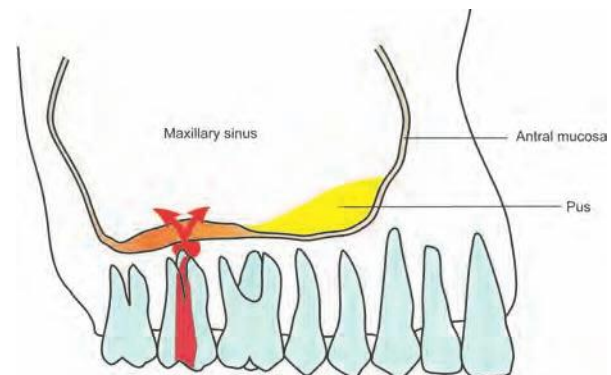


Fig. 29.71 Multiplication of bacteria invading from the focus of a dental infection results in odontogenic maxillary sinusitis.

dental or alveolar trauma, odontogenic cysts, maxillary osteomyelitis, iatrogenic or accidental displacement of foreign bodies (e.g. teeth, root tips, dental implants, osseous fragments, bone graft materials, endodontic therapy materials, and fragments of broken instruments) during routine dental treatment or dentoalveolar surgical procedures (e.g. dental extraction and dental implant surgery), and other surgical complications that result in sinus exposure.

Chronic odontogenic sinusitis results from a prolonged low-grade inflammation in the antral mucosa following acute phase or recurrence of acute sinusitis. Some cases induce a primary chronic form that manifests without an acute episode. In chronic sinusitis, the antral mucosa is thickened with edema, infiltration of leukocytes and fibers, sometimes accompanied by the creation of polyps.

Microbiology

Maxillary sinusitis usually involves mixed microflora. Acute sinusitis of non-odontogenic origin predominantly yields aerobic bacteria, in particular *Streptococcus pneumoniae*, *Haemophilus influenzae*, *Moraxella catarrhalis*, and *Staphylococcus aureus*, which are detectable in disease-free nasal cavities.^{128,129} In contrast, the microbiology of acute odontogenic sinusitis is somewhat similar to that of a dentoalveolar abscess. The most frequent bacteria isolated include viridans streptococci and anaerobes such as *Peptostreptococcus*, *Porphyromonas*, *Prevotella*, and *Fusobacterium*.¹³⁰ Nevertheless, bacteria residing in the nasal cavity and nasopharynx may be present in the microflora of odontogenic sinusitis.¹³⁰ Therefore the microbiology of this disease is complex with a wide variety of organisms involved.

The microflora of chronic sinusitis is polymicrobial, with viridans streptococci and anaerobes, and is similar to the organisms isolated from acute odontogenic sinusitis.¹³⁰ However, clinical specimens from chronic sinusitis often yield reduced numbers of bacteria.

Clinical signs and symptoms

Acute odontogenic maxillary sinusitis

As with other acute infections, acute maxillary sinusitis results in pain and systemic upset. Common signs and symptoms include dull or intense pressure-like pain, erythema, swelling of the cheek and the anterior maxilla, pressure or fullness in the vicinity of the maxillary sinus, headache, malaise, fever, oral malodor, mucopurulent rhinorrhea, nasal congestion or obstruction, and drainage of foul-smelling mucopurulent materials into the nasal cavity and nasopharynx (postnasal drip). Edema of the eyebrow occasionally accompanies the infection. In cases originating from periapical abscesses, the disease often starts with toothache and swelling in the gingiva and the buccal vestibule. A blood examination demon-



Fig. 29.72 Discharge of pus from the antral cavity through the extraction socket.

strates increased leukocyte count and an elevated CRP level.

Acute maxillary sinusitis has the potential to cause serious complications. Of particular significance are orbital cellulitis, cavernous sinus thrombosis, meningitis, and intracranial abscesses, as these can lead to a loss of vision, severe neurologic deficits, or even death.

Chronic odontogenic maxillary sinusitis

Unlike acute forms of the infection, little or no systemic upset occurs with chronic odontogenic maxillary sinusitis, although transformation into the acute form can occur spontaneously. Local signs and symptoms are generally subtle but malodor, persistent pus discharge from the oroantral fistula (Fig. 29.72), nasal congestion, and discharge, with or without a postnasal drip, are not uncommon. Toothache during chewing, increased mobility of teeth, migraine, dull headache, and anterior maxillary tenderness are also possible symptoms. Some patients have little discomfort, probably due to incomplete osteomeatal obstruction. Such "symptom-free" sinusitis may be detected by chance when performing radiographic analysis during routine dental practice or when encountering "abnormal" pus discharge from a root canal or extraction socket during dental treatment. There is generally no specific blood test for diagnosis and indeed little change in blood test parameters are associated with this infection.

Diagnosis

Imaging

Radiologic imaging is an important tool in establishing correct diagnosis. Conventional Water's, frontal, and lateral radiographs have been used in establishing a diagnosis. Due to thickened antral mucosa and fluid accumulation, a range of opacification in the affected sinus is often evident. Since the disease is usually unilateral, comparison in opacification between opposite sides is often helpful. Radiographic translucency level is generally lower in the affected

sinus than in the unaffected site (Fig. 29.73). Periapical and panoramic radiographs are useful in determining the probable source and assessing the relation and proximity between the root apices and the antral floor. A CT scan is often considered the gold standard imaging method for the maxillary sinus in developed countries and it provides much more reliable information compared with traditional radiographs.

Diagnosis principles

Diagnosis of the maxillary sinusitis of odontogenic origin is generally based on a careful dental and medical examination that includes evaluation of the patient's signs and symptoms, past medical history, adequate imaging studies, and laboratory examination. In addition, otolaryngological evaluation using rhinoscopy, nasal and sinus endoscopy, and aspiration of sinus contents for cytologic and microbiologic assessments can assist in making the correct diagnosis.

Differentiation from carcinoma, aspergillosis, and cystic disease in the maxillary sinus or maxillary bone is required because of their similar features in imaging studies.

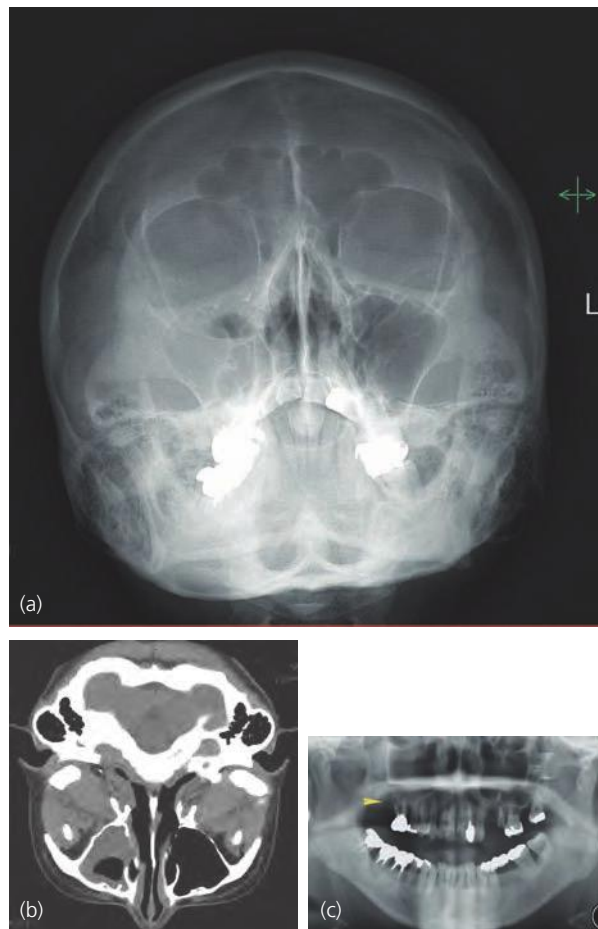


Fig. 29.73 (a) The Water's radiograph reveals opacification of the affected sinus, in which the radiographic translucent level is lower compared with the unaffected site. (b) CT scan demonstrates thickened sinus mucosa and fluid accumulation within the affected sinus. (c) Periapical abscess of the maxillary first molar is the probable cause as shown on the panoramic radiograph.

Treatment principles

Both elimination of the infection source and treatment of the antral lesion are necessary for the complete resolution and prevention of recurrence. Once treatment starts, the patient should be carefully evaluated until the condition has completely resolved.

Acute maxillary sinusitis

In the case of acute maxillary sinusitis, antimicrobial therapy is an essential part of treatment. Early and aggressive antibiotic therapy and elimination of the source may provide a full cure without prolongation of the inflammatory process. Penicillin, clindamycin, and metronidazole are adequate drugs of initial choice. However, because of the range of microorganisms that may be involved, purulent material should be obtained for culture and sensitivity testing whenever possible and the choice of antibiotic should be based on these laboratory results. Individual treatment modalities will generally result in more successful outcomes. An increase in drug dose and intravenous administration of antibiotic are especially recommended in moderate to severe cases.

Relief of pain and reduction of the nasal mucosal edema are important and can be achieved through the use of analgesics and antihistamines, respectively.

Drainage serves to reduce pain intensity, prevents disease progression, and encourages resolution. Early tooth extraction and endodontic therapy are recommended because the extraction socket and opened root canal are then available as a route of drainage. Insertion of a piece of gauze containing 0.1% ephedrine (with 4% lidocaine) into the nasal mucosa for 10–15 minutes can relieve mucosal congestion and may result in the promotion of pus drainage through the nasal passage as it expands the sinus ostium. Surgical enlargement of the natural drainage channels using an endoscope is also effective although it may need the involvement of a specialist of sinus endoscopic surgery.

The treatment outcome should be evaluated based on clinical signs and symptoms, laboratory tests, and imaging studies. Since acute maxillary sinusitis can readily spread to involve other paranasal sinus subgroups with the potential to cause serious complications, patients should be evaluated frequently. If occurrence of such complications is suspected, immediate and aggressive care by a specialist is required.

If little or no response to treatment occurs within several days after start of treatment, it is necessary to reassess the treatment strategy and antibiotic selection. Inadequate drainage with accumulation of pus is the most likely cause of a treatment failure. Drainage by tooth extraction or through the root canal should be reconsidered. Adjacent teeth should also be re-evaluated for possible involvement. Surgical drainage through the nasal passage using an endoscope is an option in severe cases. Depressed

host defense mechanisms can be involved in poor response to treatment. Such factors that may delay recovery should be reassessed and corrected. The involvement of antibiotic-resistant bacteria may be a factor affecting therapeutic outcome, highlighting the importance of selecting the antibiotic regimen based on microbiologic analysis of the clinical specimen. If these data are unavailable, an increased drug dose, or administration of antibiotics with β -lactamase stability or broad antimicrobial spectrum, such as imipenem, ampicillin-sulbactam or co-amoxiclav, should be considered. The authors suggest the use of intravenous clindamycin at high doses in cases where penicillin therapy has failed.

After the visual signs of acute infection (e.g., pain, swelling, fever) have subsided and the infection source has been eliminated, the disease commonly takes one of the following courses:

- Sinusitis heals completely without any post-treatment problems. This is often the course of "fresh" sinusitis with no previous episodes of recurrence, in which the sinus mucosa is not seriously damaged. Early diagnosis and adequate treatment for both sinusitis and disease source are necessary in order to obtain such an outcome. For prevention of recurrence, an additional course of oral antibiotic should be administered within a period of 1–2 weeks.
- Sinusitis itself resolves but an oroantral fistula persists. This situation may occur if a patient has oroantral communication or the fistula was created before treatment (usually involved in development of the present sinusitis) or results from tooth extraction during the present treatment of disease. Since long-standing oroantral communication and oroantral fistulae do not heal spontaneously and can be a cause of disease recurrence, surgical closure is required.
- Symptoms associated with low-grade inflammation in the sinus (e.g. persisting pus discharge from oroantral fistula, malodor, nasal congestion and discharge, postnasal drip, dull headache) persist. In such cases, the disease may be regarded as a chronic infection, although it is necessary to determine if these symptoms are actually associated with the sinusitis. Administration of different types of antibiotics for several more weeks may be warranted. However, most cases require surgical intervention to provide a complete cure. If an oroantral fistula is present, it should be closed surgically after sinus infection is eliminated.
- Visual signs and subjective symptoms are minimal but resolution on imaging study is not obtained in the follow-up period (12 weeks after the treatment). It generally takes time to obtain radiographic resolution, but in this case, low-grade inflammation in the sinus may persist. A follow-up for an additional period whilst reserving sinus surgery is advocated, and the patient should be informed of the increased

risk of infection recurrence and possible necessity of surgical intervention to ensure complete eradication of the infection.

Chronic odontogenic maxillary sinusitis

Treatment involves elimination of the infection source, antibiotic therapy and surgery, with the antibiotic regimen being similar to that for the acute form.

Conservative intervention may be attempted initially, without resorting to sinus surgery. Elimination of the dental source by tooth extraction, apicectomy, endodontic therapy, removal of any involved foreign body, and antibiotic therapy might lead to full recovery. If an oroantral fistula is present, frequent irrigation of the sinus cavity via the fistula can prove effective, although surgical closure of the fistula is required after sinusitis is cured. However, if complete resolution is not achieved by these treatments or if the clinician considers that the disease may not heal using conservative approaches then surgery will be required.

Surgery

The goal of sinus surgery is to remove abnormal tissue that exists inside the sinus cavity and may be involved in the disease or cause delayed healing. Surgery also serves to restore normal drainage through the original antral ostium or created sinus opening.

The Caldwell–Luc operation, a radical surgical procedure, has traditionally been employed to treat chronic sinusitis. In this operation, sinus mucosa is stripped from the antral wall, on the basis that the mucosal lesion is irreversible. However, it has been demonstrated that this pathogenic condition is reversible and the antral mucosa can be conserved through correction of the underlying obstruction by minimal surgical procedures including endoscopic sinus surgery (functional endoscopic sinus surgery).¹³¹ Sinusitis of non-odontogenic origin is primarily treated today with endoscopic sinus surgery. In cases of odontogenic sinusitis, the Caldwell–Luc operation is still widely used, but use of endoscopic sinus surgery is increasing.

Caldwell–Luc procedure

The Caldwell–Luc procedure involves the complete removal of the antral lining and creation of a new opening for more dependent drainage into the nose by the transoral approach (Fig. 29.74). This procedure allows the surgeon to inspect the inside of the sinus with the naked eye and any abnormal tissue or foreign bodies in the sinus can then be easily removed. Moreover, removal of a cyst, apicectomy of causative teeth, and surgical closure of the oroantral fistula can be performed concomitantly using the same surgical wound. This procedure is also useful in cases where endoscopic sinus surgery has failed. However, there are several disadvantages, including increased times

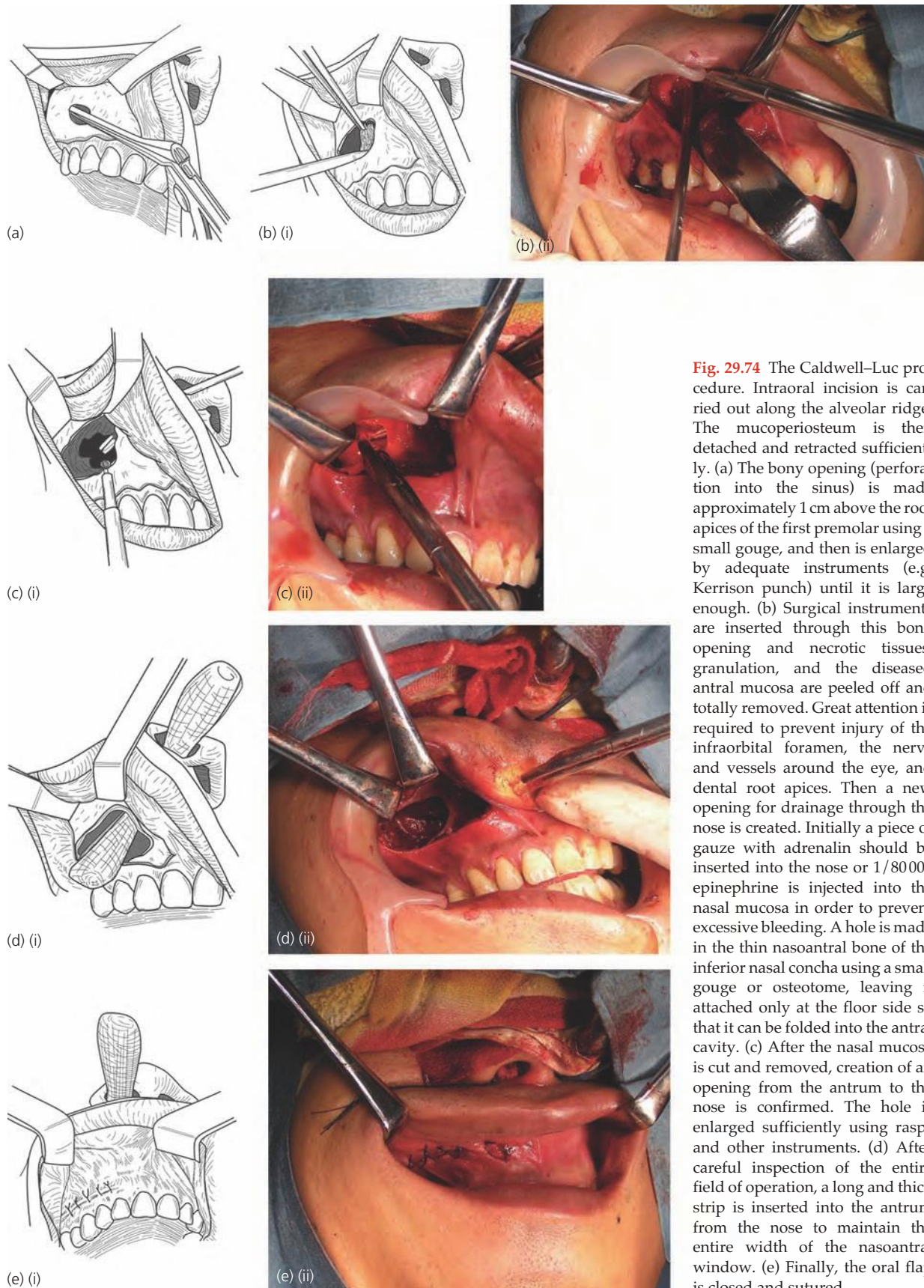


Fig. 29.74 The Caldwell-Luc procedure. Intraoral incision is carried out along the alveolar ridge. The mucoperiosteum is then detached and retracted sufficiently. (a) The bony opening (perforation into the sinus) is made approximately 1 cm above the root apices of the first premolar using a small gouge, and then is enlarged by adequate instruments (e.g. Kerrison punch) until it is large enough. (b) Surgical instruments are inserted through this bony opening and necrotic tissues, granulation, and the diseased antral mucosa are peeled off and totally removed. Great attention is required to prevent injury of the infraorbital foramen, the nerve and vessels around the eye, and dental root apices. Then a new opening for drainage through the nose is created. Initially a piece of gauze with adrenalin should be inserted into the nose or 1/80 000 epinephrine is injected into the nasal mucosa in order to prevent excessive bleeding. A hole is made in the thin nasointral bone of the inferior nasal concha using a small gouge or osteotome, leaving it attached only at the floor side so that it can be folded into the antral cavity. (c) After the nasal mucosa is cut and removed, creation of an opening from the antrum to the nose is confirmed. The hole is enlarged sufficiently using rasps and other instruments. (d) After careful inspection of the entire field of operation, a long and thick strip is inserted into the antrum from the nose to maintain the entire width of the nasointral window. (e) Finally, the oral flap is closed and sutured.

of recovery, greater blood loss, and increased postoperative pain and facial swelling compared with endoscopic sinus surgery. In addition, resulting sinus scarring may be associated with recurrence; the procedure may also cause injury to the nerve responsible

for sensation to the maxillary skin and result in permanent disturbance of the natural function of the sinus lining. The Caldwell-Luc procedure should not be used in children as it seriously affects the normal growth of the maxillary sinus.

Endoscopic sinus surgery

Endoscopic sinus surgery uses fiberoptic technology. The endoscope is a very thin fiberoptic instrument, which visualizes the antral condition with minimal surgical wounding. This surgery is performed entirely through the nostrils, leaving no external scars. Its goal is to open the natural drainage channels, restoring normal physiologic function of the sinus mucosa. Materials blocking the sinus openings and any foreign bodies are removed using surgical instruments that are inserted alongside the endoscope. There is little swelling and only mild discomfort after surgery. As this operation needs special medical instruments and careful surgical technique, it should only be performed by an appropriate specialist.

Adjunctive treatments**Surgical removal of foreign bodies from inside the maxillary sinus**

Any foreign body displaced into the sinus cavity must be surgically removed. Small foreign bodies, such as teeth, root tips, broken instrument fragments, and dental implants, can be retrieved with small forceps, or the use of suction through the expanded extraction socket or a small bony opening that is intentionally created in the canine fossa (Fig. 29.75). On the other hand, if such procedures are difficult, the Caldwell–Luc operation may be required. It is important initially to locate any foreign body in the sinus using radiographic analysis.

After removal of any foreign bodies, the sinus cavity should be washed with saline to reduce risk of contamination, and antibiotic therapy should be administered to prevent infection, even if there is not unequivocal evidence of infection in the antral cavity and wound.

Surgical closure of oroantral communications

Extraction of the tooth sometimes induces an oroantral communication. The defect usually closes spontaneously with normal blood clot formation and routine mucosal healing. However, if the defect is large or persists for a long period, it may not heal spontaneously and may need surgical closure (see the later section, Oroantral communication and fistulae).

Influence of non-odontogenic maxillary sinusitis on the teeth

As maxillary premolar and molar teeth are located close to the floor of the sinus, non-odontogenic sinusitis can affect these teeth. Its influence ranges from mild and reversible irritation in the pulp and periodontal ligament to significant damage that may cause irreversible pulpitis and pulpal necrosis. If the patient has a toothache in teeth with no apparent dental disease, and any symptoms possibly associated with sinusitis such as a stuffy nose, involvement of non-odontogenic maxillary sinusitis may be suspect-

ed. Diagnosis and treatment should be carried out with collaboration between the dentist and a specialist (otolaryngologist).

Fungal sinusitis (*Aspergillus* mycetoma of the maxillary sinus)

Occurrence of fungal infection (mycosis) in the paranasal sinuses is relatively rare. The vast majority of cases involve *Aspergillus* species. *Aspergillus*-associated sinusitis is also called *Aspergillus* mycetoma of the maxillary sinus, aspergilloma or mycetoma of paranasal sinus. *Aspergillus* species are opportunistic fungi and widely distributed in the environment.

The majority of *Aspergillus*-associated sinusitis cases are unilateral.¹³² The disease usually occurs in adult patients with a female predominance.¹³³

Amongst members of *Aspergillus* species, *Aspergillus fumigatus* is the most common isolate.¹³² Although the exact etiology of *Aspergillus*-associated sinusitis remains unclear, the disease is believed to result from opportunistic entry of the fungus into the sinus or due to iatrogenic circumstances. Previous over-extension of the root canal sealer into the maxillary sinus during root canal treatment is thought to be an etiological factor, as treatment materials are known to carry the fungus.¹³⁴

The disease can be divided into two clinical forms, non-invasive and invasive. The non-invasive form may be seen in immunocompetent patients, whilst the invasive form usually occurs in hosts with an immunodeficiency. The majority of *Aspergillus*-associated sinusitis is non-invasive. In the case of the non-invasive form, a “fungal ball” may form within the affected sinus. The common clinical presentation is not specific, with various symptoms such as nasal obstruction, purulent nasal discharge, facial pain, and a chronic cough. Thick, tenacious, dark secretions may be characteristic signs. Some cases are asymptomatic, and, in these, the disease is often detected accidentally following radiographic study during routine dental treatment. In contrast, the invasive form may induce serious symptoms such as ophthalmoplegia, ptosis, frontal hypoesthesia, postorbital pain, and visus abnormalities, resulting from spreading to the orbit and skull. The prognosis of the invasive form can involve serious sequelae or even death.

Calcifications in the affected sinus may be seen on plain film radiography or in CT scans (Fig. 29.76). In the case of invasive form, bony destruction is also evident. Differentiation from maxillary sinus carcinoma is required. Culture of clinical specimens often does not result in the isolation of *Aspergillus*, and in many cases, diagnosis is made with pathological examination of a specimen obtained by biopsy or debridement.

It is necessary to surgically remove thick, dark, greasy materials (the sinus fungal masses) and to ensure the establishment of adequate sinus drainage

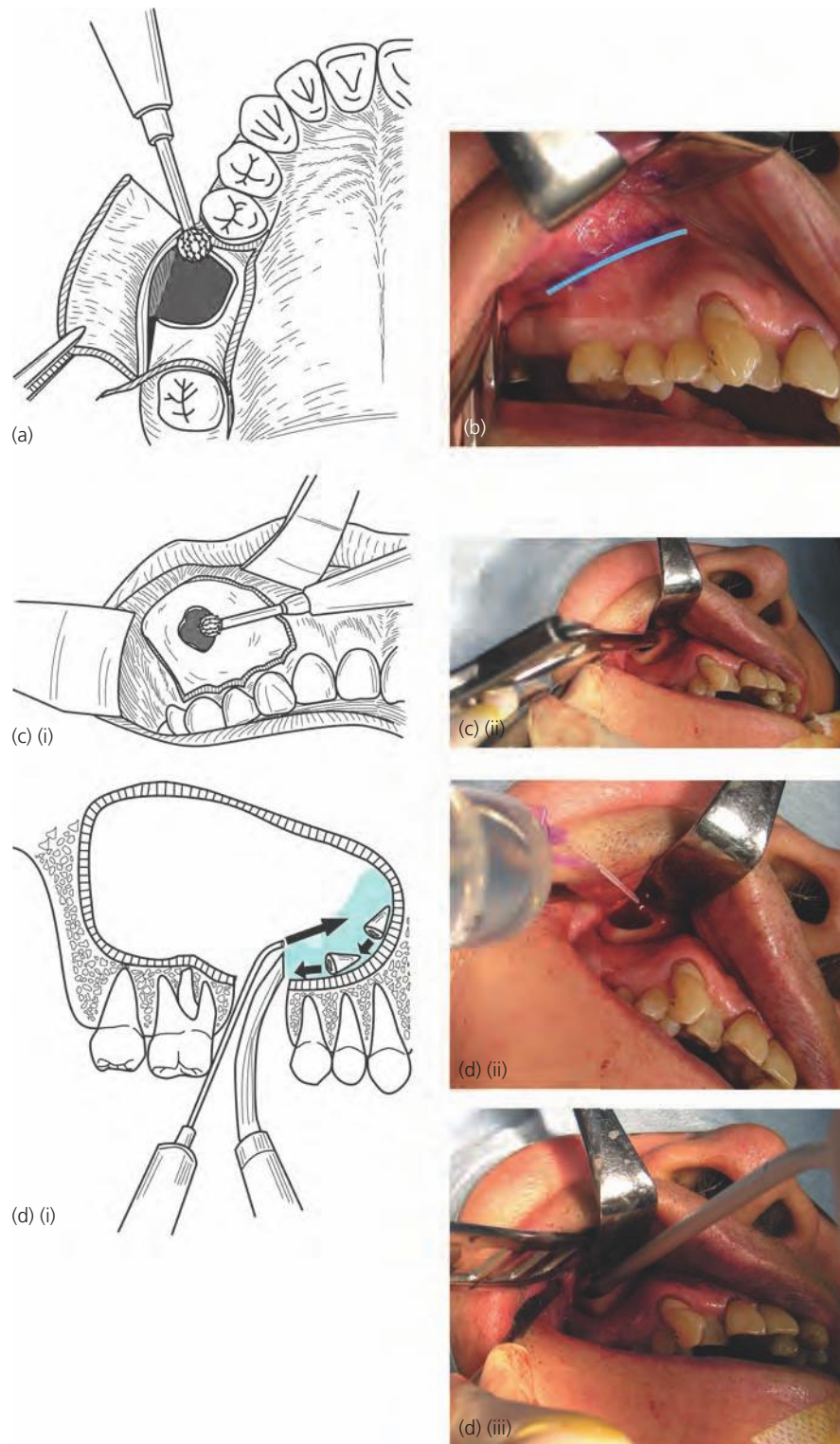


Fig. 29.75 A foreign body displaced into the antral cavity can be retrieved with small forceps and with the use of suction through the expanded extraction socket or a bone opening created in the canine fossa. (a) If the dental root or foreign body is displaced from extraction socket, the socket may be enlarged buccally after elevation of a mucoperiosteal flap to expose the maxilla above the socket. If this is difficult or inapplicable, an opening in the canine fossa should be made as follows. (b) A mucoperiosteal flap is made around the canine–premolar recess. (c) After the flap is reflected, a new small oronasal opening is created in the bone, 1 cm above the root apices of the first premolar. (d) Saline solution is injected into the antral cavity to flood the sinus through the expanded socket or the opening and then a suction tube is inserted. The foreign body is likely to be sucked out together with saline solution or moved close to the opening for easy retrieval. After removal of foreign body and irrigation of sinus using saline, the wound is primarily closed. Use of short-term prophylactic antibiotic is recommended.

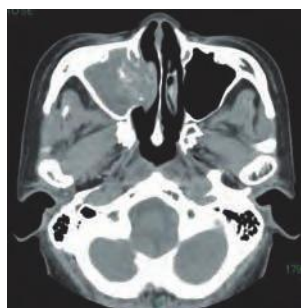


Fig. 29.76 CT of *Aspergillus*-associated sinusitis. Calcifications in the affected sinus are seen. Photograph courtesy of Dr T. Miwa (Kanazawa University).

and aeration via the Caldwell–Luc procedure or endoscopic sinus surgery. There is little tendency of infection recurrence after successful removal of the fungal masses and the use of systemic antifungal therapy for the non-invasive form is generally unnecessary. However, the invasive form may require the use of systemic antifungal therapy after debridement.

Oroantral communication and fistula

An oroantral communication is an abnormal connection between the oral and antral cavities. Various factors such as cysts, trauma, tumors, and even minor dentoalveolar surgical procedures can be involved in this pathogenic condition. The extraction of the maxillary posterior teeth, however, is the most common cause, because of the anatomically close relationship between the root apices and the maxillary antrum, and the thinness of the antral floor in that region. Despite discrepancies between studies, the maxillary second molars have been reported to be the teeth closest to the base of the sinus.¹³⁵ The root apices of these teeth are separated from the floor of the sinus by only a thin bony lamella and antral mucous membrane. In extreme cases, only the mucous membrane exists between the roots and the sinus floor, with a lack of bony intervention. Periapical infection destroys the bone near the floor of the maxillary sinus and it raises the risk of oroantral communication resulting from extraction of the tooth.

If oroantral communication is left open, epithelial tissue may develop in its track. As previously described, such a situation is defined as a fistula. Oroantral communication can close spontaneously, although once a fistula is established, it usually does not heal without surgical intervention.

Saliva harbors a large number of bacteria. Unlike oral mucosa, the antral mucosa is sensitive to salivary contamination. A long-standing oroantral communication or fistula provides the constant exposure of antral mucosa to saliva and other oral contaminants. Therefore such a condition substantially raises the likelihood of antral infection and its recurrence. The duration and width of the lumen of the fistula canal generally correlates with the likelihood of sinusitis,



Fig. 29.77 Oroantral fistula. The defect is covered with granulation and epithelium, but a small defect is seen on careful inspection.

although infection of the sinus is possible in a short period and with a very narrow fistular canal.

Clinical findings and diagnosis

Clinical symptoms of oroantral communication and fistulae are similar. Common symptoms include an unpleasant tasting discharge and odor, reflux of fluids and foods into the nose from the mouth, leakage of air, and difficulty in tobacco smoking. The presence of these symptoms can be indicators for diagnosis, although some patients remain asymptomatic.

In the case of a new oroantral communication created by tooth extraction, osseous defects and communication between the mouth and antrum may be confirmed by careful visual inspection with a dental mirror and good light. Since hemorrhage at the surgical site often obscures visibility, inspection should be performed after hemostasis. Gentle suctioning of the socket may produce a hollow sound that again is an indicator for diagnosis. It is of value to ask whether the patient feels leakage of air when blowing against the closed nostrils. Probing is generally not recommended because insertion of an instrument into the antral cavity itself can cause the antral perforation, and may also bring oral contaminants into the sinus.

In the case of a long-standing oroantral fistula, the fistular tract is often filled with granulations and epithelium (Fig. 29.77). Therefore careless visual inspection may not detect the defect. Gentle probing using a sterile and fine instrument with blunt tip is advocated if the presence of an oroantral fistula is suspected.

Radiographs assist in diagnosis and are useful to assess the size of defect, whilst it is difficult to determine if the oroantral tract actually is present using only a radiograph.

Management of oroantral communication

Although the antral mucosa is less tolerant of bacterial contamination, the sinus mucous membrane does have a local defense system. Therefore sinus exposure

or perforation does not always lead to the onset of sinusitis. Nevertheless, adequate treatment is necessary to reduce the risk of sinusitis if an oroantral communication is present.

The prime purposes of management are the closure of the defect and prevention of the sinusitis involving oral contaminants. Strategies for specific cases are described below. The patient should be carefully monitored and should be assessed often if the oroantral communication or fistula has closed, and assessed for any indication of maxillary sinusitis presence until healing of the defect is evident.

Oroantral communication created during endodontic therapy

Overfilling around the root apex of the maxillary teeth during routine endodontic treatment may cause a communication between the mouth and sinus via the root canal. If the root canal is not infected and is filled properly, the risk of sinusitis is considered minimal with no requirement of additional prophylactic procedures. However, if the root canal is infected, use of a short-term prophylactic antibiotic is advocated. Special care should be taken not to cause leakage of infected materials and any irrigation solution into the antral cavity through the root canal during endodontic therapy.

In the case of acute odontogenic sinusitis, the root canal of a causative tooth is available for drainage. If the tooth is salvaged, filling of the root canal should be performed after elimination of infection in the sinus.

Oroantral communication created during tooth extraction

Despite the performance of an adequate surgical technique, oroantral communication often occurs as a result of extraction of maxillary teeth. The treatment plan for avoiding sinus exposure or perforation should be made beforehand with evaluation of radiographs. Periapical and panoramic radiographs are available to assess if the patient has an excessively pneumatized sinus or widely divergent or dilacerated roots, which are risk factors of oral communication after the surgery.

If an oroantral communication is created, immediate and spontaneous closure of the opening by normal blood clot formation and routine mucosal healing usually occurs. Oroantral communication may not heal spontaneously if the osseous defect is large. It has been reported that if the diameter of the opening is greater than 5 mm, spontaneous healing may not occur and surgical intervention is required.^{136–138}

A less invasive treatment should be considered initially. Sutures to hold the wound edges closed are of value as an initial attempt, reserving closure with the flap technique, if the opening is not large. A relaxing incision in the gingiva around the opening is often

helpful to hold the wound edges closed and to reduce mucosal tension resulting from sutures. Additional application of a temporary appliance (such as a periodontal pack) to cover the defect is also useful to prevent the entry of food into the sinus, but it must not be inserted into the defect. Prescription of an antibiotic for prevention of sinusitis is recommended.

In contrast, immediate surgical closure with the flap technique may be necessary if the defect is sufficiently large that the surgeon feels that it may not heal by suture or other less invasive techniques.

The presence of sinusitis prevents healing of oroantral communication and fistulae. Therefore, it is imperative to eliminate antral infection before surgical closure. Conservative treatments include frequent irrigation of the fistula and use of antibiotics and decongestants. Surgical closure is available together with the Caldwell–Luc procedure or endoscopic sinus surgery.

Oroantral communication created during dentoalveolar surgery

If a small antral perforation is made accidentally or iatrogenically during a surgical procedure (e.g. removal of a cyst in the maxilla, excision of a tumor from the maxillary alveolar process, sequestrectomy, apicectomy of maxillary teeth or drilling in implant surgery), then foreign body retrieval, antral irrigation with sterile saline, and immediate primary wound closure are all necessary. Flaps should be closed and sutured tightly without excessive tension on the flaps and surrounding tissues. A drain must not be used because it can be a source of saliva contamination. In cases where the defect cannot be covered without tension using the original flap, surgical closure of the defect using a buccal or palatal flap should be considered. If the defect is extremely large and cannot be covered with these flaps, graft operation or use of a prosthesis is indicated. Prescription of an antibiotic following the operation is recommended.

Management of oroantral fistulae

As described previously, an oroantral fistula usually persists for a long period and does not close spontaneously. Consequently, surgical closure is a necessity regardless of defect size. Also, a long-standing oroantral communication that has been present for 4 weeks or longer is unlikely to heal spontaneously and needs a similar treatment to that provided for an oroantral fistula.¹³⁶

Unlike a fresh oroantral communication, the osseous defect surrounding the fistula is usually larger than the clinically apparent, soft tissue deformity. Moreover, because polypoid mucosa and epithelialized, infected or degenerated tissues in and around the fistula must be removed, the soft tissue defect may become larger. Therefore, it may be difficult to

close the defect by suture alone and surgical closure with the flap technique is then a necessity. The most common techniques are the buccal and palatal flap methods.

Closure should not be undertaken if there are foreign bodies lodged in the alveolus or the floor of the sinus. Removal of foreign bodies and ensuring a disease-free sinus are necessary prior to surgical closure.

In the case of an unsuccessful closure of an oroantral fistula by multiple surgical procedures or where there is a long-term persistent fistula, significant inflammatory changes in the sinus mucous membrane may result. In these cases, surgical closure and radical operation for the sinusitis should be performed concomitantly.

Surgical closure with the flap technique

Surgical closure of oroantral communications with the flap technique is performed either immediately after the opening is created or later, as in the case of a long-standing fistula or failure of an attempted primary closure. Although many surgical techniques have been proposed, traditional techniques using buccal advancement and palatal flaps are still widely used. The factors that should be considered for choice of each technique include whether the defect is a new oroantral communication or fistula, the location and size of the defect, the anatomic relationship between the defect and the neighboring teeth, the height of the alveolar ridge, duration of sinus exposure, the pres-

ence or absence of infection in the sinus, and the general health status of the patient.

Any surgical technique requires adequate flap design. The flap should have a broad base and margins that will be positioned over bone but not directly over the defect area of the communication, and it should not be under tension when in position.

Buccal advancement flap technique

The buccal advancement flap technique is a simple, easy and relatively non-invasive procedure (Fig. 29.78). However, it does require careful manipulation and flap design, because the flap is thin and has limited mobility. The technique is used primarily for closure of a minor alveolar communication and it is not the preferred method for large communications or recurrent fistulae. Inclusion of the buccal pad of fat increases the use of this technique.

The simplicity, ease of technique, and resulting low intensity of postoperative pain and discomfort are the major advantages of the method. It is particularly suitable in cases where the defect appears buccally in the depth of the vestibule and where the buccal flap has already been raised. However, this buccal flap technique has the following disadvantages. Firstly, the possible extent of coverage by flap is limited due to a narrow donor site and limitation in flap mobility. This technique is unsuitable for closure of a large defect. Secondly, because the flap is thin, wound dehiscence might occur after the operation. Thirdly, this technique may result in a loss of vestibular depth, which can interfere with prosthodontic

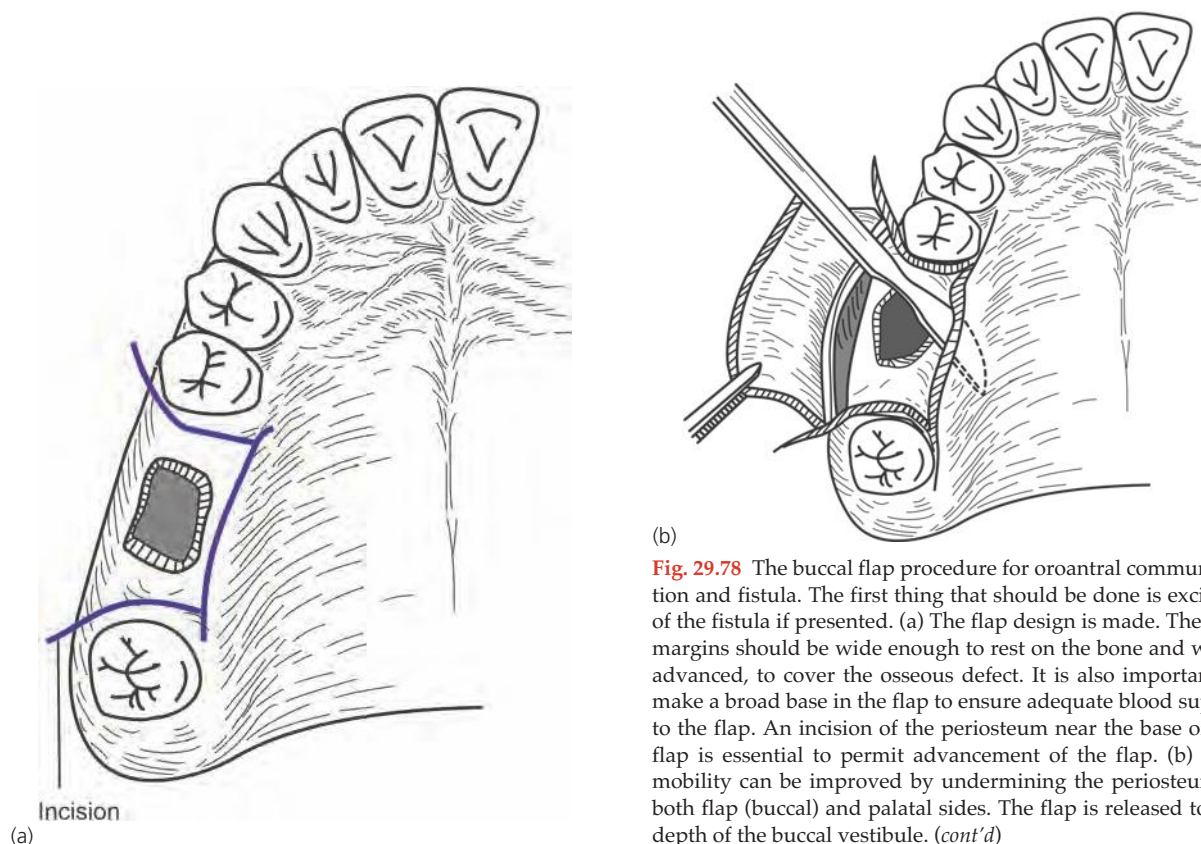


Fig. 29.78 The buccal flap procedure for oroantral communication and fistula. The first thing that should be done is excision of the fistula if presented. (a) The flap design is made. The flap margins should be wide enough to rest on the bone and when advanced, to cover the osseous defect. It is also important to make a broad base in the flap to ensure adequate blood supply to the flap. An incision of the periosteum near the base of the flap is essential to permit advancement of the flap. (b) Flap mobility can be improved by undermining the periosteum at both flap (buccal) and palatal sides. The flap is released to the depth of the buccal vestibule. (cont'd)

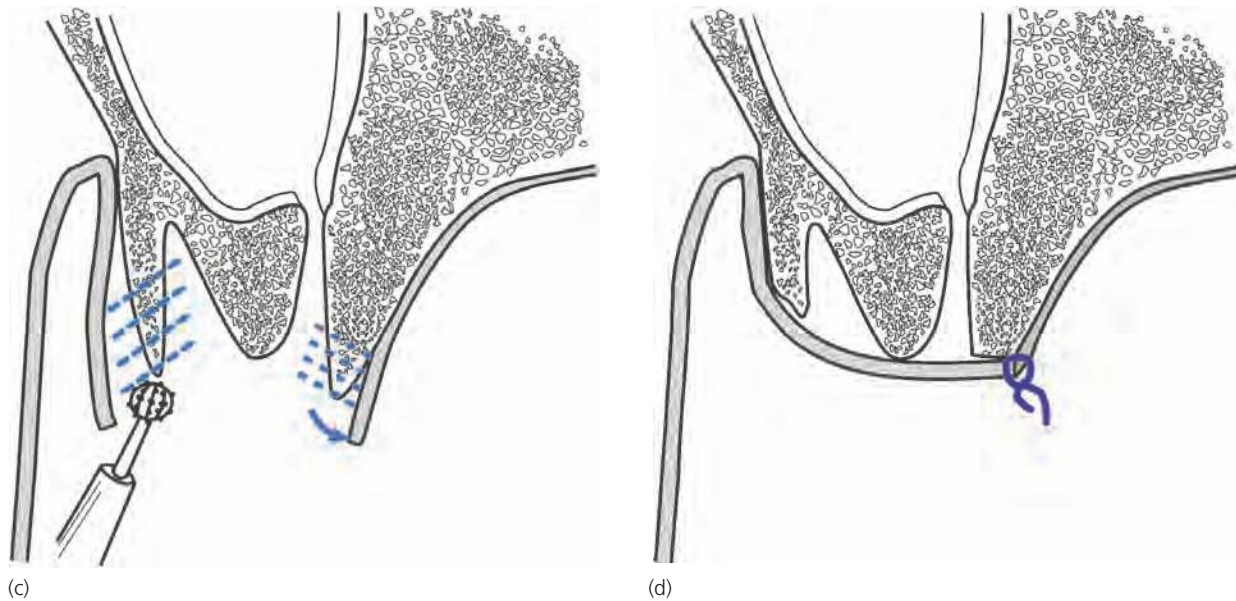


Fig. 29.78 (cont'd) (c) If necessary, the top buccal and palatal margins of the alveolar bone can be arranged to reduce the flap tension. (d) The buccal flap is pulled over the alveolar defect and fastened to the palatal gingiva by sutures. Interrupted sutures may be used to close the two buccal incisions to hold the flap.

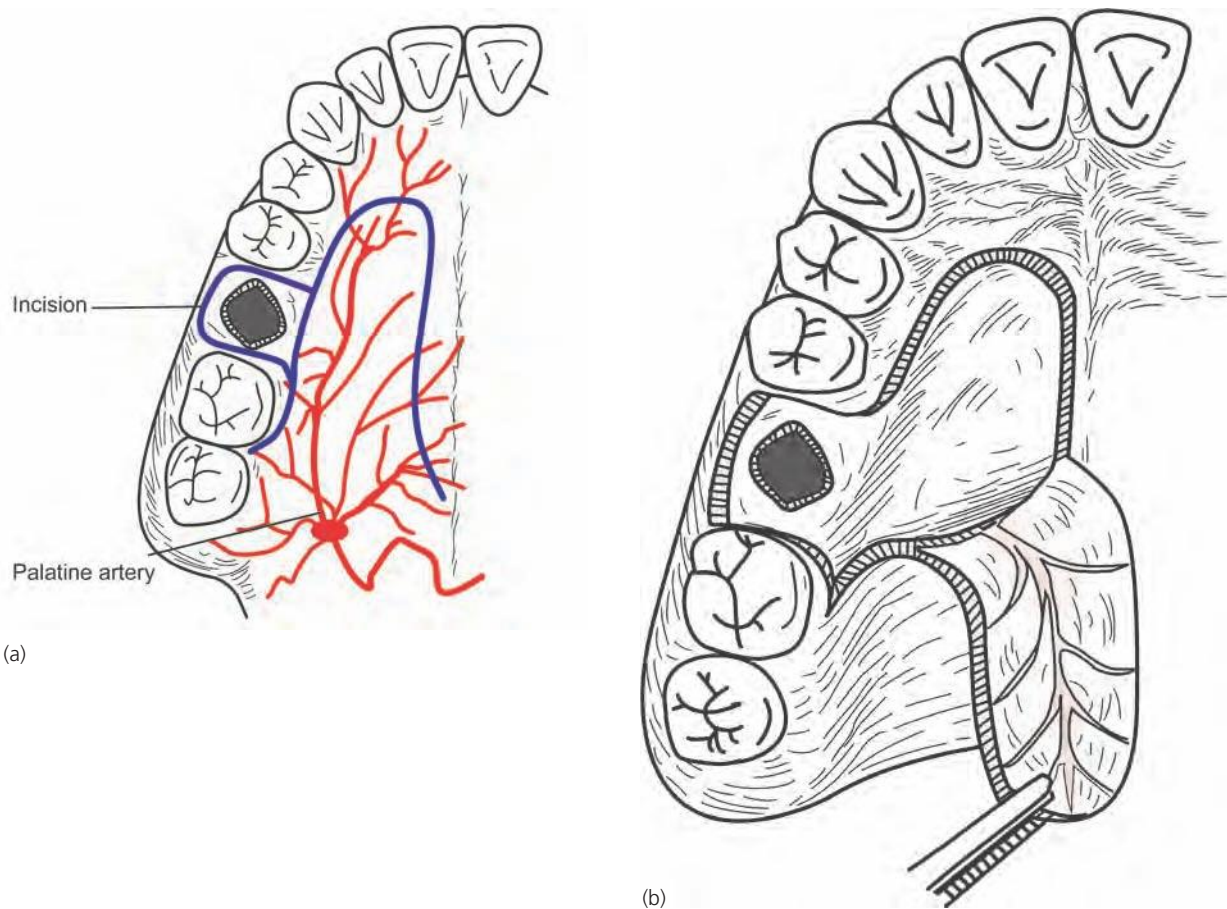
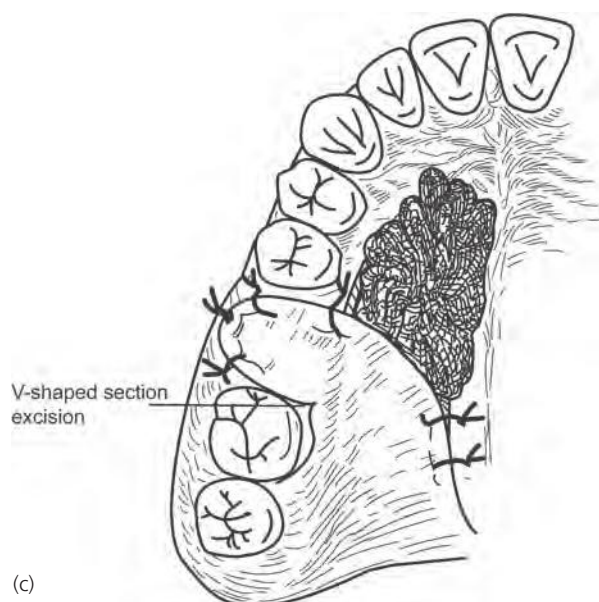


Fig. 29.79 The palatal flap procedure for oroantral communication and fistula. The clinician must know the anatomy of the greater palatine vessels beforehand. The fistula should first be excised if presented. In the case of the fistula, as the entire tract may be epithelialized, a funnel-shaped tissue section is removed down to the alveolar bone. (a) Following this procedure, the flap is then designed. As the palatal mucosa is not very elastic, a meticulous flap design is required. If the flap is adequately designed, it should extend forward sufficiently on the palate to allow flap rotation to cover the defect. Great attention during flap construction is required to prevent injury of palatine vessels and resultant hemorrhage. (b) The flap tissue is detached from the palate with the periosteum and it should contain blood vessels. A small V-shaped excision can be made at the area of the greatest bend of the flap to prevent folding and wrinkling at the base and also to allow easier rotation of the flap. The gingiva on the buccal side of the alveolar process is undermined with a periosteal elevator to facilitate the penetration of the entire thickness of the mucosa by the suture. (cont'd)



rehabilitation and maintenance of oral hygiene. Finally, subsequent postoperative scarring may cause impaired mobility of the oral mucosa around the flap donor site.

The palatal flap technique

The palatal flap harbors the greater palatine vessels that provide a rich blood supply to the flap (Fig. 29.79). This technique involves mobilization and rotation of large mucosal flaps to cover the osseous defect with soft tissues, the margins of which are sutured over.

The advantages of this method over the buccal flap method include: (1) provision of more tissue attachment without tension, which promotes healing and resists the tendency of the flap to return to its original position; (2) use of a flap that is firmer and more resistant to trauma and infection; (3) a broad spectrum of indication, including cases with large bone defects; and (4) preservation of the maxillary vestibular sulcus depth. In contrast, this method has several disadvantages including: (1) denudation of the palatal surface; (2) probably greater postoperative pain; (3) it is a relatively complicated technique that may require extensive clinical experience of the surgeon; (4) the later appearance of roughness and deepening of the donor area, resulting from secondary epithelialization; (5) possible flap necrosis; and (6) interference with wearing of a partial denture that covers the hard palate. This technique is rarely used now.

Postoperative care and possible major complications

The strict cooperation of the patient during the postoperative period is a necessity. Clinicians should ask the patient to avoid any action that causes suction in the mouth (e.g. the use of a drinking tube, drawing on a cigarette or pipe), or produces pressure in the nose, such as vigorously blowing the nose or sneezing.

Fig. 29.79 (cont'd) (c) The flap is rotated and then advanced to attach to the gingival edge of the buccal side. A properly designed flap generously covers the entire gingival excision wound, which should be larger than the perforation of the bone. The flap is then sutured to the gingiva of the buccal side. The donor site forms a deep wound into which a piece of iodoform gauze is tightly packed, to prevent the flap from returning to its natural location and to relieve the tension on the suture. Alternatively, the palatal split (that should be made in advance) is adapted.

In the case of the palatal flap procedure, the dressing should be left in place for 3–5 days. There should be frequent evaluation of the patient's condition, wound, flap, and antrum; adequate treatment is required until the evidence of healing is seen. If the flap breaks down, it is often due to a poor blood supply arising from improper flap design, excessive suture tension or infection.

Consideration for antibiotic prophylaxis and its regimen

The maxillary sinus can be contaminated with the oral microflora that may invade the antrum through the oroantral communication or fistula. Moreover, the flap may be sensitive to the infection because the blood supply to the flap is not very rich. Therefore, the use of systemic antibiotics is recommended to reduce the risk of infection. The pathogens involved in possible infection tend to be viridans streptococci and anaerobes. Amoxicillin (250 mg 8-hourly) or erythromycin (250 mg 8-hourly) prescribed for 1–5 days is therefore recommended.

Peritonsillar abscess

A peritonsillar abscess is an abscess formed in the peritonsillar space that lies between the tonsil and the pharynx. It is most common in young adults (persons 20–40 years of age), but is rare in children.

Etiology and pathogenesis

Peritonsillar abscess usually results from a complication of acute tonsillitis that has been untreated or partially treated. Other causes include mucosal injury to the tonsil and extension of infection in the third molar.

A collection of pus is usually seen 4–7 days after onset of infection. If the superior constrictor muscle is penetrated, an infection of the lateral pharyngeal space may result, raising the risk of serious complications (Fig. 29.80). In severe cases or bilateral abscesses, the oral airway may be reduced. Although peritonsillar abscesses generally respond well to treatment, approximately 10–15% of patients have recurrence after resolution of the original abscess.¹³⁹

Microbiology

Most abscesses show a polymicrobial profile, consisting of both aerobic and anaerobic organisms. The

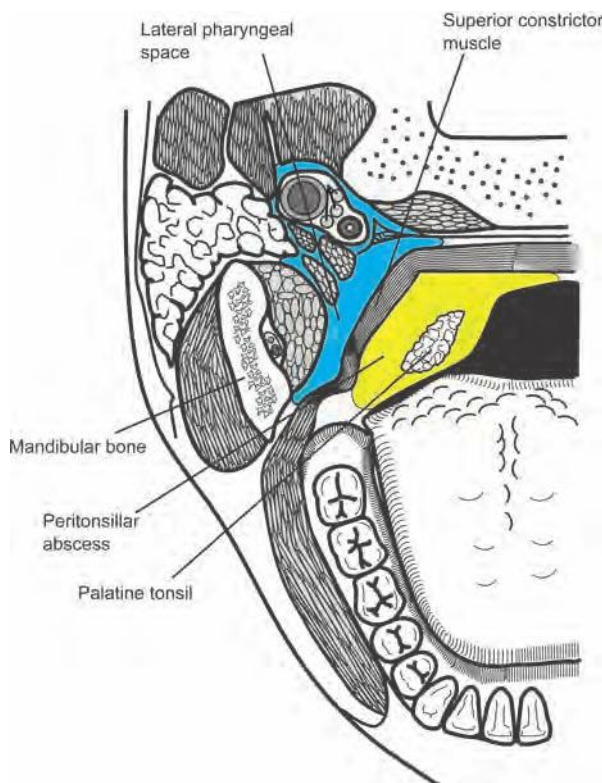


Fig. 29.80 Peritonsillar abscess is located in close proximity to the lateral pharyngeal space and separated only by the superior constrictor muscle. Therefore infection can extend to the lateral pharyngeal space.

most prevalent bacterial isolates from peritonsillar abscesses include *Streptococcus pyogenes*, *Staphylococcus aureus*, *Haemophilus influenza*, and species of strict anaerobic bacteria (e.g. *Fusobacterium*, *Prevotella*, and *Peptostreptococcus*).⁹⁴

Clinical features

The lesion is commonly seen unilaterally with edema and erythema of the tonsils (Fig. 29.81) and causes extreme soreness of the throat, odynophagia, persistent pain in the peritonsillar area, often radiating to the ear and neck, otalgia, and trismus resulting from the pterygoid muscles. The uvula may be displaced towards the unaffected side. Drooling and salivation, and trouble handling oral secretions may result. Neck pain associated with tenderness or when rotating the neck, swollen lymph nodes, and breath malodor are also common. Infection often affects the patient's speech resulting in so-called "muffled voice" or "hot potato voice" (sounds like the patient is talking with hot food in the mouth).

As the infection progresses, a range of systemic symptoms including fever, malaise, and headache may appear. As with other acute infections, increased leukocyte count and CRP level are evident on blood examination.

Diagnosis

Diagnosis is relatively easily made through the patient's medical history and a physical examination. The use of a CT scan and ultrasonography is useful for a determinative diagnosis and in identifying abscess formation (Fig. 29.81). The collection of pus from the abscess through needle aspiration is often considered to be the gold standard for diagnosis of peritonsillar abscess.

Differential diagnosis with odontogenic infections, especially pericoronitis, salivary gland infection, peritonsillar cellulites, lateral pharyngeal infection, and neoplasms (including lymphoma) is required. A thorough intraoral inspection and the patient's



Fig. 29.81 Edema and erythema of the tonsils and peritonsillar area. A CT scan (right) demonstrates the formation of an abscess.

medical history are useful to rule out infection of the salivary glands and odontogenic infection. In contrast with peritonsillar abscess, lateral pharyngeal infection may also induce swelling in the lateral aspect of the neck.

Treatment

Treatment of peritonsillar abscess requires both administration of antibiotics and surgical drainage. Incision and drainage are the essential aspects of treatment. As vital structures are located around a peritonsillar abscess, care should be taken during needle aspiration and incision.

The choice of antibiotics should depend on the result of microbiologic examination of the clinical specimen obtained by needle aspiration. Penicillin has been the traditional antibiotic of empiric choice. However, due to an increased prevalence of penicillin-resistant bacteria, mono-antibiotic therapy with penicillin may be inadequate. If microbiologic data is unavailable, a regimen consisting of oral penicillin and metronidazole is recommended.¹⁴⁰ Alternatively, oral clindamycin, second- or third-generation cephalosporins or quinolones may be effective. In severe cases or in immunocompromised patients, intravenous administration of either or both of imipenem and clindamycin may be advocated. For prevention of recurrence, an additional course of oral antibiotic should be taken for 7–10 days after the infection has clinically resolved.

Suppurative arthritis of the temporomandibular joint

The occurrence of suppurative (septic) arthritis of the temporomandibular joint (TMJ) is rare and usually associated with depression of host defense mechanisms.

Suppurative arthritis takes either an acute or chronic form. Acute infection has the potential to extend extensively, especially to the temporal space, often resulting in serious complications. Aggressive or long-standing infections cause significant destruction of the components of the TMJ, which is associated with functional sequelae including disturbances of jaw growth, fibrosis, and ankylosis of the joint; the latter is the most common complication. Once the infection becomes chronic, the disease may become long-standing and unresponsive to treatment. Therefore, early diagnosis and timely treatment are a necessity.

Etiology, pathogenesis, and microbiology

Suppurative arthritis of the TMJ may result from direct extension of an adjacent infection such as osteomyelitis of the jaw, tympanitis, traumatic or sur-

gical wound infection of the TMJ or hematogenous dissemination of bacteria from a distal site secondary to a systemic process. Staphylococci and streptococci are predominant isolates.^{141,142}

Clinical signs and symptoms

Suppurative arthritis of the TMJ usually manifests unilaterally. Common clinical signs of acute arthritis are preauricular edema with heat and pain, trismus, malocclusion, and contralateral mandible deviation resulting from the increased joint fluid. Patients may present with regional lymphadenopathy, malaise, and fever. In the chronic form, swelling and pain may be minimal but trismus and malocclusion are evident.

Diagnosis

Diagnosis must be made rapidly to prevent permanent joint damage. Diagnosis is made based on the patient's medical history, local signs and symptoms, any adjacent infection, laboratory testing, and imaging. Aspiration from the joint is helpful to confirm the diagnosis and to identify the causative organism. Imaging studies (plain X-ray, CT scan or MRI) may show joint space widening and anterior/inferior displacement of the condylar head (Fig. 29.82). Bone destruction is not seen in the early stages but may appear in the case of longer-standing infections. Arthroscopy allows direct visualization of the joint cavity.¹⁴¹ Arthroscopic features include a reddened and swollen synovial membrane that can sometimes appear necrotic, exhibit bleeding, and have a proliferation of synovial villi, and a degenerative-appearing periosteum exposing the bone of the glenoid fossa.

An elevated leukocyte count and increased CRP levels may be evident in the acute phase of the infection.

Differential diagnosis is required from TMJ disorder, fractures, acute parotitis, osteomyelitis, rheumatologic disease, condylar hypertrophy, and neoplasm.

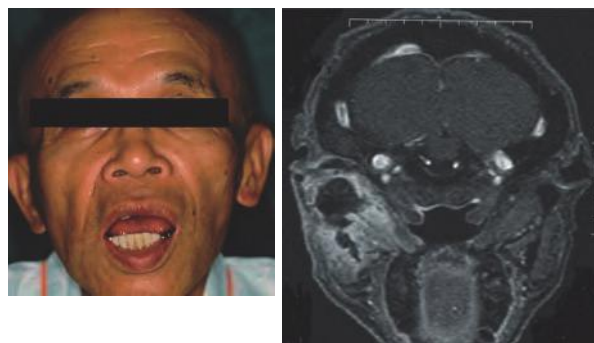


Fig. 29.82 Patient with chronic suppurative arthritis of the temporomandibular joint. Trismus and contralateral mandible deviation are seen. MRI demonstrates an inflammation of the temporomandibular joint and surrounding tissues.

Treatment

Treatment includes antibiotic therapy, aspiration and drainage of pus and infected synovial fluid by incision, proper hydration, pain control, and restriction of jaw movement. Although parenteral penicillin G is adequate as a drug of empiric choice, selection of an antibiotic should be made on the basis of culture and sensitivity testing. Prolonged administration of an antibiotic is recommended to prevent recurrence. Once infection has been controlled, physical therapy should start to improve the mandibular range of motion and deviation on opening movement. Early rehabilitation exercises are also necessary to avoid fibrosis and ankylosis of TMJ.

Long-standing infections with significant pathogenic changes and destruction of the joint may necessitate condylar surgery.

Actinomycosis, tuberculosis, and syphilis

Actinomycosis, tuberculosis, and syphilis are uncommon and present unique lesions that are characterized by chronic granulomatous inflammation. The etiology, pathogenesis, and microbiology are different between the conditions.

Actinomycosis

Actinomycosis in the maxillofacial region is uncommon. It is characterized by a chronic granulomatous process with formation of one or multiple abscesses and indurated swelling of the soft tissue (Fig. 29.83). On rare occasions, the infection localizes in the bone, causing actinomycotic osteomyelitis, although this is not included in the remit of this section. Although actinomycosis may occur at any age, it most commonly occurs between 20 and 60 years of age and cases in men are the most prevalent.¹⁴³

Actinomycosis is distinct from other types of orofacial infections. Odontogenic infections and other orofacial bacterial infection sometimes yield



Fig. 29.83 Patient with mandibular actinomycosis. Indurated swelling with fistulae on the skin is evident.

Actinomyces species, but these are distinct from actinomycosis.

Aetiology and pathogenesis

Since the virulence of *Actinomyces* species is low, the exact etiology and pathogenesis of actinomycosis remains unclear. However, poor dental hygiene, occurrence of surgical procedures within the mouth, oral trauma, odontogenic infection, chronic mastoiditis, chronic otitis, and chronic tonsillitis are associated with its occurrence.^{143–145} These factors may play a role in the invasion of *Actinomyces* into deeper tissues by a break in the mucosal barriers and thus causing an enhancement of bacterial pathogenicity.

Microbiology

Actinomyces species are pleomorphic non-spore-forming and non-acid-fast anaerobic Gram-positive bacilli.⁹ *Actinomyces* species are members of the commensal flora of the mouth. Actinomycosis is primarily caused by *Actinomyces israelii* but *Actinomyces naeslundii*, *Actinomyces viscosus*, *Actinomyces gerencseriae*, *Actinomyces graevenitzii*, and *Actinomyces meyeri* can also be involved.^{143–145} The infection is usually polymicrobial with other oral commensal bacteria, especially anaerobic Gram-negative bacilli, often isolated from the infection (Fig. 29.84).

Clinical features

Actinomycosis commonly occurs in the soft tissue of the mandible, especially around the third molar and

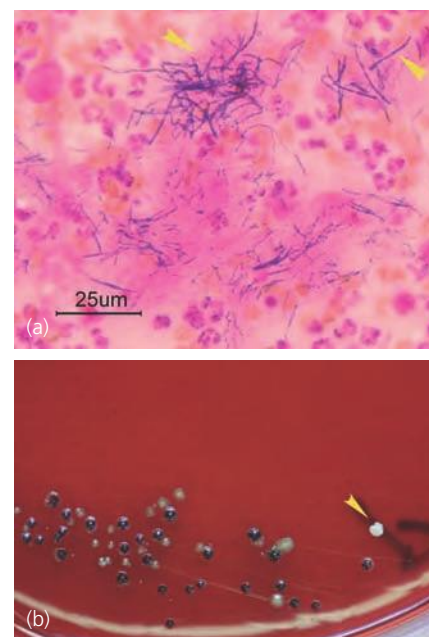


Fig. 29.84 Microscopic view (a) of pus obtained from a patient with mandibular actinomycosis and (b) the isolated bacterial colonies on an agar plate. The arrows indicate cells (a) and colonies (b) of *Actinomyces israelii*. Photographs courtesy of Dr A. Kanekawa (Yamaguchi Grand Medical Center).

the mandibular angle. It may present in either an acute or chronic form, with the latter being the most common.

Initial clinical signs and symptoms of chronic actinomycosis are similar to those of common suppurative infection. As the swelling increases, enlarging tissues become indurated, which can manifest as tumor-like masses. There may be no fever associated with this inflammatory mass. The surface of the skin overlying the jaw becomes rough and has a reddish-brown discoloration. In the case of actinomycosis in the posterior mandible, the infection is likely to affect the masticatores and cause a resultant trismus. Unlike other infections, actinomycosis does not follow the usual anatomic planes and tends to erode and to burrow through them. Infection occasionally spreads to involve the bone. The lesions of actinomycosis enlarge slowly with minimal elevation of temperature and little pain. Over time, abscesses form within firm swellings with the development of multiple fistulae on the skin. The amount of pus or exudate from each abscess tends to be small and any exudate often contains sulfur granules (Fig. 29.85), which are collections of microcolonies of the organisms. The fistulae may persist for long periods or close spontaneously, but even when they close, new tracts may quickly develop. Chronic actinomycosis may persist or recur over weeks, months or even years. A pattern of remission and exacerbation may characterize the disease. Patients often have a history of multiple episodes of recurrent infection, which initially respond well to antibiotic therapy but recur after antibiotic therapy stops.

There is no characteristic marker of actinomycosis in blood specimens and the leukocyte count is often normal or only minimally elevated.

The acute form of the infection causes a painful and fluctuant swelling that resembles or mimics acute odontogenic infection, with a range of elevated body temperature.

Diagnosis

Diagnosis of actinomycosis, and especially differentiating it from pyogenic infection, may be difficult. Therefore, diagnosis is usually made based on the consideration of all factors, including clinical signs and symptoms, the patient's medical history, the presence of sulfur granules, and microbiologic findings.

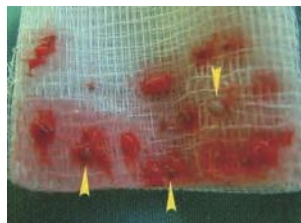


Fig. 29.85 Sulfur granules originating from a pus specimen. Photograph courtesy of Dr A. Kanekawa (Yamaguchi Grand Medical Center).

The detection of sulfur granules either macroscopically or microscopically is a characteristic finding but it is not usual. If a patient exhibits indurated swelling with multiple fistulae and trismus (if disease affects the masticatores) and a history of recurrent or late infection after tooth extraction, actinomycosis should be considered. The microbiologic examination of clinical specimens provides significant information for diagnosis. It is important to note that the growth of *Actinomyces* colonies on culture media is slower than that of other anaerobic bacteria. Specimens should be incubated in an anaerobic environment for at least 10 days to obtain adequate results. Although isolation of *Actinomyces* from specimens can be definitive evidence for the diagnosis of disease, it should be remembered that *Actinomyces* culture-negative samples frequently occur in patients with actinomycosis. Conversely, *Actinomyces* isolation may result from contamination of the specimen with oral commensal bacteria. The results of microbial examination should therefore be interpreted appropriately and with caution.

Radiographs are non-specific for actinomycosis even in the presence of soft tissue swelling and fistula. CT scans may reveal loculations and dense fibrosis.

Differential diagnosis between actinomycosis and suppurative infection, orofacial tuberculosis, and neoplasms is essential.

Treatment

Actinomyces species are highly susceptible *in vitro* to various types of antibiotics including β -lactam antibiotics, macrolides, clindamycin, and tetracycline. However, highly fibrous tissues around lesions can prevent penetration of antibiotics to the infected area. Therefore use of higher doses of antibiotics and surgical intervention is necessary.

Surgical intervention includes frequent incision, drainage, and excision of all fistulae. The prime purpose of these procedures is to ensure the delivery of adequate amounts of antibiotic to the infected area and to reduce the levels of involved organisms by exposing them to air (oxygen).

Initial antibiotic therapy involves high-dose intravenous infusion of penicillin G (10–20 million U per day) for 3–14 days. The use of a prolonged course of antibiotic is required because the infection often recurs after short-term antibiotic use. After the initial course of antibiotic, additional oral antibiotic is administered for at least 3 months.

Other effective antibiotics include erythromycin, cephalosporins, tetracycline, clindamycin, and imipenem, but metronidazole and aminoglycosides are ineffective.

Tuberculosis

Tuberculosis is a serious infectious disease caused by mycobacteria, mainly *Mycobacterium tuberculosis*.⁹

Although tuberculosis most commonly attacks the lungs, it can also affect other organs including the central nervous system, the lymphatic system, the circulatory system, the genitourinary system, bones, joints, and the skin. The oral mucosa, skin of face, and lymph nodes are included as possible sites of tuberculosis lesions, and these usually manifest as secondary lesions of the disease.

Mycobacterium tuberculosis withstands certain disinfectants and survives in a dry state for weeks. Limited types of antibiotics are effective against this organism. An increased prevalence of multidrug-resistant strains of *M. tuberculosis* is now a serious public health issue in many countries, especially in developing countries.

Etiology, pathogenesis, and microbiology

Tuberculosis is one of the most prevalent infectious diseases in the world, particularly in developing countries. Infection is spread via droplets of sputum containing the acid-fast bacillus, *M. tuberculosis*, from patients with active pulmonary tuberculosis.

Clinical features in oral and maxillofacial regions

The classical intraoral presentation is an ulcer on the dorsal surface of the tongue but lesions may affect any site (Fig. 29.86). The ulcers are irregular with raised borders and often resemble deep fungal infection or squamous cell carcinoma. Tuberculosis can appear in the skin on the face around the nose and ears as lupus vulgaris, which is characterized by painful reddish-brown nodules that tend to ulcerate and form scars.

Tuberculosis can attack cervical lymph nodes and painless swelling of lymph nodes may result. In the more advanced cases, fistulae associated with the lymph node lesions may appear. Some cases present as asymptomatic calcification in the cervical lymph node (Fig. 29.87), and may be discovered by a routine panoramic survey.



Fig. 29.86 Oral tuberculosis exhibiting an ulcer on the dorsal surface of the tongue.

Diagnosis

Mucosal biopsy should be undertaken to demonstrate characteristic granulomatous inflammation with well formed granulomata, Langerhans giant cells, and necrosis. Ziehl-Neelsen or Fite stains may be used to detect the tubercle bacilli. Microbiologic culture of suspected clinical material may also be useful in the diagnosis of tuberculosis, although specialized media and prolonged incubation are required for recovery of the organism, and it often gives false-negative results. Molecular microbiologic methods are also available to detect the organism and aid diagnosis.

Treatment

Localized treatment is not required because oral lesions resolve when systemic chemotherapy with rifampin, isoniazid or ethambutol is provided. Typically, combinations of these drugs are used. Instead of the short course of antibiotics normally used to cure other bacterial infections, tuberculosis requires much longer periods of treatment (around 6–9 months) to entirely eliminate mycobacteria from the body.

Syphilis

Syphilis is a curable sexually transmitted disease caused by the spirochete, *Treponema pallidum*. The disease primarily affects the genitals, but can induce lesions in the skin, mucous membranes, and many other parts of the body including the brain and heart. The initial discovery of penicillin resulted in a dramatic decline in the incidence of the disease, but the incidence of syphilis has recently begun to rise along with the incidence of HIV/AIDS. If left untreated, syphilis can lead to serious complications or even death. However, severe cases are now seldom encountered in many countries due to early diagnosis and treatment.

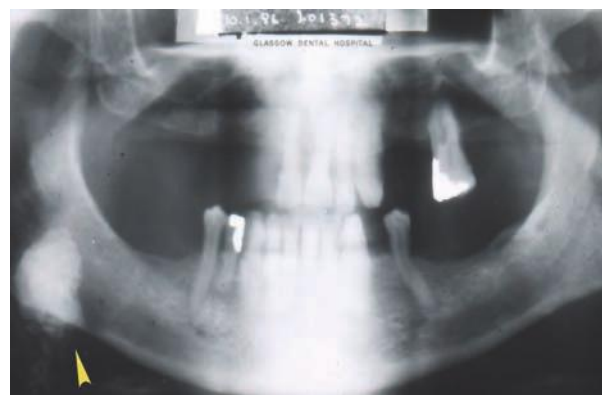


Fig. 29.87 Calcification in the cervical lymph node involved in tuberculosis.

Etiology and pathogenesis

Microbial transmission is usually by sexual contact, although congenital syphilis can occur via transmission from mother to child *in utero*. Iatrogenic transmission between clinician and patient is possible via exposed wounds on fingers and hands.

Clinical features of syphilis in oral and maxillofacial regions

The signs and symptoms of syphilis may occur in four stages consisting of primary, secondary, latent, and tertiary. The primary lesion appears within a few weeks after exposure. The primary lesion is referred to as a chancre and is characterized by the development of a firm nodule at the site of inoculation, which breaks down after a few days to leave a painless ulcer with indurated margins. A single chancre is typical, but there may be multiple sores. Cervical lymph nodes are usually enlarged and rubbery in consistency. The lesion of primary syphilis typically disappears without treatment within 3–12 weeks without scarring, but the underlying disease remains.

The signs and symptoms of secondary syphilis may begin 6–10 weeks after the chancre appears. Secondary syphilis is characterized by a macular or papular rash, febrile illness, malaise, headache, generalized lymphadenopathy, and sore throat. The oral mucosa is involved in approximately one third of patients. Oral ulceration, often described as “snail track ulcer”, develops. Mucous patches or opaline plaques may also appear in the mouth. These signs and symptoms may disappear within a few weeks or repeatedly recur for up to a year. Lesions of secondary syphilis are infective and dentists must take care to prevent cross-infection.

Patients with untreated secondary syphilis can develop the latent form many years after initial infection. In this stage, no symptoms are present, although even in these cases the disease may progress to the tertiary stage.

The tertiary stage begins years after the original infection. In this stage, the pathogen may spread, leading to serious internal organ damage and death. Neurological and cardiovascular problems are significant, with the former causing stroke, meningitis, poor muscle coordination, numbness, paralysis, deafness or visual problems, personality changes, and dementia. Cardiovascular problems include aneurysm and inflammation of the major artery and other blood vessels, and various valvular heart diseases. Two oral lesions are recognized in the tertiary form of syphilis: gumma in the palate and leukoplakia affecting the dorsal surface of the tongue.

A pregnant patient with primary or secondary syphilis may infect the developing fetus, resulting in characteristic congenital abnormalities (congenital syphilis). Infection of the developing vomer produces a nasal deformity known as saddle nose. The feature

of Hutchinson’s triad is a common pattern of presentation for congenital syphilis, and includes interstitial keratitis, deafness, and dental abnormalities involving notched or screwdriver-shaped incisors and mulberry molars.

Diagnosis

Diagnosis of syphilis is supported when dark-field microscopy of smears taken from either a primary or a secondary lesion reveals numerous spirochetes in size and form typical of *T. pallidum*. However, serologic investigation is the most reliable way of syphilis diagnosis from the late stages of primary infection onwards. It is important to note that routine laboratory culture of *T. pallidum* is problematic and can be unreliable.

Treatment

Treponema pallidum has remained sensitive over the years to penicillin, erythromycin, and tetracyclines, and indeed the most effective treatment of any stage of syphilis is intramuscular procaine penicillin. Patients should be monitored for at least 2 years and serologic examinations repeated over this period.

Complications of maxillofacial infections

Although infections at oral and maxillofacial sites are usually localized, they may have systemic effects and/or involve contiguous structures. As a consequence, a variety of complications can occur (Fig. 29.88). Although complications can develop in immunocompetent patients, they are more prevalent in immunocompromised patients. Since these complications are often serious and potentially life-threatening, early recognition and prompt treatment are a necessity. Although the definitive diagnosis and management are usually undertaken by a specialist, dentists must be well aware of the seriousness of complications and recognize their clinical features.

Airway obstruction

A compromised airway is the most common life-threatening complication of maxillofacial infections. It is particularly prevalent when an infection involves the submandibular and cervical spaces. In the complete absence of inspired oxygen due to an airway block, brain damage occurs within 3–5 minutes. If an airway problem is not serious, patients can be managed with expeditious surgical drainage and appropriate use of antibiotics. However, the clinician must be aware that such conditions can rapidly progress within a few hours and at any time. Patients with a risk of a compromised airway should be monitored

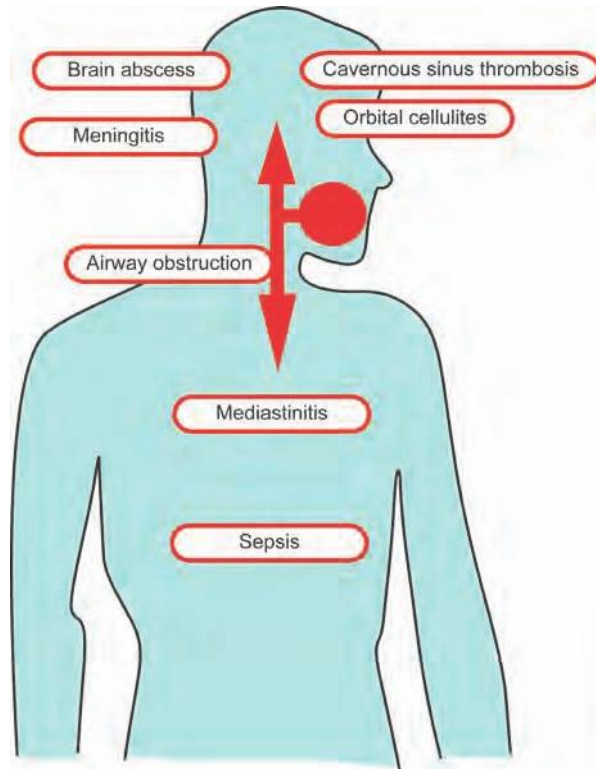


Fig. 29.88 Orofacial infections can cause serious complications. Such complications arise through spread of the infection either by direct extension or via the hematological route from the infection site in the oral and maxillofacial regions.

constantly and early intervention should be undertaken without delay.

The clinician must not overlook any indications of a compromised airway and examples of these are listed in Table 29.29. Signs of cyanosis and altered consciousness result from hypoxia, indicating an airway problem. The elevation of the floor of the mouth, difficulty to move or protrude the tongue, and the presence of trismus, limited ability to extend and flex the neck, dyspnea, dysphagia, dysphonia, tachypnea, and stridor are common manifestations of airway problems. An inability to breathe in the supine position, labored breathing, shortness of breath, and a sensation of a lump in the throat also suggest a possible airway problem. Pain on speaking and a change in the character of a patient's voice are all important indicators in determining an airway problem. The patient may assume a "sniffing" position, extending the neck and chest slightly forward as if smelling a rose. This maneuver straightens the upper airway, decreasing airway resistance and thus is a sign of a compromised airway.

Assessment of oxygen saturation level using pulse oximetry without administration of supplemental oxygen is useful in inferring the status of oxygenation. An oxygen saturation value $>97\%$ generally indicates respiratory sufficiency, whilst a saturation value $<94\%$ or a diminishing trend suggests the need for more urgent airway intervention. It should be remembered that, although administration of supplemental oxygen provides the patient with satisfactory oxygen

Table 29.29 Possible indications of a compromised airway.

Cyanosis and altered consciousness
Elevation of the floor of the mouth
Difficulty to move or protrude the tongue
Limited ability to extend and flex the neck
Labored breathing
Shortness of breath
Inability to breathe in the supine position
Assuming the 'sniffing' position, seen as an extension of the neck and chest slightly forward as if smelling a rose
Inability to sleep in the supine position
Unusual snoring whilst sleeping
Sensation of a lump in the throat
Pain on speaking
Change in the character of a voice
Oxygen saturation of less than 97% when measured by pulse oximetry
Trismus, dyspnea, dysphagia, dysphonia, tachypnea, and stridor

levels, it may mask the actual problem of the compromised airway and may provide a false sense of security.

Radiographic imaging, especially a CT scan, can help determine the extent of infection and any distortion of the airway. Except for the patient in acute or imminent respiratory distress requiring immediate airway management, the CT scan should be an important component of the evaluation and provides information pertinent to airway management.

Sepsis

As the definition of sepsis has been diverse, a standardized terminology is required to eliminate confusion. An American College of Chest Physicians/Society of Critical Care Medicine Consensus Conference published consensus conference definitions concerning sepsis (Table 29.30).¹⁴⁶ Sepsis is termed as the clinical syndrome defined by the presence of both infection and a systemic inflammatory response. It is often confused with bacteremia or systemic inflammatory response syndrome (SIRS). Bacteremia is the presence of bacteria in the bloodstream, regardless of a provoked host inflammatory response. SIRS is a systemic inflammatory response that is triggered not only by infectious but also non-infectious conditions. The interrelationship between sepsis, bacteremia and SIRS is presented in Fig. 29.89.

Sepsis can progress to shock, disseminated intravascular coagulation multiple organ dysfunction syndrome, and eventually death. Sepsis is more common and dangerous in elderly, immunocompromised, and critically ill patients. In adult patients, sepsis is considered to be present if infection is suspected or proven and two or more of the following SIRS criteria are met: (1) temperature $>38^{\circ}\text{C}$ or $<36^{\circ}\text{C}$;

Table 29.30 Definition of sepsis and other associated terms.

Infection
Microbial phenomenon characterized by an inflammatory response to the presence of microorganisms or the invasion of normally sterile host tissue by those organisms
Bacteremia
The presence of viable bacteria in the blood
Systemic inflammatory response syndrome (SIRS)
The systemic inflammatory response to a variety of severe clinical insults
Sepsis
Toxic condition resulting from the systemic inflammatory response to infection

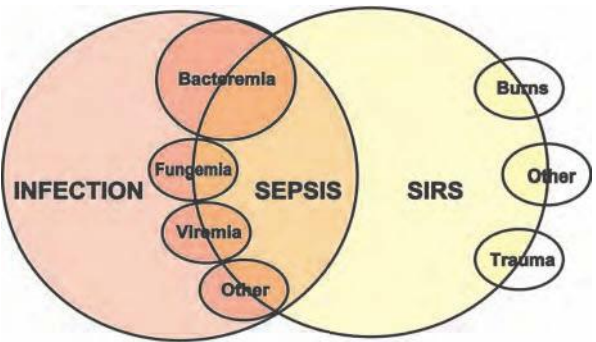


Fig. 29.89 The interrelationship between systemic inflammatory response syndrome (SIRS), sepsis, and infection.

(2) heart rate >90 beats/min; (3) respiratory rate of >20 breaths/min or partial pressure of carbon dioxide (PaCO₂) of <32 mmHg; and (4) leukocyte count of >12 000 cells/μl or <4000 cells/μl, or >10% immature (band) forms (Table 29.31).¹⁴⁶

Blood culture is important in demonstrating the presence of bacteria in the bloodstream and identifying involved microorganisms. However, blood culture often provides false-negative results. Although blood culture positivity supports diagnosis, if the above SIRS criteria are met then sepsis should be diagnosed regardless of results of the microbial examination.

Table 29.31 Diagnostic criteria for sepsis.

Infection is suspected or proven
AND
Two or more of the following criteria are met:
(1) Temperature >38°C or <36°C
(2) Heart rate >90 beats/min
(3) Respiratory rate of >20 breaths/min or partial pressure of carbon dioxide (PaCO ₂) of <32 mmHg
(4) Leukocyte count of >12 000 cells/μl or <4000 cells/μl, or >10% immature (band) forms

Mediastinitis

The mediastinum is the potential space between the pleural cavities. It contains the heart, the great vessels, the trachea and main bronchi, the esophagus, the thymus gland, connective tissues, and lymph nodes. Fascial planes connect the mediastinum with the neck.

Mediastinitis is inflammation of the mediastinum. Mediastinal involvement following a maxillofacial infection is rare, but, if it is involved, the mortality rate reaches 40–60% despite the use of antibiotics and surgical therapy. A late recognition of disease or an improper course of treatment will result in a poor prognosis for patients. Mediastinitis associated with maxillofacial infection generally results from an extension of an infection through the pretracheal fascia, the lateral pharyngeal space, and/or the retropharyngeal–danger spaces (Fig. 29.90).¹⁴⁷

Common signs and symptoms of patients with mediastinitis include fever, chills, a shortness of breath, chest pain, swelling in the neck, dyspnea and dysphagia. A CT scan can indicate the presence of purulence and aid in an early diagnosis (Fig. 29.91). Prompt incision and drainage or debridement and

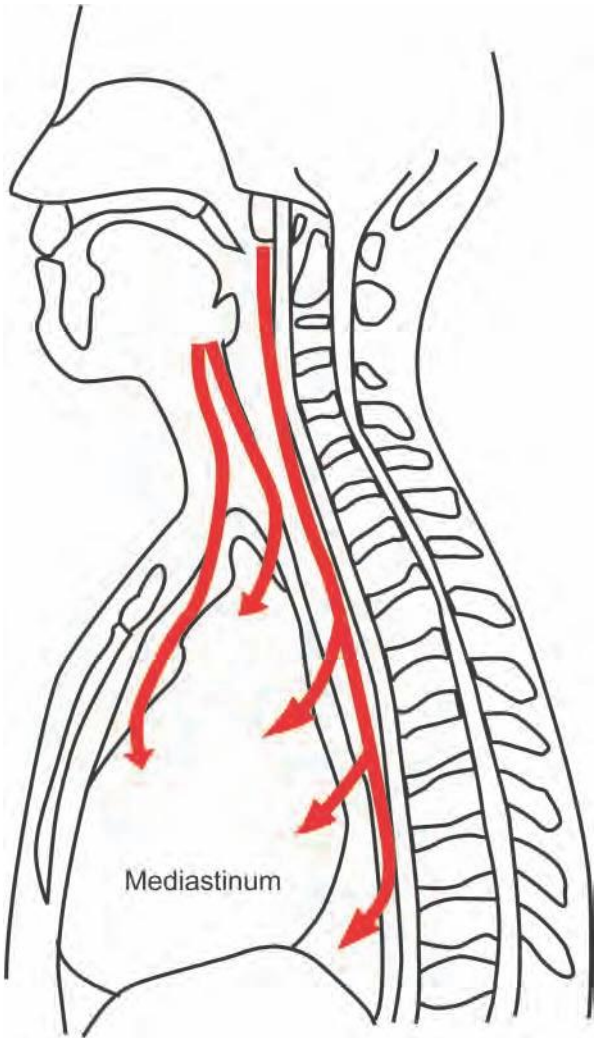


Fig. 29.90 The routes of extension of infection from the oral and maxillofacial region to the mediastinum.

surgical airway management, usually by thoracic surgeons, is required.

Orbital cellulitis

Orbital cellulitis is caused by infection of the orbital soft tissues by extension of infection from periorbital structures (especially the paranasal sinuses), direct bacterial inoculation from accident, trauma or surgery, and hematogenous spread of infection.^{148,149} Odontogenic infections, maxillary osteomyelitis, and dental extractions are also possible origins of orbital cellulitis. The condition can progress rapidly and involve the loss of sight and fatal cerebral complications.

Orbital cellulitis typically begins with a painful and erythematous swelling of the eyelid. Patients may be systemically ill with severe orbital pain, fever, proptosis, conjunctivitis and chemosis, impaired movement of the eye, and signs associated with optic nerve damage (e.g. decreased visual acuity, visual field loss, problems with color vision). A CT scan is the most useful investigation for its diagnosis.

Cavernous sinus thrombosis

The cavernous sinus is a large collection of thin-walled veins creating a cavity bordered by the sphenoid bone and the temporal bone of the skull (Fig. 29.92). It lies within the skull, immediately behind each orbit, and contains the oculomotor nerve (cranial nerve III), the trochlear nerve (cranial nerve IV), the ophthalmic nerve and maxillary nerves that are the branches of the trigeminal nerve (cranial nerve V), the abducens nerve (cranial nerve VI), and the internal carotid artery. The purpose of the cavernous sinus is to drain blood from the brain and face back to the heart.

Cavernous sinus thrombosis is blockage of a large vein within the cavernous sinus by the formation of a blood clot. The usual cause is a bacterial infection that has spread from the paranasal sinuses, ears, eyes, nose, or skin of the face, but an abscess in the maxillofacial region may also be involved in the etiology.¹⁵¹ Contamination of the cavernous sinus due to direct extension or hematogenous dissemination of infection can result in thrombosis. The initial infection

usually involves one side but can easily spread to the opposite side through the circular sinus.

Abrupt onset of unilateral periorbital edema, headache, an inability to move the eye, photophobia, loss of vision, drooping eyelids, lacrimation, proptosis, chemosis, and retinal hemorrhages may all be evident. There may also be cranial nerve involvement, which results in ophthalmoplegia, periorbital sensory loss and impaired corneal reflex, ptosis, and dilatation of the pupil. Severe systemic illness with a high fluctuating fever, chills, headache, rapid pulse, and sweating can all occur.

The diagnosis of cavernous sinus thrombosis is made through imaging studies (sinus radiography, CT scan, and MRI), in addition to the clinical presentation. Cavernous sinus thrombosis can progress rapidly and even today has a high mortality rate. Aggressive medical and surgical treatment are therefore required.

Neurological complications

Despite being very rare, acute suppurative maxillary infection has the potential for brain and nervous system involvement. Common neurological complications include meningitis and brain abscesses. These may be derived from metastatic spread by means of the bloodstream or may develop from close proximity to thrombophlebitis. Signs and symptoms of neurological complications should be looked for and recognized as early as possible because of the risk of fatality.

The meninges are the membranes that cover the brain and spinal cord, and act as an extra barrier to infection. Meningitis is an infection of the meninges, and is the most common neurological complication resulting from infections of the oral and maxillofacial region.¹⁵² Initial signs and symptoms are not specific. Symptoms may include a constant generalized headache, confusion, drowsiness, a high temperature,



Fig. 29.91 Chest CT scan of a patient with severe lateral pharyngeal infection. The CT scan shows the presence of pleural effusion, indicating mediastinitis.

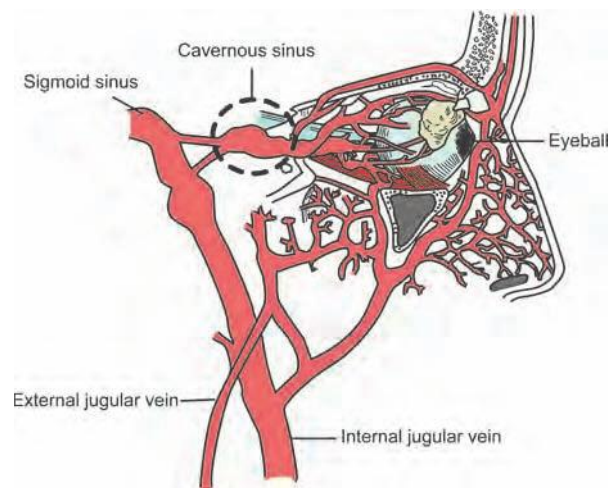


Fig. 29.92 The anatomy of the cavernous sinus. (Illustration was redrawn with permission from Kirikae I, Nomura K, editors, *Modern Otorhinolaryngology*, 10th edition. Tokyo: Nanzando, 2004.¹⁵⁰ Copyright © I. Kirikae and K. Nomura.)

stiffness of the neck, vomiting, stomach pain, rapid breathing, convulsions and coma. Kernig's sign (a strong passive resistance when an attempt is made to fully straighten the patient's leg when the hip is flexed at 90°) and Brudzinski's sign (a spontaneous flexion of both the knees and hips in response to passive flexion of the neck towards the chest) are frequent positive indicators of meningitis. Diagnosis is made on the basis of the symptoms and clinical signs, the culture of microorganisms from the blood, and analysis of cerebrospinal fluid through lumbar puncture.

Bacteremia produced during maxillofacial infections and invasive oral manipulations can instigate brain abscess, although the blood–brain barrier along with the immune response usually excludes bacteria that attempt to gain an entry into the cranial vaults. This event is, therefore, very rare.

Depending on the specific location of an abscess within the brain, systemic responses to infection can lead to an increased intracranial pressure and damage to the focal cerebral tissue. As a result, headache, fever, papilledema, drowsiness, confusion, convulsions, seizures, dysphagia, ataxia, visual-field deficits, hemiparesis (paralysis or weakness affecting one side of the body) or speech difficulties may appear. The diagnosis is usually established through MRI (Fig. 29.93). Single or multiple ring-enhanced lesions in the brain parenchyma are seen with gadolinium MRI. Diffusion-weighted MRI features of a brain abscess have a specific high signal intensity.

Infections of oral and maxillofacial wounds

Wounds can be divided into surgical and traumatic wounds. Surgical wounds occur in optimum conditions with anesthetic and operating theatre support. In contrast, traumatic wounds are generally accidental and arise under non-sterile conditions and, as a result, tend to be associated with much more serious underlying injury with a range of bacterial contamination.

The Centers for Disease Control and Prevention (CDC) has defined surgical site infections as those associated with surgical procedures that occur within 30 days of an operation, or within 1 year of an important procedure.¹⁵³ The CDC currently advocates the term “surgical site infection” instead of “surgical wound infection”. The infection of a surgical incision is distinct from the infection of a traumatic wound. However, infections of surgically treated traumatic wounds can be categorized as surgical site infections. Wounds associated with traumatic injury generally have a higher infection risk than wounds originating from surgical procedures alone.

Surgical site infections may provide not only physical discomfort with a prolonged period of wound healing but also elevated financial costs to the health care provider. Furthermore, as a result of wound infection there may be unacceptable functional and esthetic sequelae. In extreme cases fatality can occur.

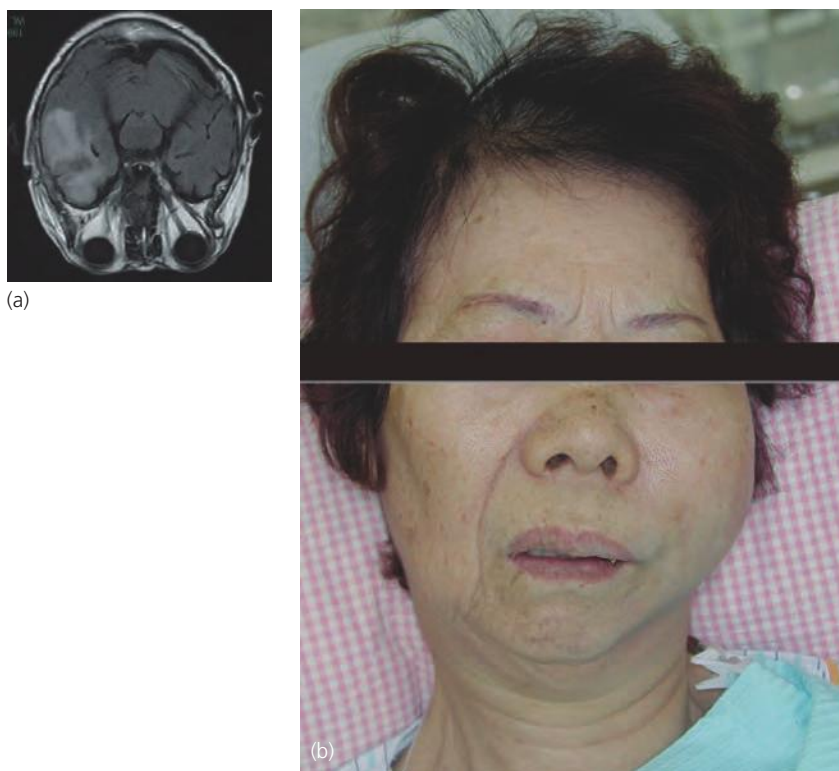


Fig. 29.93 MRI of brain abscess (a) associated with left buccal abscess involved in acute periapical infection (b). Pus accumulation and edema in the right brain are seen. Viridans streptococci, *Prevotella* spp., and *Fusobacterium* spp. were recovered both from brain and buccal abscess. Photographs with permission from Miss T. Tanbo (Kanazawa University).

This section discusses the basic principles of prevention and management of wound infections.

Etiology and pathogenesis

Key requirements for the establishment of a wound infection are the presence of a sufficiently large bacterial inoculum with sufficient virulence, the availability of a suitable environment for bacterial growth, and the impairment of local and systemic host defense mechanisms. There are various factors which predispose to wound infection (Fig. 29.94). These factors can be divided into systemic, local, and technical.

Systemic factors

Immunocompromising factors are generally systemically related. The long-term receipt of corticosteroid therapy, administration of cytotoxic and immunosuppressive drugs, uncontrolled diabetes, alcohol abuse, malnutrition, malignancies, myeloma, and HIV are all examples of predisposing factors that reduce host resistance. Amongst these, diabetes is perhaps the most frequently encountered. The likelihood of infection does indeed correlate with fasted blood sugar level. The glycosylated hemoglobin test or hemoglobin A1c is used together with fasted blood sugar level when assessing the level of diabetes control over the past 1–3 months.

Local factors

The number and type of contaminating microorganisms are determinants for the risk of wound infection. Although surgery in the absence of aseptic methods does undoubtedly increase the risk of wound infection, even a surgical wound created under aseptic conditions can be contaminated. Wounds with a pre-

existing infection are more likely to be infected compared with infection-free wounds, due to the high number of bacteria present. The location of a wound is also important, as the quantity and type of bacteria in the normal microflora can vary between sites thus affecting the nature of initial infection. Perioral and oral tissues are generally resistant to the establishment of infection, whilst the maxillary sinus is not as tolerant of contamination.

Factors that interfere with normal wound healing mechanisms contribute to the risk of wound infection. These factors include the presence of foreign bodies and necrotic tissue, devascularization or ischemia of the wound and the presence of dead spaces.

Foreign bodies may be contaminants that serve as sources of infection. Moreover, they may be recognized as non-self and can induce chronic inflammation, which may provide suitable conditions for the overgrowth of pathogenic bacteria. Examples of foreign bodies include trapped food or debris, fragments of non-viable bone, environmental materials left in the wound, and contaminated implanted medical equipment (e.g. sutures, wire ligatures, metal mesh, and implanted screws or plates).

Necrotic tissue acts as a foreign body and inhibits the normal wound healing mechanism. Due to a lack of adequate blood supply, dead or dying tissue is often infected. In addition, such tissue may represent a source of nutrients for bacterial growth. Tissue devitalized due to chronic inflammation and previous radiotherapy is also susceptible to infection.

In a wound with impaired vascular flow or ischemia, the delivery of oxygen, nutrients, components of immunity, and administered systemically antibiotics may be impeded. Dead spaces and hematomas usually fill with blood and/or exudates. Host defense mechanisms and the effects of antibiotics are all

Systemic factors	Local factors	Technical factors
Depression of host defense mechanisms	• Certain location of wound • Pre-existing infection • Devitalized or necrotic tissue • Tissue with ischemia and devascularization • The presence of foreign bodies, hematomas or dead spaces • Type, cause and mechanism of injury in the case of traumatic wound	• Excessive operating time • Wide extent of surgical area • Procedure without aseptic surgery • Unnecessary tissue damage • Improperly designed flaps and incorrect placement of incision • Excessive external and internal pressure on a wound • Insufficient hemostasis • Inadequate management of a hematoma or dead space • Insufficient tissue debridement • Inadequate suture and retractor that may be involved in tissue ischemia • Instability of fracture fragments • Primary closure of infected wounds • Delayed treatment for traumatic wounds

Fig. 29.94 Factors affecting the likelihood of wound infection.

impaired due to a limited blood supply at such sites. The contents of these areas can again serve as an excellent nutrient source for bacterial growth. Moreover, infection at such sites can cause hypertension in surrounding tissues, which in turn induces an additional impairment of blood flow.

In traumatic wounds the likelihood of infection is influenced by wound size, configuration depth and location, and the mechanism and cause of injury.

Technical factors

The duration of the operation and extent of the surgical area all correlate with the likelihood of wound infection.¹⁵⁴ Although the exact reasons remain unclear, a long and extensive operation provides a greater opportunity of tissue drying, tissue trauma, and external contamination. An inadequate surgical technique predisposes patients to wound infection. Examples include unnecessary tissue damage through inadequate use of clamps, improperly designed flaps, creation of tissue tension, and associated tissue devascularization and ischemia caused by hematomas, edema or tight or incorrectly located sutures, and the use of contaminated instruments or medical equipment. Inadequate layered closure, postoperative hemostasis, and the lack of or ineffective drainage producing dead spaces are also factors. Furthermore, an instability of fracture fragments and resulting motion inhibits the natural healing process and increases the risk of infection.

Delayed treatment of traumatic wounds results in a depression of tissue vitality, increased wound desiccation, and morbid edema. These changes increase the likelihood of infection by promoting microbial redistribution, wound contamination, and growth of pathogenic organisms.

Microbiology

Most infections of surgical wounds are contaminated by the patient's own endogenous microflora present on the skin or mucous membranes. Therefore, the microbiology is largely determined by the location of the wound and the presence or absence of salivary contamination. However, sources of pathogens also include surgical/hospital personnel and intraoperative factors including surgical instruments, articles brought into the operative field, and the operating room air.

Infections of oral wounds usually involve viridans streptococci and anaerobic bacteria. The most common group of bacteria responsible for cutaneous wound infection are *Staphylococcus aureus*, *Staphylococcus epidermidis*, and *Enterococcus* species.^{155,156} Cutaneous or sinus wounds are sometimes contaminated with saliva or other oral contaminants. In these cases, the microflora causing the infection may contain oral bacteria.

The prevalence of antibiotic-resistant strains has increased considerably in recent years. Methicillin-resistant *S. aureus* (MRSA) is proving to be the nemesis of modern surgery. Methicillin (meticillin) is a member of the penicillin class of antibiotic, and MRSA is resistant to this agent. However, MRSA is also resistant to a wide range of β -lactam and non- β -lactam antibiotics. As result, MRSA is often multi-drug-resistant, and once MRSA infection occurs, successful treatment may be very difficult to achieve. *Pseudomonas aeruginosa* is also an important pathogen as it is resistant to a wide range of antibiotics. Its involvement in infection is often indicated by the presence of a blue-green coloration of pus or exudate. This blue-green color is derived from pigments secreted by *P. aeruginosa*. MRSA and *P. aeruginosa* can colonize the skin and body of an individual without causing sickness and can cross-infect patients via hospital staff or contaminated medical equipment. Special attention must therefore be given to prevent the spread of this organism.

In the case of infected wounds associated with traumatic injury, exogenous organisms may enter into the wound with foreign bodies at the time of injury. The infection of simple traumatic wounds usually yields bacteria pre-existing at the site of injury. It should be remembered that spores of *Clostridium tetani* (the causative agent of tetanus) can enter the body through many kinds of wounds and although the topic is beyond the remit of this chapter it is important that clinicians understand the pathogenesis, clinical features, diagnosis, and prevention of tetanus.

Wound infections involving human bites commonly yield viridans streptococci, *Eikenella corrodens*, and oral anaerobes,^{157,158} whilst animal bites may involve unique microorganisms that reside within the mouth of the animal concerned. In bites inflicted by dogs and cats, *Pasturella* species are frequently involved.^{157,158}

Prevention of wound infection

Many risk factors influence the development of wound infections and awareness of these will help promote effective preventive strategies. Modern surgical techniques, medical care, and the use of prophylactic antibiotics will go some way to reduce this risk.

Risk of microbial contamination

The level of bacterial burden is the most significant risk factor. The US National Research Council group developed a system of classification for operative wounds based on the degree of microbial contamination.¹⁵⁹ A wound can be categorised as being clean, clean-contaminated, contaminated or dirty in terms of the risk of wound contamination and resultant infection. This classification is widely used to predict the likelihood of postoperative infection and to plan a

prophylactic antibiotic regimen. However, as the original version of this classification was made primarily for operative procedures in the respiratory, gastrointestinal, biliary, and genitourinary tracts, the definitions must be altered to fit the wound site involved.

Clean surgical wounds are defined as wounds with the lowest risk of infection and the smallest likelihood of contamination with endogenous bacteria (Table 29.32). Wounds in cutaneous tissue and paranasal sinus are sensitive to bacterial contamination. In wounds at these sites, saliva is the most common and clinically important contaminant. A clean wound is one where the surgical site can be prepared thoroughly, involves no significant tissue trauma or inflammation, and there is no communication with the oral cavity. The wound is closed by primary intention and, if necessary, drained with a closed drainage system. In contrast, although the oral microflora always contaminate oral wound sites, intraoral surgical wounds usually tolerate this contamination. This may be due to the host defense system and the excellent blood supply to the tissues. Therefore, inex-

tensive oral surgical wounds are generally managed in a similar manner to clean wounds, unless a patient's immunity is impaired or the wound exhibits significant tissue trauma or inflammation.¹⁶⁰

Clean-contaminated wounds are similar to clean wounds but infection is possible due to the likelihood of communication with the oral cavity under controlled conditions. The operative procedure usually commences in an uninfected environment and proceeds to incorporate entry into the oral cavity. Contaminated wounds result from operations where acute inflammation (without pus) is encountered or where there is a major breakdown in aseptic technique for fresh traumatic wounds. Dirty wounds occur when there is delayed treatment for a traumatic injury or the wound contains devitalized tissue or foreign bodies or involves an existing clinical infection.

Preventive strategies

The principle of preventive strategies for wound infection is to consider the presence or absence of any

Table 29.32 Classification of operative wounds and suggested strategies of prophylaxis for wound infection.

Classification	Criteria	Involved procedures and situation	Prophylactic antibiotic
Clean	Extraoral surgical wounds that are made under aseptic conditions and with no break in sterile technique, and have no evidence of infection, inflammation or contamination of the wound, and are primary closed	Tooth extraction and dentoalveolar surgery Submandibular and parotid gland surgery TMJ surgery	Unnecessary
	Unextensive oral wounds without significant inflammation or tissue devitalisation	Simple soft tissue surgery in oral or perioral regions	
Clean-contaminated	Same as clean but wound has the likelihood of communication with the oral cavity under controlled conditions (in the case of extraoral wound)	Major head and neck surgery Orthognathic surgery	Penicillin/cefazolin Penicillin
	Extensive operation within the mouth	Dental implants	Penicillin
	Intraoral wounds involving surgical placement of an implant	Surgery for simple fractures treated by transoral approach	Penicillin/cefazolin
	Graft surgery	Oroantral communication resulting from oral surgery	Penicillin
Contaminated	Operations where acute inflammation (without pus) is encountered or where there is major breakdown in aseptic technique or fresh traumatic wounds	Surgery for compound jaw fractures	Penicillin
		Fresh traumatic wound in the orofacial soft tissue*	Cefazolin
Dirty	Operations in which there is established clinical infection (with pus) or old traumatic wounds	Visibly contaminated or old orofacial traumatic wounds	Cefazolin
		Blunt trauma, gunshot wounds, bite wounds	Cefazolin
	Wounds containing significantly devitalized or infected tissues or foreign bodies	Surgery for suppurative osteomyelitis in the jaw	Penicillin

* Antibiotic prophylaxis is unnecessary where there are simple and fresh extraoral lacerations made with relatively clean objects.

factors that have an effect on the potential for wound infection and to manage them adequately before, during, and after surgery.

A healthy wound has a well maintained microbial homeostasis and therefore the first priority is to promote a healthy wound environment. Provision of a clean environment in the operative area is critical. Decontamination of the skin around the wound using soap, povidone-iodine, hexachlorophene solution or water is effective in reducing the risk of infection. Although surgeons may have concerns over bacteria residing in a patient's hair and advocate gentle shaving of the operative site, this action may increase the likelihood of wound infection as shaving induces minor trauma and may cause the breakdown of microbial homeostasis on the skin.^{160,161} Therefore, shaving should be kept to a minimum. It has been reported that the risk of infection increases with the length of time between shaving and surgery.¹⁶⁰ Therefore, if shaving is necessary, it should be performed as close to the time of surgery as possible. Tooth brushing, mechanical removal of dental plaque, and gingival scaling before surgery are, however, recommended.

Strict adherence to aseptic techniques and postoperative wound care are all necessary. Prolonged use of drains or long placement of sutures must be avoided, as they may become sources of postoperative contamination. Frequent irrigation of the wound with adequate use of antiseptics during the postoperative period reduces the likelihood of wound contamination.

Adequate surgical procedure, in accordance with the principles of surgery that optimize the wound-healing condition, is critical. Appropriate placement of the incision, rational flap design, meticulous hemostasis, elimination of dead spaces, and fastidious wound closure all help prevent wound infection. The prevention of tissue desiccation by constant irrigation is also effective. If the wound cannot be closed without tension or the persistence of pre-existing infection is suspected, then leaving the wound open should be considered. The wound edges may then come together naturally by means of granulation and contraction.

A traumatic wound within soft tissue should be treated as early as possible. In general, closure within 4 hours after injury is strongly recommended. Trapped foreign bodies, debris, clot, and necrotic tissue must be thoroughly removed through mechanical debridement and irrigation with copious amounts of saline solution. Open packing of a visibly contaminated wound with multiple foreign bodies that cannot be completely removed, may be effective in preventing infection.

Fractures should be treated soon after the injury has occurred. In particular, a compound fracture must be treated as soon as possible to minimize the size of the inoculum of bacteria contaminating the wound. Bony fragments must be fixed and immobilized adequately without any loose screws. Loose

teeth at the fracture site and teeth with a pre-existing abscess at the fracture site may be considered for removal to minimize the risk of postoperative infection.

It is important to correct pre-existing diseases that may depress the host defense system. Diabetes mellitus is perhaps the most common immunocompromising condition and therefore control of blood sugar is important. Diabetes also affects wound healing. Given the large number of undiagnosed cases of diabetes, blood glucose levels should be examined in all patients and controlled adequately before planned surgery. Careful monitoring and adequate control of blood glucose levels in the healing period in diabetic patients are also important to reduce the risk of wound infection. It has been suggested that the patient's blood glucose should be maintained below 200 mg/dl in the postoperative period.¹⁶²

Antibiotic prophylaxis

Antibiotic prophylaxis has had a positive impact on the incidence of infection after certain types of surgery. However, the clinician should not overestimate the effects of antibiotic prophylaxis and must recognize its limitations. Systemic prophylactic antibiotic should not be used routinely and should be tailored to the individual patient.

Benefit and risk of antibiotic prophylaxis

Antibiotic prophylaxis is highly effective in reducing the likelihood of infection. However, it has the potential risk of toxic and allergic reactions, drug interactions, alteration in the composition of normal host flora, and may promote an emergence of resistant bacteria. The cost of the antibiotic is also a matter that needs to be considered. In cases where the risk of infection is extremely low, an antibiotic provides little additional reduction in the incidence of infection. Such cases, therefore, do not need prophylactic antibiotic treatment.

Principles of antibiotic prophylaxis

The benefits versus the risk and the effectiveness versus cost should be considered for antibiotic prophylaxis. The most important factor to consider is the patient's host defense activity. In cases where the host immunity is significantly compromised or the surgical site may have reduced resistance to infection (e.g. a history of radiation therapy or poor blood supply), then antibiotic prophylaxis may be indicated.

Wounds categorized as clean-contaminated, contaminated, and dirty generally require antibiotic prophylaxis. As stated previously, oral wounds tend to have higher tolerance to microbial contamination and thus are more resistant to the establishment of infection. As such they are often treated as clean surgical wounds, in which antibiotic prophylaxis is unnecessary unless the patient has an immunocompromising

condition. However, if treated with a free flap, skin graft or implantation of foreign materials, prophylaxis should be used. Use of a prophylactic antibiotic is also advocated in surgery where there is an extensive operative area. The necessity of antibiotic prophylaxis for traumatic wounds within the oral cavity depends on the cause and mechanism of injury and the freshness and type of the wound.

Choice of antibiotic

The effectiveness of a prophylactic antibiotic is reliant on its efficacy against the predicted bacterial microorganisms most likely to cause infection, the extent of its tissue penetration, a lack of host toxicity, and its ability to cause a minimal disturbance to the intrinsic body microflora. An antibiotic with a narrow spectrum of activity should be chosen. Cost effectiveness should also be taken into consideration. In the case of oral wound, it is the penicillin class of antibiotic that fulfills these requirements. First-generation cephalosporin antibiotics may be adequate for the cutaneous or antral contamination.

Dose of antibiotic and duration of administration

The dosage of the prophylactic antibiotic should be the same or greater than that for therapeutic use. It should be remembered that the dose of the antibiotic is primarily determined by the serum level of the drug and the minimum inhibitory concentration (MIC) for the pathogen involved. If the surgical wound has a poor blood supply or the host defense mechanism is compromised, then a high dose of the antibiotic should be administered.

The timing of administration is critically important because the concentration of the antibiotic should be at its therapeutic level at the time of incision, during the surgical procedure, and, ideally, for a few hours postoperatively. An antibiotic administered preoperatively does exhibit a greater prophylactic effect than one administered postoperatively. Although many practitioners prescribe antibiotic postoperatively, it does not accomplish the true purpose of prophylactic antibiotic treatment. Oral antibiotics should be administered 30 minutes to 1 hour prior to the start of the operation. Intravenous or intramuscular antibiotics should be administered within 30 minutes of the incision time. Antibiotics should not be administered more than 2 hours prior to surgery. An additional dose of an intraoperative antibiotic should be administered to maintain an adequate serum concentration of the antibiotic during surgery if the operation is prolonged.

In the case of immunocompetent patients, if the surgery involves bacterial contamination that may occur only during the operation, antibiotic coverage during the operation may be sufficient and additional postoperative antibiotic may be unnecessary. In contrast, in circumstances where recontamination is suspected, use of an antibiotic postoperatively is rec-

ommended. Patients who have any factor that depresses the host defense mechanism or impedes wound healing have a great risk of recontamination during the healing period. In these cases, use of an antibiotic until evidence of biological wound sealing appears, is advocated.

Prophylaxis for wound infection on specific surgical procedure

There is contradiction over the effectiveness of prophylactic antibiotic use for each surgical procedure between published investigations. Although this section presents the guidelines for indication of antibiotic prophylaxis based on the latest pathologic and microbiologic knowledge and published clinical trials together with the authors' clinical experiences, actual use and regimen of antibiotic must be tailored to individual cases.

- *Endodontic procedures.* In pulpectomy, use of antibiotic prophylaxis is generally unnecessary regardless of the host's immunity level. However, antibiotic prophylaxis is advocated in cases of removal of infected necrotic material from the root canal of immunocompromised patients (e.g. treatment of asymptomatic (chronic) periapical lesions). In these situations the endodontic procedure itself can act as a trigger for acute infection.
- *Tooth extraction and other dentoalveolar surgery.* Most routine surgical procedures performed by dentists in healthy patients, such as tooth extraction, apicectomy, periodontal surgery, endodontic surgery, biopsy, do not require prophylactic antibiotics. Also, despite being a subject of controversy, third molar surgery (removal of impacted third molar in the mandible) does not require antibiotic prophylaxis. However, in cases of an immunocompromised host, prophylactic antibiotic treatment is recommended. The use of systemic antibiotics is also recommended where oroantral communication occurs. Use of antibiotics should be considered in cases of dental implant treatment. In situations where the maxillary sinus or the nasal cavity is exposed, prophylactic antibiotics should be used.
- *Removal of oral benign tumors and cysts in the jaw.* Indication of antibiotic prophylaxis follows the same principles as those of common dentoalveolar surgery. An extensive operation may require the use of prophylactic antibiotic.
- *Management of soft tissue trauma.* In cases of fresh wounds caused by simple traumatic injury within the mouth, antibiotic prophylaxis is unnecessary. Antibiotic prophylaxis is also unnecessary in simple and fresh extraoral lacerations made with relatively clean objects. However, in cases of blunt traumatic wounds, gunshot wounds, bite wounds caused by human or animal, lacerations from dirty

objects, traumatic injuries with delayed treatment, or contact with the saliva or any oral contaminants, use of antibiotic prophylaxis is recommended.

- *Management of fracture.* Fresh simple fractures of the condyle, ramus, and body of the mandible, with no saliva contamination, can be treated without antibiotic prophylaxis if treated through an extraoral approach.¹⁶⁰ However, if treatment is performed through intraoral incision, if treatment is delayed or if there is a pathologic fracture, use of antibiotic prophylaxis is recommended. Antibiotic prophylaxis is also advocated in cases of compound fracture of the mandible. Maxillary fractures involving the nasal cavity or paranasal sinuses similarly require antibiotic coverage.
- *Orthognathic surgery.* Orthognathic surgery performed via an extraoral approach is considered a clean procedure and antibiotic prophylaxis is unnecessary. However, if the surgery is undertaken transorally or if salivary or antral communication is anticipated, use of prophylactic antibiotics may be advocated.
- *Major maxillofacial surgeries.* Despite no scientific data, major surgical procedures in the mouth and face (e.g. sectioning and reconstruction of the jaw, plastic surgery using free flap, pedicle flap or biomaterials and bone or skin graft surgery) generally involve prophylactic antibiotic coverage.

Management of surgical site infections

Host defense systems serve to prevent microbial infection in cases where the tissue has received trauma through injury or surgery. The patient's own self-care as well as professional intervention through administration of antibiotic prophylaxis also minimize the likelihood of wound infection. Nevertheless, wound infection remains a clinical problem and might cause serious complications and sequelae.

Clinical findings of surgical site infections

The National Nosocomial Infections Surveillance System, which is the American national system for tracking health care-associated infections, has provided the diagnostic criteria of surgical site infections.¹⁶³ In summary, surgical site infections can be diagnosed if patients have at least one of the following conditions within 30 days of an operation or within 1 year of an important procedure: (1) purulent drainage becomes evident; (2) pathogenic organisms are isolated from the wound fluid or tissue; (3) at least one sign of inflammation (e.g. pain or tenderness, induration, erythema, local warmth of the wound) is present; (4) fasciae in the wounds spontaneously dehiscence; or (5) an abscess is formed. However, although these criteria are indeed helpful, diagnosis is often difficult because of the wide spectrum of possible clinical features and a variety of possible clinical

presentations. Most wound infections start with erythema and edema, which may be also seen as normal post-traumatic or postoperative inflammatory reactions. Therefore, it can be difficult to distinguish between pathogenic alternation and normal post-traumatic or postoperative inflammation.

In general, redness, pain, heat, and swelling associated with postoperative inflammatory reaction in the wound and periwound area decrease after the first few days, whilst such signs and symptoms related to surgical site infections tend to be persistent. The recurrence or further progression tends to manifest through increased erythema or swelling, thereby suggesting wound infection. High-grade, prolonged or recurrent fever can also be involved in wound infection, but postoperative fever is not a specific sign of wound infection and it is necessary to determine if the fever is actually due to infection. Invasive surgery itself can produce a low-grade fever for several days. Moreover, adverse effects of blood transfusion, side-effects of administered drugs, and other infections (e.g. acute viral nasopharyngitis, lung infection, urinary tract infection) can also induce fever.

The presence of purulent drainage and the formation of a fistula or abscess obviously indicate establishment of infection. The appearance of a purulent effluent or an increase in effluent volume may be indicative of wound infection. Therefore, the volume and nature of the drained effluent should be monitored carefully. Spontaneous dehiscence of fasciae is an indication of wound infection.

Infections of traumatic wounds that are not managed by surgical intervention are not considered as surgical site infections. However, the clinical features of these infections are generally similar to those of surgical site infections.

Diagnosis

Diagnosis is usually made with the consideration of all available factors, with special consideration given to the progress and course of local and systemic parameters. Imaging, in particular CT scans when available, can provide precise localization of any infection and source of contamination. However, these examinations are not necessarily helpful in all cases. Wide access and direct visualization through surgical reopening of the wound and drainage are in fact the most reliable means of investigation.

Laboratory data are sometimes helpful for diagnosis. However, it should be remembered that blood tests often reveal similar findings for acute infection in the postoperative period and for a few days after surgery if the wound is not infected. Moreover, the results may be affected by other underlying infections.

Management

Early intervention is important to prevent the progression of infection, with a subsequent onset of

associated complications and induction of sequelae. Management of wound infection will vary depending on the patient and commonly consists of cleansing the wound, application of surgical procedures, and antibiotic therapy.

An infected head and neck wound should be irrigated with copious amounts of physiologic saline solution. Dressing changes allow tissues to granulate, and the wound then generally heals by secondary intention over several weeks. Early closure of infected wounds is often associated with a relapse of infection and wound dehiscence. Opening the wound is recommended to ensure complete drainage, to promote exploration of all contiguous sites contacting the infection, and to allow an assessment of the potential sources of contamination. Surgical closure of fistulas and procedures to stop salivary contamination in the extraoral wound are both necessary. Rejection of the infected implanted material is the body's natural response and this does require removal. Antibiotic therapy is rarely sufficient on its own to salvage a contaminated implant.

In mild cases with no apparent wound necrosis or fistulae, management can be achieved with antibiotic therapy alone, avoiding the need for surgical intervention. However, a postponement or unnecessary delay in performing surgery must be avoided if intervention is required.

With regard to choice of antibiotics, penicillin, clindamycin, and metronidazole are all adequate candidates for empiric selection for those infections involving only oral contamination. For infections involving mixed contamination of oral and cutaneous organisms, use of broad-spectrum antibiotics, such as co-amoxiclav, β -lactam agents with sulbactam, cephalosporins, imipenem, and fluoroquinolones, is advocated. As mentioned so far, wound infection involves various types of organisms. Moreover, the wound infection often includes organisms that are resistant to antibiotics often used in the prophylaxis regimen. Culture and sensitivity testing should be performed in all cases and antibiotic selection should be reassessed based on results. A specimen from an infected

wound is usually contaminated with bacteria from the normal commensal microflora even if the specimen is obtained by careful aspiration. Careful interpretation of microbiologic data is therefore required, although it is often problematic to distinguish causative pathogens from those uninvolved organisms that colonize the wound.

Postextraction infection and alveolar osteitis (dry socket)

Postextraction infection is a form of surgical site infection that is perhaps the most commonly encountered by dentists. It is, however, distinct from alveolar osteitis which may exhibit a similar clinical picture.

Postextraction infection

Clinical signs and symptoms of postextraction infection usually appear 2–4 days after surgery. A pre-existing infection, inadequate oral hygiene in the healing period, peeled calculus or restoration material trapped in the operative site, and root or bone fragments left in the socket, all predispose to the infection. Immunocompromising conditions are also involved in predisposition. The microbiology of the infection is similar to that of other odontogenic infections. The disease often takes an acute form, and spontaneous pain, erythema, and elevated temperature of the wound may result. These symptoms are somewhat similar to alveolar osteitis, but, in contrast, postextraction infection presents with suppuration and apparent swelling. In the case of the mandibular molar, trismus can occur. Infection may spread extensively to involve adjacent soft tissues and jaw, and might cause fascial space infections, osteomyelitis, and maxillary sinusitis.

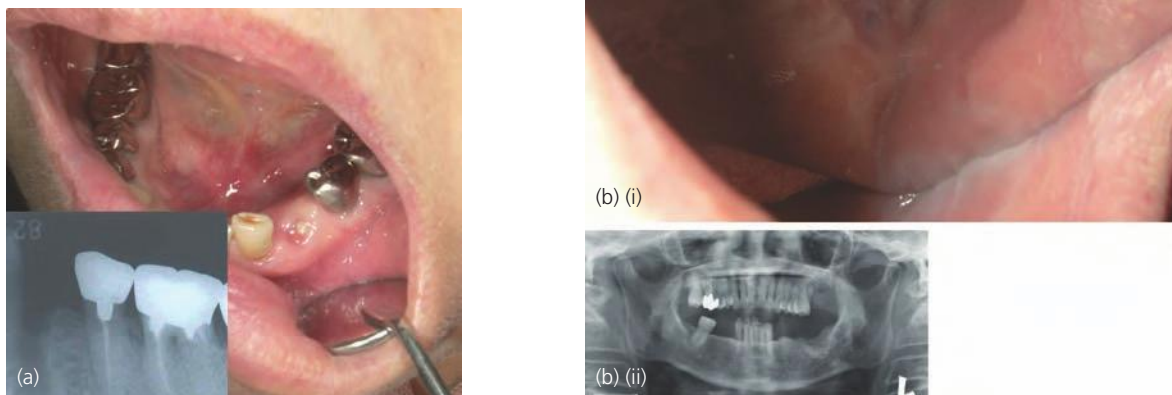


Fig. 29.95 Intraoral views and radiographs of cases of postextraction infection and squamous cell carcinoma (SCC). (a) Spontaneous drainage of pus from the extraction socket is evident in patients with postextraction infection. (b) (i) The SCC patient had no particular signs or symptoms except delayed healing of an extraction socket. (ii) Radiographic imaging of the SCC patient shows relatively wide destruction of bone around extraction socket.

Some cases primarily have a chronic form, without an apparent acute stage episode. In these cases, subjective symptoms are minimal, but patients often complain of delayed wound healing with or without pus discharge. The socket is usually filled with inflammatory granulation, and sometimes contains foreign materials, including necrotic bone fragments and broken tooth pieces.

Diagnosis is made based on the patient's history, clinical signs, and symptoms, laboratory tests, and imaging analysis. It should be noted that malignant tumors in the gingiva, jaw, and maxillary sinus can have a similar clinical presentation. These tumors produce a granulation-like mass in the socket after extraction of tooth (Fig. 29.95). Although differentiation from carcinoma is often difficult, careful radiographical study is often helpful. In the case of oral carcinoma, a range of irregularities of alveolar bone absorption in the tooth socket might be seen due to tumor invasion of the bone.

Treatment of the acute phase includes antibiotic therapy and drainage by incision. After the acute phase has subsided, debridement or curettage to remove necrotic tissues, inflammatory granulations, and foreign bodies from the socket may be necessary to prevent recurrence. As stated previously, the clinician must keep in mind the possible involvement of a malignant tumor. If the lesion is malignant, an invasive procedure may promote additional growth of the tumor and increase the risk of metastasis. If the possible involvement of a malignant tumor cannot be ascertained, a biopsy should be performed prior to surgical treatment.

Alveolar osteitis

Alveolar osteitis refers to the inflammation of alveolar bone following extraction of the tooth. It occurs when the blood clot at the site of the tooth extraction is dislodged, exposing underlying bone and nerves and thus causing increasing pain (Fig. 29.96). This condition has also been referred to as a dry socket and is not a suppurative infection. Alveolar osteitis is a major complication of tooth extraction, even when the extraction has been performed expertly and in an



Fig. 29.96 Alveolar osteitis. Exposed bone is seen.

aseptic manner. It occurs in 1–4% of all routine tooth extractions, and is more common following mandibular third molar extraction.^{164,165} Incidence rates of between 5 and 10% have been reported after removal of impacted mandibular third molars.^{166,167}

Although the etiology of alveolar osteitis has not fully been clarified, it is thought to involve a complex interaction between excessive localized trauma, bacterial invasion and their association to plasmin and the fibrinolytic systems.¹⁶⁷ Suggested risk factors include previous experience of alveolar osteitis, technical difficulties of surgery, poor oral hygiene of patient, active or recent history of acute ulcerative gingivitis or pericoronitis associated with the tooth to be extracted, smoking, use of oral contraceptives, and immunocompromised individuals.¹⁶⁷ It has been reported that less experienced surgeons may more often cause alveolar osteitis than more experienced surgeons and this has been related to the likely extent of traumatic tissue damage to the socket.

Symptoms generally start on the first to third day after extraction of the tooth. The exact duration of the condition varies and is dependent on the severity of the disease, but it usually ranges from 5–14 days. The denuded alveolar bone may be painful and tender. The pain is usually described as a throbbing ache or intense continuous pain irradiating from the empty socket to the ipsilateral ear and temporal region. The socket is very sensitive to chemical and thermal irrigation due to the exposure of nerve endings in the alveolar bone. Alveolar osteitis may also be accompanied by malodor and/or taste, and suppuration is generally absent. Regional lymphadenopathy is occasionally seen, but fever is uncommon.

It is necessary to exclude any other cause of pain on the same side of the face, especially simple postextraction pain and infection. Alveolar osteitis usually does not occur before the first postoperative day and the pain commonly appears suddenly or increases 1–3 days following extraction of the tooth. In contrast, in normal postextraction pain, the peak of pain intensity is usually on the operative day and this decreases with time. Postoperative infection usually induces a degree of swelling and often shows pus discharge.

Although alveolar osteitis is a condition in which there is loss of the blood clot from the socket, the socket may not always be completely empty. It may still contain a partially necrotic blood clot, often seen as a gray or dark yellow material. Diagnosis is confirmed by gently passing a small probe into the extraction wound. During probing, bare bone is encountered, which is extremely sensitive.

Alveolar osteitis is a self-limiting condition and therefore it generally resolves spontaneously. As there is no real treatment for alveolar osteitis itself, management of alveolar osteitis is directed primarily toward the relief of pain. Treatment options include gentle irrigation of the socket with a warm sterile isotonic saline solution, and packing with a zinc oxide–eugenol paste on iodoform gauze for relief of acute

pain. Coverage of the socket using a periodontal pack may be effective in preventing additional irritation of the socket. NSAIDs should be administered systemically for pain relief. The agent should be chosen based on the severity of pain, but use of potent types of NSAIDs is often required. Curettage should not be employed because it may destroy any previous attempt at normal healing and increase pain. The routine use of systemic antibiotics is not recommended, because alveolar osteitis is not a true infection. Use of topical antibiotics is also of little therapeutic value. However, if the patient is immunocompromised, use of a systemic antibiotic such as metronidazole may be advocated.

Prevention of hematogenously spreading infections

Hematogenous spread of bacteria from the wound site to a distal location can be of clinical concern. Oral bacteria can cause infections at locations physically separate from the portal of entry of the bacteria. These infections result from hematogenous spread of bacteria and bacteremia plays a significant role.

Although blood is normally sterile, bacteria in the local infection site or commensal bacteria within the body may enter into the bloodstream. The condition of bacteria being present in the blood is called bacteremia (see the earlier section, Complications of maxillofacial infections). Although the immune response to the bacteria could lead to sepsis and septic shock, bacteremia is usually harmless and transient, because of the actions of a vigorous immune system response. Body sites that harbor high numbers of microorganisms are likely sources of bacteremia. Although suppurative infection sites and the mucosa of the gastrointestinal tract are included in such instances, the mouth is the most important source of bacteremia. Normal daily activities, such as the use of mouth wash, tooth brushing, and chewing, all induce bacteremia.^{168–170} In addition, surgical procedures in the mouth or dental procedures inducing bleeding cause bacteremia, and occurrence of bacteremia following tooth extraction is virtually 100%.^{171–173} During bacteremia, bacteria do spread via the bloodstream throughout the body. Although these organisms are usually completely eliminated by the body's reticulo-endothelial system, on rare occasions, bacteria can survive and settle in certain parts of the body and cause infections away from the original site. Infective endocarditis (IE) and total joint replacement (TJR) infection are representatives of infections involving hematogenous dissemination of bacteria.

This section discusses the prevention of these infections based on the latest guidelines provided by authoritative medical organizations, namely the American Heart Association (AHA), the National Institute for Health and Clinical Excellence (NICE, UK) the British Society for Antimicrobial Chemo-

therapy (BSAC), the American Academy of Orthopedic Surgeons (AAOS), and the American Dental Association.

Infective endocarditis

IE is an inflammation of the inner layer of the heart (the endocardium), caused by microorganisms. The most common structures involved are the heart valves, especially if previously damaged congenitally, from surgery, by autoimmune mechanisms or simply as a consequence of old age. Bacteremia is a necessary event in the development of IE and delivers microorganisms to the surface of the valve. Although bacteremia would normally be cleared quickly with no adverse consequences in a healthy individual, a deformed heart value with its altered endothelial surface can be susceptible to bacterial adherence and subsequent multiplication. Therefore, if heart valves have been damaged, or replaced with an artificial biomaterial, bacteria have a greater chance of colonization.

In the case of IE, bacteria multiply and small clumps of material called vegetations may develop on the infected valves. These vegetations contain microorganisms, small blood clots, and other debris from the infection. The vegetations may prevent function of affected valves and allow infection to spread to other areas of the endocardium or heart tissue. Fragments of the vegetations (emboli) can detach and travel in the bloodstream to other parts of the body and cause distal infections with additional signs and symptoms. Usual initial signs of IE are intermittent fever, and subsequently heart murmurs, which result from abnormal flow of blood through faulty or damaged valves. Complications may appear if the infection is left untreated or if treatment is delayed. Damage to heart valves may lead to serious heart problems, including heart failure and heart abscess. Moreover, emboli may cause vascular obstruction (embolism) and block blood flow in other parts of the body. This can result in stroke, kidney failure, heart attack, and damage to gastrointestinal organs. Splinter hemorrhages, Janeway lesions (painless hemorrhagic cutaneous lesions on the palms and soles), Osler's nodes (painful subcutaneous lesions in the distal fingers), and Roth's spots (oval retinal hemorrhages) are all related to embolisms.

A variety of causative organisms are associated with IE, with staphylococci, viridans streptococci, and enterococci being the most common pathogens involved.¹⁷⁴ Despite improved prognosis due to advances in treatment, IE remains a life-threatening disease.

As described previously, bacteremia is an important factor in the development of IE. Surgical procedures that may cause bacteremia, and in particular invasive surgical procedures within the mouth, are therefore thought to be associated with development of IE in patients with predisposing factors. Prevention

of IE associated with surgical procedures has been the subject of much research, and several international medical societies and organizations, especially the AHA, have provided guidelines for prevention based on research findings.

In the AHA guidelines released in 1997, cardiac conditions are divided into high-, moderate-, and negligible-risk categories based on potential outcome if IE develops; the use of antibiotic prophylaxis is recommended when performing certain surgical and dental procedures in patients with high or moderate risk.¹⁷⁵ However, these guidelines were not evidence

Table 29.33 Indications of antibiotic prophylaxis against infective endocarditis (AHA).¹⁷⁶

Cardiac conditions where prophylaxis is recommended

Prosthetic cardiac valve

Previous infective endocarditis

Congenital heart disease (CHD):*

Unrepaired cyanotic CHD, including palliative shunts and conduits

Completely repaired congenital heart defect with prosthetic material or device, whether placed by surgery or by catheter intervention, during the first 6 months after the procedure

Repaired CHD with residual defects at the site or adjacent to the site of a prosthetic patch or prosthetic device (which inhibit endothelialization)

Cardiac transplantation recipients who develop cardiac valvulopathy

Dental procedures requiring antibiotic prophylaxis

All dental procedures that involve manipulation of gingival tissue or the periapical region of teeth or perforation of the oral mucosa in patients with the cardiac conditions outlined above

* Except for the conditions listed above, antibiotic prophylaxis is no longer recommended for any other form of CHD.

based. There has been no reliable clinical trial to assess the effect of antibiotic regimens in the prevention of IE. It has been noted that “appropriate” antibiotic prophylaxis does not prevent IE in all patients, and the risk of fatal anaphylaxis caused by administration of antibiotic may be greater than the effect of the antibiotic on prevention of IE. In addition to these criticisms, IE causation has recently shifted from procedure-related bacteremia to cumulative bacteremia involving daily activities such as chewing or tooth brushing. The AHA radically revised their guidelines in 2007.¹⁷⁶ The new guidelines emphasize good oral hygiene for preventing IE, and restrict the indication for antibiotic prophylaxis. Although all dental procedures that involve manipulation of gingival tissue or the periapical region of teeth or perforation of the oral mucosa remain as factors for prophylaxis indication, the indication for antibiotic prophylaxis should be restricted only to patients with the highest-risk cardiac conditions. These include a history of previous endocarditis, cardiac valve replacement surgery or a surgically constructed systemic or pulmonary shunt or conduit (Table 29.33). With regard to a regimen of antibiotic prophylaxis, administration of a single oral or intravenous dose of antibiotic is recommended. Oral administration of amoxicillin 1 hour prior to a dental procedure is recommended as a first-choice antibiotic, although first- or second-generation oral cephalosporin, clindamycin, azithromycin, and clarithromycin are alternatives. In patients who are unable to take oral medication, intramuscular or intravenous administration of ampicillin, cefazolin, ceftriaxone or clindamycin is recommended (Table 29.34).

NICE is an independent organization that provides national guidance on promotion of good health and prevention and treatment of ill health to the National Health Service in the UK. NICE has recently recom-

Table 29.34 AHA regimens for antibiotic prophylaxis of infective endocarditis.¹⁷⁶

Situation	Agent	Regimen: single dose 30–60 minutes before procedure	
		Adults	Children
Oral	Amoxicillin	2 g	50 mg/kg
Unable to take oral medication	Ampicillin	2 g, IM/IV	50 mg/kg, IM/IV
	or Cefazolin or ceftriaxone	1 g, IM/IV	50 mg/kg, IM/IV
Allergic to penicillins or ampicillin	Cephalexin*	2 g	50 mg/kg
	or Clindamycin	600 mg	20 mg/kg
	Azithromycin or clarithromycin	500 mg	15 mg/kg
Allergic to penicillins or ampicillin and unable to take oral medication	Cefazolin or ceftriaxone	1 g, IM/IV	50 mg/kg, IM/IV
	or Clindamycin	600 mg, IM/IV	20 mg/kg, IM/IV

IM/IV, Administration by intramuscular or intravenous route.

Cephalosporins should not be used in a person with a history of anaphylaxis, angioedema or urticaria with penicillins or ampicillin.

* Or other first- or second-generation oral cephalosporin in equivalent adult or pediatric dosage.

mended that antibiotic prophylaxis should not be given to people at risk of IE undergoing dental procedures including tooth extraction.¹⁷⁷ This guidance is more restrictive compared with the AHA guide to clinical use of antibiotics in preventing IE. Although there remains debate over the strategy of antibiotic prophylaxis for patients with the highest-risk cardiac conditions, most authorities, including the AHA, agree that there are limited effects of antibiotic prophylaxis in prevention of IE. The non-use of antibiotic prophylaxis during invasive procedures in the mouths of patients with the highest-risk cardiac conditions would be advocated unless reliable evidence to support the effect of antibiotic prophylaxis is present.

Table 29.35 Indications of antibiotic prophylaxis for hematogenous total joint replacement infection (AAOS, 2003).¹⁷⁸

Conditions placing patients at risk for prosthetic joint infection
Prosthetic joint placed within 2 years
Rheumatoid arthritis
Systemic lupus erythematosus
Drug- or radiation-induced immunosuppression
Previous prosthetic joint infections
Malnourishment
Hemophilia
HIV infection
Insulin-dependent (type 1) diabetes
Malignancy
Dental procedures requiring antibiotic prophylaxis for patients with condition outlined above
Tooth extractions
Periodontal procedures including surgery, subgingival placement of antibiotic fibers/strips, scaling and root planing, probing, recall maintenance
Dental implant placement and replantation of avulsed teeth
Endodontic instrumentation or surgery only beyond the apex
Initial placement of orthodontic bands but not brackets
Intraligamentary and intraosseous local anesthetic injections
Prophylactic cleaning of teeth or implants where bleeding is anticipated

Infection of total joint replacement

TJR is a surgical procedure in which certain parts of an arthritic or damaged joint, such as a hip, knee or shoulder, are removed and replaced with a prosthesis. TJR is generally indicated only for those patients who have severe arthritic conditions. Infections of a TJR can result in failure of the replacement and the need for extensive revision and treatment. Infections usually result from hematogenous seeding of bacteria on to joint implants from a variety of sources, both in the early postoperative period and for many years following implantation. There has been great concern over the involvement of oral bacteria, which can disseminate from the oral cavity in the blood after normal daily activities such as toothbrushing and chewing, and after invasive dental treatment such as tooth extraction.

The analogy of prosthetic joint infections with IE is invalid as the anatomy, blood supply, microorganisms, and mechanisms of infection are all different. Therefore, in contrast with IE, antibiotic prophylaxis for TJR infection remains supported. AAOS recommends antibiotic prophylaxis for all TJR patients who have had previous prosthetic joint infections, and for those with other conditions that may predispose the patient to infection (Table 29.35) prior to any invasive procedure that may cause bacteremia. Dental extractions, periodontal procedures (including surgery, scaling and root planing, probing, recall maintenance), dental implant placement, replantation of avulsed teeth, endodontic treatment, and intraligamentary and intraosseous local anaesthetic injections are included as invasive procedures for which antibiotic prophylaxis is indicated. In contrast, antibiotic prophylaxis is not advocated for patients with pins, plates and screws or other orthopedic devices that are not within a synovial joint or at increased risk for hematogenous seeding by microorganisms. The recommended antibiotic regimen is presented in Table 29.36.

In 2009, the AAOS released new information for antibiotic prophylaxis which included changes with regard to patients who have undergone TJR surgery. It is now advised that the length of time after the surgery is not an influencing factor and as such therefore

Table 29.36 Suggested antibiotic prophylaxis regimens against hematogenous total joint replacement infection.¹⁷⁸ No second doses are recommended for any of these dosing regimens.

Patient type	Suggested drug	Regimen
Patients not allergic to penicillin	Cephalexin, cephadrine or amoxicillin	2 g orally 1 hour prior to dental procedure
Patients not allergic to penicillin and unable to take oral medications	Cefazolin or ampicillin	Cefazolin 1 g or ampicillin 2 g intramuscularly or intravenously 1 hour prior to the dental procedure
Patients allergic to penicillin	Clindamycin	600 mg orally 1 hour prior to the dental procedure
Patients allergic to penicillin and unable to take oral medications	Clindamycin	600 mg intravenously 1 hour prior to the dental procedure

prophylactic antibiotic should be given to all patients receiving dental treatment who have previously undergone TJR surgery. However, it is generally accepted that there is little evidence to support the effectiveness of antibiotic prophylaxis on the prevention of infection. Concern has been expressed that the 2009 revision will result in an increased use of prophylactic antibiotic therapy and some of this increase may be unnecessary. Prophylactic antibiotic usage in accordance with the recent statement has been accepted in some countries but not others, including the UK. BSAC has advised that antibiotic therapy is not required for dental treatment of patients with TJR. BSAC consider that it is unacceptable to expose patients to potential adverse effects of antibiotic therapy when there is no evidence that it has any benefit. Differences in opinion will continue to exist and it is likely that further changes in the recommendations for the use of prophylactic antibiotic therapy in relation to dental treatment in patients with TJR will be proposed in the future.

Established odontogenic infections are highly likely to induce a bacteremia of sufficient magnitude to cause a prosthetic joint infection. Therefore, these infections should be eliminated prior to implementation of a TJR.

Oral fungal infections

A variety of fungal species have been recovered from the oral cavity of humans including *Saccharomyces* spp., *Geotrichum* spp., and *Cryptococcus* spp. However, by far the most prevalent fungal isolate are those species belonging to the genus *Candida*.

Candida infections

It is the infections caused by *Candida* (candidoses or candidiasis) which are the most frequent forms of oral fungal infection encountered. Oral candidosis primarily occurs in debilitated hosts, with perhaps the best example of this being the high incidence of

oral candidosis in human immunodeficiency virus (HIV)-positive individuals and acquired immunodeficiency syndrome (AIDS) sufferers.

Etiology and pathogenesis

Candida species are actually regarded as being members of the normal oral microflora and their involvement in infection in the debilitated host leads to their description of being opportunistic pathogens.⁹ The rate of commensal carriage of *Candida* varies in the literature and depends on the demographics of the studied population group. Carriage rates ranging from 20% to 80% have been reported. Although approximately 10–12 *Candida* species have been associated with human infections, over 80% of infections are caused by *Candida albicans*.

The transition from healthy oral carriage to the diseased state can occur due to a variety of reasons including both physiologic and iatrogenic host factors (Table 29.37).

Clinical features

Oral candidosis is not a single infection, and indeed four distinct primary forms of oral candidosis have been described, based on clinical presentation.

Pseudomembranous candidosis

Pseudomembranous candidosis (Fig. 29.97) manifests as the occurrence of white plaque-like lesions on the oral mucosa. The infection is synonymous with the description “oral thrush” and in the past has been most frequently seen in the mouths of neonates and elderly individuals. The infection can present on any oral mucosal surface but is most likely to be seen on the surface of the labial and buccal mucosa, hard and soft palate, and tongue. One of the key distinguishing features of pseudomembranous candidosis is the ability to remove the pseudomembranes by gentle scraping, thus revealing an erythematous underlying mucosa. Microscopic examination of the pseudomembranes reveals the presence of desquamated epithelial cells together with fungal elements that can be seen by either traditional Gram staining or the use of

Table 29.37 Host-related factors associated with oral candidosis.

Local host factors
Denture wearing
Steroid inhaler use
Reduced salivary flow
Carbohydrate-rich diet
Systemic host factors
Extremes of age
Endocrine disorders, e.g. diabetes
Immunosuppression
Receipt of broad-spectrum antibiotics
Nutritional deficiencies



Fig. 29.97 Pseudomembranous candidosis.

other staining techniques including the Periodic Acid Schiff stain or Gomori's methenamine silver staining.

Pseudomembranous candidosis has historically been considered to be an acute infection and correction of any identified host-related factor generally results in resolution of the condition. However, the advent of HIV infection in recent years and the associated increase in AIDS has resulted in a more chronic variant of pseudomembranous candidosis. In immunocompromised individuals, progression of oral infection to esophageal involvement can arise, leading to complications such as difficulties in swallowing and chest pain. Steroid inhaler use, particularly in young adults as part of the management of asthma, is associated with frequent cases of pseudomembranous candidosis in the soft palate.

Acute erythematous candidosis

Acute erythematous candidosis (Fig. 29.98) is seen as painful reddened patches on the oral mucosa and most frequently on the dorsum of the tongue. The administration of broad-spectrum antibiotics is associated with the occurrence of this form of oral candidosis, which is believed to occur as a result of the subsequent reduction of the bacterial community within the oral microflora. This in turn allows the numbers of *Candida* to increase due to reduced competition in the oral environment. Once antibiotic treatment has stopped, the lesions tend to resolve naturally without therapeutic intervention. It is the relationship between antibiotic therapy and acute erythematous candidosis that has given rise to its alternative description, namely, antibiotic sore mouth.

Chronic erythematous candidosis

The most prevalent form of oral candidosis is chronic erythematous or "*Candida*-associated denture stomatitis" (Fig. 29.99). The infection presents as a reddened mucosa beneath the fitting surface of a denture and up to 65% of denture wearers show clinical signs of this infection.¹⁷⁹ The condition is often asymptomatic and will develop under any acrylic denture or intra-oral appliance. It is, however, almost invariably seen in the palate rather than on the mandibular mucosa.



Fig. 29.98 Acute erythematous candidosis of the tongue.



Fig. 29.99 Chronic erythematous candidosis of the palate.

A key factor in the occurrence of this infection is poor oral hygiene or the presence of an ill-fitting denture.

Chronic hyperplastic candidosis

Chronic hyperplastic candidosis (CHC) (Fig. 29.100) is a less common form of oral candidosis and its occurrence is highest amongst middle-aged men who are tobacco smokers. CHC tends to be an asymptomatic infection, although some untreated cases (5–15%) may become dysplastic.¹⁸⁰

CHC is characterized by hyphal invasion of the oral epithelium by *C. albicans* and these can be seen following histological staining of biopsy material. It is the presence of the hyphae, together with an inflammatory cell infiltrate, which are the diagnostic features of this condition. As a consequence a biopsy is an essential component of the diagnostic procedure for CHC.

Whilst CHC can occur at any site on the oral mucosa it is most frequently encountered bilaterally as white patches in the buccal commissure regions. CHC lesions cannot be removed by gentle scraping without bleeding, thus distinguishing it from pseudomembranous candidosis. Two lesional types are described based on clinical appearance: (1) homogeneous lesions which are smooth and white; and (2) heterogeneous lesions appearing with a nodular or speckled appearance. It has been proposed that the heterogeneous form is more likely to undergo malignant transition.¹⁸¹



Fig. 29.100 Chronic hyperplastic candidosis in the commissure region of the mouth.



Fig. 29.101 Angular cheilitis.

Secondary forms of oral candidosis

In addition to the above described primary forms of oral candidosis, *Candida* are associated with other oral lesions, including those of angular cheilitis, median rhomboid glossitis, and chronic mucocutaneous candidosis.

Angular cheilitis

Angular cheilitis (Fig. 29.101) is seen as erythematous lesions at the angles of the mouth and is often associated with the occurrence of other forms of oral candidosis, most notably chronic erythematous candidosis. The exact role of *Candida* in the development of the lesion is unclear since microbial culture often reveals co-isolation with the bacterial species *Staphylococcus aureus*.

Median rhomboid glossitis

Median rhomboid glossitis (Fig. 29.102) presents as a symmetrically shaped area in the midline of the dorsum of the tongue. This chronic condition is strongly



Fig. 29.102 Median rhomboid glossitis.



Fig. 29.103 Chronic mucocutaneous candidosis.

associated with both smoking and the use of inhaled steroids. Isolation of *Candida* from the lesions is very common.

Chronic mucocutaneous candidosis

Chronic mucocutaneous candidosis (CMC) (Fig. 29.103) describes a collection of candidal infections largely confined to the skin, mucous membranes, and nails. A key predisposing factor would appear to be impaired cellular immunity against *Candida* and CMC is generally associated with an underlying congenital condition.

Diagnosis

Diagnosis of oral candidosis is dependent on the infection type and, whilst clinical appearance is extremely important, culture of *Candida* directly from the lesion is often used. Oral samples are generally cultured on Sabouraud dextrose agar which supports the growth of all oral *Candida* species with the added benefit of suppressing bacterial growth due to its relatively low pH.¹⁸² Occasionally microbiologists will incorporate antibiotics into the agar to further increase its selectivity. In recent years, other media, e.g. CHROMagar® *Candida*, have been developed that differentiate *Candida* species based on colony color (Fig. 29.104). The advantage of such media is that the presence of multiple *Candida* species in an infection can be determined which has importance in subsequent therapeutic selection. Identification of yeasts is confirmed through supplemental tests based on morphologic and physiologic characteristics.

Management

In the correct management of oral candidosis, it is firstly imperative that identification and control of any underlying and predisposing host factor is established. In addition, a range of antifungal agents is available to directly target the infecting *Candida*. The polyene antifungals, amphotericin B and nystatin are



Fig. 29.104 Various types of yeast colonies grown on CHROMagar® *Candida*. *Candida albicans*, which is the primary pathogen of oral fungal infections, yields green colonies on the agar, whilst the large majority of non-*albicans* *Candida* species produce purple colonies.

generally regarded to have the broadest spectrum of antifungal activity and are frequently used topically in a variety of oral formulations, including suspensions, lozenges, and pastilles. Polyenes are poorly absorbed through the gut and as such their use is relatively limited.

Azole antifungals have a fungistatic rather than fungicidal activity against *Candida* species¹⁸³ and consequently it is important to correct underlying host conditions during therapy to promote infection resolution. The azole antifungals are divided into two classes, imidazoles and triazoles, by chemical property. The two triazole agents frequently used to treat oral candidosis are fluconazole and itraconazole. The major benefit of these drugs is that they can be given orally and are well absorbed from the gut. Miconazole is a representative of imidazole agents, which is only used topically. It interacts with warfarin even when applied topically and should therefore be avoided in patients on coumarin anticoagulants.

Other fungal infections

Whilst *Candida* colonization of the oral cavity and associated infection are relatively frequently encountered, other oral fungal infections are rare and often restricted to infection in seriously immunocompromised patients. Often, oral infection occurs following spread of the causative organisms to the oral cavity from an already existing infection elsewhere in the body, often the lungs. The reverse is also true, with risk of dissemination of oral infection to cause subsequent systemic infection. Whilst much less common than candidosis, infections by these other fungal species are often more serious, with not just superficial injury to the oral mucosa occurring, but also infection to the paranasal sinuses, the orbit, and cranial base. Examples of these infections include mucormycosis, paracoccidiomycosis, and aspergillosis.

Mucormycosis

Mucor and *Rhizopus* species are those most responsible for mucormycosis. The infection is extremely rare in healthy individuals and is most often encountered in patients in African or Latin American countries. Conditions most commonly associated with mucormycosis include organ transplantation, leukemia/lymphoma treatment, and AIDS. The causative fungi are normal environmental organisms, often associated with decaying vegetation. During human infection these fungi can be isolated from the nose and mouth. Symptoms of this infection include fever, an acute sinusitis with erythema on the overlying skin, and orbital edema. Treatment of mucormycosis must involve surgical removal of all infected tissues which may include the palate, and nasal or eye structures. Amphotericin B may be used in treating this aggressive infection but its value in combating the disease remains uncertain.

Paracoccidiomycosis

The organism responsible for paracoccidiomycosis is *Paracoccidioides brasiliensis*. This species is a dimorphic environmental fungus that infects immunocompromised individuals through inhalation of conidia. At 37°C, yeast is the predominant growth form (as opposed to a mould at temperature <25°C) and these yeasts are responsible for the observed ulcerative lesions that manifest within the mouth, nose, larynx, and oropharynx. As with *Candida* species, Sabouraud dextrose agar will permit isolation of *P. brasiliensis* and histologic analysis of infected tissue will reveal characteristic granulomatous inflammation with infecting yeast. Amphotericin B in combination with sulfa drugs and itraconazole is the preferred treatment regimen for this infection; with early treatment the prognosis for recovery is usually good.

Aspergillosis

The term *Aspergillosis* is a collective one for infections caused by fungi of the genus *Aspergillus*. Around 20 species have been associated with human infection with *A. flavus*, *A. fumigatus*, *A. nidulans*, and *A. niger* being frequently implicated. As with the fungi mentioned previously, *Aspergillus* species are primarily associated with the environment, and soil and vegetation in particular.⁹ Infection of debilitated individuals generally occurs following inhalation of conidia.

Oral invasive aspergillosis is a very rare condition. Early recognition through biopsy and microbiologic analysis of the lesions, followed by immediate antifungal therapy is essential to prevent extensive tissue damage. Aspergillosis of the oral region is believed to start on the marginal gingiva and then spread to the adjacent tissues, including the mucosa, muscle, and alveolar bone. Treatment of oral invasive aspergillosis involves both surgical and medical intervention with aggressive systemic antifungal therapy accompanying the debridement of all necrotic tissue required after diagnosis to ensure successful treatment.

Oral and perioral viral infections

A variety of viral infections have been encountered in the mouth and perioral tissues. However, by far the most prevalent are those caused by members of the DNA herpes group of viruses (Table 29.38). It has been estimated that 90% of adults are chronic carriers of at least one type of herpes virus, which are acquired as a result of infection during childhood or early adulthood. Following initial (primary) infection, these viruses have the property of latency and reside in the tissues asymptotically for the remainder of the patient's life. Under certain conditions latent virus will reactivate to cause recurrent symptoms (secondary infection). It is also recognized that herpes viruses are periodically shed without causing symptoms in the saliva of up to 70% of the population at least once

Table 29.38 Orofacial conditions associated with the human herpes group of viruses.

Virus	Trivial name and acronym	Clinical condition
Human herpes virus 1	Herpes simplex virus 1 (HSV-1)	Primary herpetic gingivostomatitis Herpes labialis Recurrent herpetic ulceration
Human herpes virus 2	Herpes simplex virus 2 (HSV-2)	Genital herpes (Rarely: Primary herpetic gingivostomatitis Herpes labialis Recurrent herpetic ulceration)
Human herpes virus 3	Varicella zoster virus (VZV)	Chickenpox Herpes zoster (shingles) Ramsay Hunt syndrome
Human herpes virus 4	Epstein Barr virus (EBV)	Infectious mononucleosis (glandular fever) Nasopharyngeal carcinoma Burkitt's lymphoma Hairy leukoplakia
Human herpes virus 5	Human cytomegalovirus (HCMV)	(Possibly oral ulceration or salivary gland swelling in immunocompromised patients)
Human herpes virus 6	Human herpes virus 6 (HHV-6)	Roseola infantum (Nagayama's spots)
Human herpes virus 7	HHV-7	None known
Human herpes virus 8	Human herpes virus (HHV-8)	Kaposi's sarcoma

a month. Such shedding represents a risk of spread of the virus and in part explains the widespread nature of herpes simplex type 1 (HSV-1) in the community. Reactivation of HSV-1 has been implicated as an etiological factor in periodontal disease and erythema multiforme.^{184,185} Other viruses that may manifest in the mouth include cosackieviruses, paramyxoviruses, and papilloma viruses.

Primary herpetic gingivostomatitis

This condition is almost uniformly caused by primary infection with HSV-1 although rare cases involving herpes simplex type 2 (HSV-2), the herpes virus associated with genital tissues, have been reported.⁹ Infection classically occurs during the first few years of life and the symptoms of mild oral discomfort over 4–5 days may be misdiagnosed as an episode of teething. In older children or early adulthood, the presentation is often more dramatic and characterized by the development of blood-crusted lips (Fig. 29.105),



Fig. 29.105 Primary herpetic gingivostomatitis presenting as widespread oral vesicles and erosions.



Fig. 29.106 Multiple ulcers due to primary herpetic gingivostomatitis.

gingival swelling, multiple oral ulcers (Fig. 29.106), lymphadenopathy, and pyrexia. The mucosal lesions develop as intraepithelial vesicles but these soon rupture to produce the typical shallow yellow ulcers with surrounding erythema. All signs and symptoms of primary infection resolve within 10 days.

Diagnosis can often be made on the nature of the clinical presentation alone. However, it can be confirmed by isolation of the virus in tissue culture. Since high numbers of virus will be present not only on the ulcerated mucosa but also within the saliva it is not necessary to identify specific lesion and a swab from any oral site will be sufficient. The swab should be placed in viral transport medium for transfer to the laboratory. Results of tissue culture are rarely available before 3 days and may take up to 10 days. A rapid diagnosis can be achieved by use of immunofluorescence on a smear of the lesion although this technology is only used in specialist centres. Alternatively, an acute blood sample (taken at time of presentation, day 1) and convalescent sample (taken after healing, day 7–10) can be used to demonstrate an increase in antibody titre to HSV-1.

Treatment is primarily palliative and includes bed rest and oral anti-inflammatory such as ibuprofen or paracetamol. It is essential that the patient maintains adequate fluid intake. Consideration should be given for the need to provide the patient with antiviral therapy. If the patient is otherwise healthy and symptoms are relatively mild then there is no requirement for an antiviral since it is unlikely to make any difference to recovery time. However, if the symptoms are severe or the patient is immunocompromised then it is wise to give systemic antiviral therapy.

There are relatively few antiviral agents compared to the number of antibacterial drugs that are available. The development of aciclovir led to the first specific antiviral agent. Aciclovir is a nucleoside analog drug that has activity against members of the herpes group of viruses, in particular HSV-1 and HSV-2. Aciclovir acts by blocking viral replication and therefore it is important to provide the drug at an early stage, preferably within 48 hours of the onset of acute symptoms. Two alternative antiviral agents used to treat infection caused by the HSV-1 or HSV-2 are penciclovir (available only in topical form) and famciclovir (the oral pro-drug of penciclovir).

The agent of choice for primary herpetic gingivostomatitis is aciclovir, preferably as a suspension since the tablet form is difficult to swallow in the presence of widespread oral ulceration. Aciclovir should be given at a dose of 200 mg, five times daily for 5 days. Children under the age of 2 years should receive a half-dose, while those over 2 years of age should receive the standard 200 mg dose.

Herpes labialis

Approximately a third of patients who have had primary herpetic gingivitis will subsequently suffer recurrent episodes of herpes labialis, also known as cold sores or fever blister, as a result of reactivation of latent HSV-1. There have been reports of the isolation of HSV-2 from recurrent herpes labialis but this is extremely rare. The recurrent lesions characteristically begin with a tingling or burning sensation (prodrome) in the vermillion border of the lip due to reactivation of latent virus in local neural tissues. In classic herpes labialis, a collection of painful intraepi-

thelial vesicles will develop within 24 hours at the site of the prodrome symptoms. However, approximately 25% of episodes have no prodromal stage and the lesion presents directly as vesicles. Within 48 hours the vesicles rupture and coalesce to leave erosions, which subsequently crust over (Fig. 29.107) and eventually heal within 7–10 days. The clinical appearance is so characteristic that diagnosis is based on this alone. However, if necessary, the presence of HSV, either type 1 or type 2, can be confirmed by isolation in tissue culture from a swab or the use of immunofluorescence on a smear of the lesion.

Factors that predispose to the development of herpes labialis in susceptible individuals include sunlight, trauma, stress, fever, menstruation, and immunosuppression. A sunscreen applied to the lips can be effective in reducing the frequency of sunlight-induced recurrences. A number of topical antiseptic or analgesic preparations, based on ammonia, povidone–iodine or lidocaine, are widely available to the general population for treatment of herpes labialis. However, these non-specific products are of limited proven benefit. Clinical trials have demonstrated that the topical application of an antiviral agent, either aciclovir or penciclovir, can reduce the duration of the outbreak and reduces viral shedding. Docosanol, an agent that alters the cell membrane to prevent viral entry, is also available for topical application for orofacial herpes simplex infections. Aciclovir, at a dose of 200 mg five times daily should be given systemically if herpes labialis develops near the eye (Fig. 29.108). Individuals with severe or frequent recurrences can benefit from the prophylactic use of systemic aciclovir taken at a dose of 200 mg three times daily for 6–9 months.



Fig. 29.107 Early scabbing stage of herpes labialis.



Fig. 29.108 Recurrent herpes simplex viral infection causing facial swelling and closure of the eye.

Recurrent herpetic ulceration

Reactivation of latent herpes simplex virus also produces intraoral ulceration. The hard palate is the site most frequently involved and the lesions present as a single localized crop of small shallow painful ulcers with surrounding erythema (Fig. 29.109). The outbreak may be preceded by prodrome symptoms of itching or pain similar to herpes labialis. Vesicles soon rupture to produce the characteristic ulcers which heal within 7–10 days. Whilst almost exclusively due to HSV-1, there is increasing evidence that HSV-2 can also cause this type of ulceration. This observation is probably due more to the frequent transmission of virus as a result of direct contact



Fig. 29.109 Reactivation of HSV-1 producing cluster of ulcers on hard palate.

during orogenital sexual practices but may in part also reflect the more widespread use of typing methods for suspected orofacial herpetic infections.

Chickenpox

Primary infection with varicella zoster virus (VZV) causes chickenpox, which occurs most frequently in childhood. The characteristic presenting clinical sign is a blistering skin rash on the abdomen. The skin lesions may occasionally be accompanied by the development of vesicles in the palate and faucial region, which rapidly rupture to produce small ulcers (2–4 mm). The diagnosis of chickenpox is usually made from the classic history and nature of the cutaneous lesions. Fluid from a skin blister can be sent for viral culture or a lesional smear examined using immunofluorescence to obtain definitive diagnosis. Treatment is supportive including a systemic antihistamine to reduce itching. Total resolution of symptoms will follow within 7–10 days. If the patient is immunocompromised then aciclovir at a high dose (800 mg five times daily) or standard doses of famciclovir (either 250 mg three times daily or 750 mg once daily) or valaciclovir (1 g three times daily) should be given. A vaccine for chickenpox is now available.

Herpes zoster (shingles)

Herpes zoster, which is also known as shingles, is caused by reactivation of latent VZV in sensory nerve ganglia. Most cases of herpes zoster affect the trunk and produce a unilateral horizontal band of skin lesions on the back and chest wall, restricted to one dermatome. When the trigeminal nerve is involved, which accounts for 10–15% of episodes of herpes zoster, it also manifests unilaterally in one division of the nerve. In the case of the maxillary or mandibular branch, the severe pain, which often precedes the skin and mucosal lesions by a few days, may be confused with toothache. However, once the characteristic distribution of vesicular lesions and mucosal erosions occurs (Fig. 29.110), the diagnosis of herpes zoster is usually obvious.

Although the diagnosis can be made on the basis of clinical presentation alone, confirmation of herpes zoster can be made by isolation of VZV in cell culture or immunofluorescence on a smear. Antiviral treatment should be given as early as possible, preferably within the first 48 hours of symptoms. Whilst in the past aciclovir was used for this infection, it is now



Fig. 29.110 Herpes zoster due to reactivation of VZV causing unilateral ulcerative lesions in the maxilla.

preferable to administer either famciclovir (250 mg three times daily or 750 mg once daily) or valaciclovir (1 g three times daily) due to the increased bioavailability of these antiviral drugs. Postherpetic neuralgia may subsequently be a major problem in these patients and therefore it has been recommended that antiviral therapy should be provided for at least 10 days to reduce the likelihood of this extremely painful condition. The symptoms of postherpetic neuralgia are extremely difficult to treat although some benefit may be gained by use of tricyclic antidepressant drugs and gabapentin. Topical application of capsaicin 0.075% cream three or four times daily has been reported to be of benefit in difficult cases.

Ramsay-Hunt syndrome

The development of a unilateral lower motor neuron facial nerve palsy (Fig. 29.111) combined with the appearance of a painful vesicular rash on the tympanic membrane and external auditory canal on the same side is termed Ramsay-Hunt syndrome. It has been proposed that this situation develops as a result of reactivation of varicella zoster virus in the region of the geniculate ganglion of the facial nerve. The clinical diagnosis can be supported by detection of VZV in the fluid of the vesicles. Treatment consists of provision of standard zoster doses of an antiviral (valaciclovir or famciclovir) to limit viral replication combined with prednisolone (typically 20–30 mg daily) to reduce inflammation. Therapy should be continued until the symptoms resolve, which is usually within 14 days.

Infectious mononucleosis (glandular fever)

Infectious mononucleosis, also known as glandular fever, is characterized by the onset of a painful throat and submandibular lymphadenopathy, which may occasionally be accompanied by fine petechial hemorrhages in the hard and soft palate. In addition, a white pseudomembrane may develop on the tonsils. This condition is caused by infection with Epstein-Barr virus (EBV), probably as a result of kissing.¹⁸⁶



Fig. 29.111 Facial nerve palsy.

Diagnosis is confirmed by demonstration of a lymphocytosis and atypical mononuclear cells (Downey cells) in a blood film or, alternatively, by detection of heterophile antibodies ("Monospot test"), EBV-specific antibody (immune-fluorescence) or EBV DNA (by polymerase chain reaction (PCR)). Treatment is supportive and includes bed rest. Ibuprofen or paracetamol is helpful in relieving the symptoms of sore throat. Approximately 70% of adults acquire the virus by the age of 30 years and periodically shed it in the saliva without symptoms.

Hairy leukoplakia

Oral hairy leukoplakia was first described in 1984 as a non-painful corrugated white lesion on the lateral border of the tongue in HIV-positive individuals (Fig. 29.112).^{187,188} Whilst originally only seen in HIV disease, a case was described in 1999 in an HIV-negative acute leukemia patient and subsequently in recipients of solid organ transplants.^{189,190} Furthermore, it has also been described at non-lingual sites such as the buccal mucosa (Fig. 29.113). Whilst described as "hairy", the appearance of the side of the tongue is often more corrugated. Diagnosis is confirmed by demonstration of EBV in lesional tissue using *in situ* hybridization with appropriate DNA probes. The exact role of EBV in hairy leukoplakia remains uncertain. Imprint cultures of hairy leuko-



Fig. 29.112 Hairy leukoplakia on the lateral margin of tongue.



Fig. 29.113 Hairy leukoplakia presenting as a white patch in the buccal mucosa.

plakia often yield candidal species, but it is likely that this represents opportunistic secondary infection rather than a primary etiologic factor.

Hairy leukoplakia does not require specific treatment unless causing discomfort or esthetic problems. Systemic aciclovir (800mg five times daily) or preferably ether valaciclovir (1g three times daily) or famciclovir (500mg times daily) should be given over 2 weeks. Unfortunately, the lesions often recur a few months after the antiviral therapy. The topical application of podophyllin resin (25% solution) or retinoic acid has been described, but this is also of limited benefit due to recurrence. Small localised lesions can be excised surgically. In addition, the application of cryotherapy has been reported but is not widely employed.

Burkitt's lymphoma

Burkitt's lymphoma is named after the surgeon Denis Burkitt, who in 1958 mapped the geographical distribution of this condition across Africa.¹⁹¹ This aggressive rapidly growing B-cell tumour has two major forms, the endemic (African) form and the non-endemic (sporadic) form. The African form most frequently presents as a swelling of either the mandible or maxilla with mobility of the teeth and cervical lymphadenopathy in childhood. There is a strong association with EBV in the African form but its role in the sporadic form elsewhere in the world is less clear. Chemotherapy should be started as soon as possible but unfortunately the outcome remains poor, especially in later stages, and death often occurs rapidly after diagnosis.

Nasopharyngeal carcinoma

Nasopharyngeal carcinoma is a relatively rare tumor but is seen more frequently in China and South-East Asia.¹⁹² Patients will present with difficulty in breathing or speaking and complain of swelling in the throat, bleeding from the nose, and earache. The spread of extensive lesions will be visible in the posterior oropharynx (Fig. 29.114). There is a direct asso-



Fig. 29.114 Extensive squamous cell carcinoma in posterior oropharynx.

ciation with this cancer and EBV. Radiotherapy is the treatment of choice, together with chemotherapy in advanced cases.

Roseola infantum

This acute and extremely common condition of childhood is characterized by mild fever and a skin rash on the body that last only a few days. Whilst the skin rash does not affect the face, erythematous papules in the soft palate and uvula, sometimes referred to as Nagayama's spots, may be observed. The intraoral lesions are thought to be caused by human herpes virus type 6. Diagnosis is made on clinical grounds and no active treatment is required since the condition is self limiting.

Kaposi's sarcoma

Kaposi's sarcoma was first described by Moritz Kaposi, a Hungarian dermatologist, in 1872.¹⁹³ The original cases presented as multiple pigmented papular lesions on the skin of elderly men in the Mediterranean region or of Eastern European descent. It received particular attention in the 1980s when a high incidence was observed in association with HIV infection and subsequently other immunosuppressed patients (Fig. 29.115). The mouth is the initial site of involvement in 15% of AIDS-related cases. The hard palate is the most frequent site, although it may also arise on the gingivae. This tumor is not a true sarcoma but a proliferation of lymphatic endothelial cells. The lymphatic channels fill with blood to give the classic appearance of a bruise in the early stages, then a dark swelling. Human herpes virus 8 has been encountered in all forms of Kaposi's sarcoma and is believed to be the etiologic agent of this condition.¹⁹⁴ Small lesions can be excised whilst larger ones may be treated with either low-dose radiotherapy or by the injection of a chemotherapeutic drug, such as vinblastine. Extensive or multiple lesions may require the provision of systemic chemotherapy.



Fig. 29.115 Kaposi's sarcoma presenting as a typical pigmented lesion in the hard palate.

Hand, foot, and mouth disease

Hand, foot, and mouth disease acquired its name from the characteristic distribution of lesions (Figs 29.116, 29.117, and 29.118), that present as bright red macular or vesicular eruptions on the skin of the hands and feet and mucosa of the pharynx, soft palate, buccal sulcus or tongue. The development of oral lesions is preceded by facial pain. The signs and symptoms are transient and last less than 3 days. This condition is often caused by the highly infectious



Fig. 29.116 Hand, foot, and mouth disease presenting as vesicular lesions on the fingers in combination with ulceration of the oral mucosa.



Fig. 29.117 Hand, foot, and mouth disease presenting as vesicular lesions on the toes in combination with ulceration of the oral mucosa.



Fig. 29.118 Hand, foot, and mouth disease presents as vesicular lesions on the fingers and toes in combination with ulceration of the oral mucosa.

enterovirus, Coxsackie virus subspecies A16. However, Coxsackie virus types A4, A5, A9, and A10 have also been isolated from patients with this condition. The diagnosis of hand, foot, and mouth disease is usually made on the basis of the characteristic clinical signs. Culture of Coxsackie viruses is not widely available and therefore if confirmatory diagnosis is required this has to be based on demonstration of increased levels of specific antibody in acute and convalescent blood samples. Treatment is supportive and antimicrobial agents are not required.

Herpangina

Herpangina occurs predominantly in children, and presents as sudden onset of fever and sore throat with subsequent development of papular, vesicular lesions on the oral mucosa and within the pharynx. Coxsackie virus subspecies A2, A4, A5, A6, and A8 have all been isolated from patients with herpangina. The severity of symptoms is variable, but clinical resolution usually occurs within 7–10 days. The diagnosis of herpangina is usually made on the basis of the characteristic clinical signs and symptoms. If definitive diagnosis is required this has to be based on demonstration of increased levels of specific antibody in acute and convalescent blood samples. Treatment consists of bed rest and the use of an antiseptic mouthwash, such as 0.2% chlorhexidine gluconate two or three times daily. Patients should be encouraged to maintain adequate fluid intake.

Verruca vulgaris

Verruca vulgaris, also known as oral warts, present most frequently as one to four small localized growths on the labial or lingual mucosa (Fig. 29.119). Involvement of the mouth is often associated with lesions on the fingers which may act as a source of the causative human papilloma virus (HPV), usually HPV type 2 or HPV type 4. Alternatively, the oral lesions may arise from orogenital contact, where HPV types 6, 11, and 16 are more prevalent. The clinical appearance is characteristic, but diagnosis can be

confirmed by histopathologic findings of excisional biopsy tissue. Immunostaining will determine the presence of the particular type of papilloma virus type. Excision is usually curative and recurrence is uncommon except in immunocompromised individuals.

Verruca vulgaris is a benign tumor, but in recent years there has been increasing interest in the potential role of HPV 16 in the etiology of oropharyngeal carcinoma as a result of oral sexual practices.¹⁹⁵ Further evidence of the involvement of HPV 16 in this situation would support vaccination against this virus. The recognized association of this virus in cervical cancer has led to the proposal that all females are vaccinated against HPV 16 at the age of 12 years.

Focal epithelial hyperplasia (Heck's disease)

Heck's disease is a rare condition that is characterized by the development of multiple smooth-topped pink papules on the buccal mucosa, labial mucosa, tongue, and gingivae (Fig. 29.120). Molecular methods and epidemiologic studies have supported the involvement of HPV types 13 and 32 in this condition.¹⁹⁶ It was first described in Native Americans and the Inuit but has also been described in other populations. No active treatment is required although larger lesions can be surgically removed if causing esthetic problems or local symptoms.

Mumps

Mumps (endemic parotitis) classically presents as painful swelling of either one or both of the parotid glands. However, other major salivary glands may also occasionally be involved. Other frequent features include sore throat, earache, and pain on chewing. The orifice of the parotid duct may appear erythematous with reduced salivary flow. The differential diagnosis of acute suppurative sialadenitis can be made by the absence of a purulent discharge.



Fig. 29.119 Two localized lesions of verruca vulgaris on the ventral surface of the tongue.



Fig. 29.120 Multiple localized epithelial swellings in Heck's disease.

Complications include meningoencephalitis and orchitis in about a third of cases or more rarely myocarditis and nephritis. This condition is caused by an enveloped RNA paramyxovirus that initially infects circulating T lymphocytes that subsequently infect salivary duct epithelial cells.

Diagnosis can usually be made on the basis of clinical symptoms. However, if there is doubt then serologic investigation to demonstrate the presence of specific IgM antibody can be used. In addition, a sample of saliva, collected by cannulation of the parotid duct, can be examined by electron microscopy for virus particles. Treatment is supportive and antibiotic therapy is not required. The MMR (mumps, measles, and rubella) vaccine provides immunity.

Measles

Measles is a common and highly infectious disease of childhood that presents as an acute exanthematous skin rash and fever. The patient is likely to develop conjunctivitis, nasal discharge, headache, and sore throat. The extraoral signs may be accompanied by transient small discrete macules in the buccal mucosa (Koplik's spots), the appearance of which may aid diagnosis. Definitive diagnosis involves serologic detection of measles IgM antibody or isolation of measles RNA from a nasal swab or sputum. Treatment is supportive and most patients will recover without problems. However, it must be remembered that this infection may be fatal, especially in malnourished states or immunocompromised individuals. The MMR (mumps, measles, and rubella) vaccine provides immunity but in areas of low uptake or non-availability of the vaccine, measles still presents a serious illness due to potentially life-threatening bronchopneumonia or encephalomyelitis. In Saharan Africa, the virus has also been implicated in gross destruction of the orofacial tissues (noma) in malnourished individuals.¹⁹⁷

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Part 5: Oral Pathologic Lesions

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Chapter 30

Initial Evaluation and Management of the Oral and Maxillofacial Pathology Patient

Allen Cheng and Brian L. Schmidt

In this chapter, the following topics are covered:

- How to take an adequate history and carry out an examination for a pathological lesion in the oral cavity and related structures.
- Current imaging techniques for further defining pathological lesions of the head and neck.
- The principles of biopsy for oral and related lesions including incisional biopsy, excisional biopsy, and fine needle aspiration. Biopsy techniques applicable to different oral sub-sites are described.
- Adjunctive diagnostic techniques including toluidine blue, brush technique, VELscope®, and chemiluminescents.
- The diagnosis and management of dysplasia and other premalignant lesions.

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In seeking absolute truth we aim at the unattainable and must be content with broken portions.

Sir William Osler

Oral cancer is an uncommon disease. Despite being the sixth most common malignancy in the world,¹ it is a clinical rarity in most practices. Maybe because of the low incidence of oral cancer, the diagnosis and treatment of it are relegated to a few with interest in this esoteric field. However, this also means that many providers, whether they are primary care physicians, dentists, oral and maxillofacial surgeons, otolaryngologists, or pathologists, do not feel adequately equipped to diagnose oral cancer. Patients are often unaware that such cancers exist. A direct consequence of this common inexperience is that patients with oral cancer have a significant delay in

diagnosis from initial presentation to evaluation by a specialist, leading to diagnoses when tumors are advanced in stage.² A delay in diagnosis contributes to the relatively poor prognosis of oral cancer, which is disturbing given that oral cancer involves an anatomic location that can be evaluated by direct visual and tactile examination.^{3,4}

The purposes of the first part of this chapter are to provide an overview of the diagnostic armamentarium available for oral cancer and to then outline a basic algorithm for management of suspicious oral lesions. The second part of the chapter will discuss the methods and shortcomings of surgical pathology with the intent to provide a framework for which the surgeon may best assist the pathologist in making the correct diagnosis and then subsequently interpret the pathologist's report.

Approaching the oral and maxillofacial pathology patient

The first step to approaching a patient with oral and maxillofacial pathology is to take a thorough history and perform a physical examination. Information gleaned from the history and physical examination will provide clues to the correct diagnosis and guide the clinician in selecting the appropriate tools to validate the diagnosis.

Obtaining the history

In taking the history of present illness, one should first establish a timeline for the disease. One should ask when the patient first noticed symptoms. Symptoms of interest include pain, paresthesia, dysphagia, dyspnea, otalgia, trismus, voice changes, weight loss, fevers, chills, and malaise. Although not necessarily present, pain is the most common presenting symptom for head and neck cancer.^{4,5} Paresthesia in the head and neck area is also highly concerning, and should be considered a sign of potential malignancy until proven otherwise. Either of these symptoms, when occurring without a likely explanation, should prompt a thorough investigation including a neurologic exam, imaging studies, and, potentially, tissue sampling.

The surgeon should inquire about any prior treatment or diagnostic tests. If biopsies have been performed in the past, it is essential to review both the pathology report and the pathology slides. One should not take a prior pathologic diagnosis at face value, as errors may have occurred at any point from the biopsy, to the receipt of the specimen by the pathologist, to the final report. In addition, because oral cancer and other oral neoplasms are relatively rare, it is important to have slides reviewed by pathologists with experience in diagnosing disease in the head and neck region. Given the gravity of a diagnosis of oral cancer, validation is warranted.

Other diseases, such as infectious diseases and autoimmune diseases, may present with oral lesions similar to oral cancer. Ask about symptoms that can rule in or out these other diseases if the initial history is suggestive of pathology other than cancer. The patient's history should be reviewed for exposures, constitutional symptoms, or systemic manifestations. An acute onset, presence of fever or chills, rapid swelling, and signs of acute inflammation are suggestive of an infectious process. A chronic process with dermatologic findings, arthralgias, or multiple organ system involvement is suspicious for an autoimmune disorder.

The clinician should take the time to take a thorough past medical history. The history may provide additional information helpful for stratifying a patient's risk for developing oral cancer. The past medical history may provide the etiology of the

disease. Inquire about a history of dysplasia, immunocompromised states, other types of cancer, and a history of head and neck radiation.

A history of tobacco and alcohol use should be determined. Ask about cigarette usage in addition to other forms of tobacco use, such as snuff, cigar, or pipe tobacco. The quantity of tobacco and length of time used should be recorded. An accurate alcohol history is often difficult to gather, as most people underestimate their use. It is helpful to corroborate history with family and friends. It is also helpful to inquire about betel nut use, particularly in patients of Asian or South Asian descent. One should inquire about environmental exposures at work and home. A history of dental neglect should be recorded.

The focused physical examination

The focused examination of the oral and maxillofacial pathology patient should always include an examination of the neck, the face, the salivary glands, and the oral cavity. Examination of the eyes, ear, nose, pharynx, larynx, and cranial nerves should be performed as directed by the patient's presenting symptoms and the clinician's differential diagnosis. The following section will briefly describe some of the more critical aspects of the physical examination. However, it is beyond the scope of this chapter to fully detail all the nuances of the physical exam of the head and neck.

Neck

The focused exam should begin with a careful examination of the neck. All levels of the neck should be palpated for lymphadenopathy. Fig. 30.1 illustrates the different levels of the neck. However, the actual sensitivity of physical exam for detecting metastatic nodes is low.⁶ Regardless, this provides the basis for clinical staging and subsequent treatment planning. Neck swellings that are palpable should be described by location, size, tenderness to palpation, temperature, compressibility, texture, fluctuance, induration, whether the swelling is freely mobile in vertical and horizontal planes or is fixed to underlying tissues, and whether there are changes in the overlying skin. In certain cases, transillumination may aid in determining whether the lesion is fluid filled. If in the midline, the swelling should be evaluated while the patient is swallowing. Swellings that rise and fall with swallowing are likely thyroglossal duct cysts. Swellings that are pulsatile, have a palpable thrill, or have a bruit on auscultation, may be indicative of a vascular lesion.

Thyroid gland

The thyroid gland is located inferiorly, in the anterior compartment of the neck, situated just inferior to the level of cricoid cartilage. The gland is comprised of

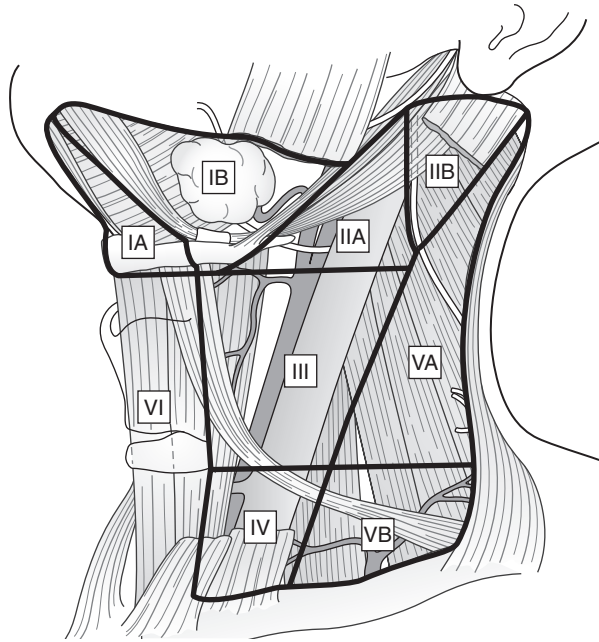


Fig. 30.1 Illustration of the levels and sublevels of the neck. Notice that level II is divided into IIA and IIB by the spinal accessory nerve and level V is divided into VA and VB by a horizontal plane from the inferior border of the cricoid arch. Illustration from Cheng and Schmidt, used with permission.

two lobes that extend laterally and are connected medially by a central isthmus. Occasionally, there is a third pyramidal lobe that extends superiorly along the midline. Examination of the thyroid gland can be performed from behind or facing the patient. The exam is performed one lobe at a time. One hand is used to stabilize the lobe contralateral to the one being examined by placing the fingers of the opposite hand firmly into the contralateral tracheal–esophageal groove. The examining hand then palpates the lobe of interest, taking note of size, texture, and nodularity. The exam is then repeated on the contralateral lobe. A normal thyroid gland may be difficult to discern, especially in patients with significant soft tissue redundancy. As mentioned above, having the patient drink water while palpating the gland can help with defining its location, as the thyroid gland should elevate with swallowing.

Face

The skin of the scalp, neck, and face should be examined carefully for lesions or masses. Hair should be parted in order to adequately inspect the scalp. Any asymmetries in the face should be noted. The facial skeleton should be palpated for expansion or tenderness.

The temporomandibular joint should also be assessed. This can be performed by palpating the preauricular area while asking the patient to open and close. Any tenderness to palpation is abnormal and should be documented. The examiner should note any clicks, popping, crepitus, or tenderness, and whether the findings are unilateral or contralateral.

The examiner should also make note of the timing of the findings, whether they occur with jaw opening, closing, or both, and whether they are early or late along the arc of opening or closing. The examiner should ask the patient to move their lower jaw to the right, to the left, and forward, to evaluate the patient's excursive and protrusive function. If there are any limitations in movement, the examiner should record the maximal incisal opening and maximum range of excursive and protrusive movements. Normal maximal incisal opening is considered to be in the range 53–58 mm. Neoplasms of the temporomandibular joint, ramus of the mandible, and lateral pharyngeal space can all cause limitation of movement and pain, although temporomandibular disorders are more frequent causes for these symptoms.

Parotid and submandibular glands

The parotid and submandibular glands should be evaluated for nodules, swelling, tenderness, warmth, erythema, or changes in the overlying skin. It is important to remember that the tail of the parotid extends to a level inferior to the angle of the mandible and swelling of the deep lobe of the parotid may manifest as a mass in the lateral pharyngeal wall, so examination of these areas is necessary. There are several lymph nodes located within and around the parotid tissue that receive lymphatic drainage from the skin of the upper face, mid face, and parts of the scalp. While palpating, be sure also to palpate for the pre-auricular and retroauricular nodes.

Eye

When indicated, the eyes should be examined for symmetry, pupillary reflex, and function of extraocular muscles. The palpebra, conjunctiva, and lacrimal ducts should be inspected. Dry and injected conjunctiva is a finding common to several autoimmune syndromes. Any discrepancy with alignment of the eyes should be documented. Tumor involvement of the maxillary sinus may cause vertical dystopia, as shown in Fig. 30.2. Involvement of the posterior orbit can also manifest as exophthalmos due to mass effect. If excessive epiphora is present, this should also be recorded. If the recent history includes a complaint of change in vision or ocular pain, a fundoscopic exam should be performed to rule out intraocular pathology.

Ear

If the patient's presentation includes symptoms of otalgia, hearing changes, vertigo, ear "stiffness", facial paralysis, or unexplained facial pain, an examination of the ear should be performed. Ear stiffness is a common presenting complaint in patients with nasopharyngeal carcinoma, which is of particular concern among patients of Chinese descent. The clini-



Fig. 30.2 A 2-year-old boy with an aggressive poorly differentiated spindle cell tumor. Notice the vertical dystopia and increased scleral show caused by elevation of the left orbit due to tumor mass effect.

cian should keep this in mind when a Chinese patient complains of epistaxis and ear stuffiness. The examination of the ear begins with careful inspection of the auricle, as auricular neoplasms can be easily missed in the posterior auricular area or within the concha or antihelix. The mastoid should be evaluated for signs of mastoiditis, a complication of otologic infections.

An otoscope should be used to examine the external auditory meatus, tympanic membrane, and middle ear. With the clinician facing the ear to be examined, the posterior hand should grasp the posterior–superior pinna and retract in the posterior direction. This maneuver straightens the external auditory meatus. The otoscope is carefully inserted into the canal with the anterior hand, resting on the patient's ipsilateral shoulder as a fulcrum. Cerumen disimpaction may be necessary for adequate visualization of the proximal ear. Many otoscopes allow the magnifying lens to be removed so that a curette may be introduced into the canal for removal of cerumen. Great care should be taken when doing this as the external auditory meatus is richly innervated.

If the chief complaint includes ipsilateral facial paralysis, the auricle and external auditory meatus should be inspected for vesicles or ulcers. Herpes zoster or herpes simplex of the ear is related to Ramsay-Hunt syndrome, a cause of Bell's palsy. In otitis externa, gentle traction of the auricle can be quite painful. The external auditory meatus can be quite edematous and inflamed. The tympanic membrane should be inspected carefully for perforation or fluid (which may be serous, cloudy, or bloody) behind the membrane. These may be signs of otitis media. However serous otitis may also be a sign of eustachian tube obstruction from tumor involvement. Weber and Rinne tests should be performed if indicated by a complaint of hearing loss or stuffiness.

Nasal cavity and nasopharynx

If the patient is complaining of epistaxis or nasal stuffiness, a nasal examination may be indicated. A nasal speculum with a good light source can be used to examine the anterior nasal cavity. Attention should be paid to the mucosa of the lateral and medial walls. Any ulcers, masses, or lesions should be noted. Kiesselbach's plexus, which is the anastomosis of the anterior ethmoidal, greater palatine, sphenopalatine, and superior labial arteries, is the most common site for epistaxis. Allergic rhinitis is typified by pale, boggy, and edematous mucosa. Infectious sinusitis manifests as erythematous edematous mucosa.

If examination of the posterior nasal cavity or nasopharynx is required, as is the case when there is clinical suspicion of nasal or nasopharyngeal carcinoma, this can be performed using endoscopy under local anesthesia. Following administration of local anesthesia and a decongestant spray, with the patient in a sitting position with chin lifted slightly upward, a flexible or rigid endoscope may be introduced along the floor of the nasal cavity. The examiner should make the effort to visualize the nasal septum, inferior turbinate, and opening of the eustachian tube with this initial pass. The endoscope is then repositioned above the inferior turbinate so that the middle turbinate comes into view. To examine the nasopharynx, a flexible endoscope is passed along the floor of the nasal cavity posteriorly and angled inferiorly into the nasopharynx, taking care to fully visualize both the right and left pharyngeal walls by rotating the scope. The nasopharynx may also be indirectly visualized with adequate lighting by placing a hand mirror into the oropharynx, posterior to the soft palate. Contact with the tongue base should be avoided. Heating the mirror with warm water can help prevent fogging of the reflective surface.

Oral cavity and oropharynx

When examining the oral cavity, the buccal mucosa, vestibular mucosa, alveolar mucosa and gingiva, the palatal mucosa, lingual mucosa, the floor of mouth, and all borders of the tongue should be inspected both visually as well as by palpation. If the patient is wearing any removable dental prostheses, these should always be removed prior to inspection. Special attention should be paid to inspecting the posterior lateral tongue and the floor of mouth as they are the two most common sites for oral cancer. When examining the oral tongue, it may be necessary to grasp the tongue with a square of gauze and retract it anteriorly in order to fully visualize the posterior–lateral aspect of the tongue. Tongue and floor of mouth mobility should be assessed. Any tethering of the tongue or floor of mouth should be noted and may be indicative of a neoplastic process. The floor of mouth should be palpated bimanually, particularly in the lingual gutter. Examination of the maxilla and mandible should

be done by bimanual palpation to assess for swelling or cortical expansion. The characteristics of any swelling should be described. If glandular pathology is suspected, salivary function should also be assessed. This can be performed by using gauze to dry the mucous membranes around the parotid duct or submandibular duct, and then palpating the gland to promote salivation. The quantity and character of the salivary flow should be noted.

As an oral and maxillofacial surgeon, one should also take a moment to do a cursory dental exam, making note of any teeth with questionable prognoses. If radiation therapy is to be considered, then extraction of compromised teeth could be performed at the same time as the ablative surgery to prevent delays in treatment. It is important to measure the maximal incisal opening, as well as make note of the Angle's classification for the patient's occlusion, the position of the maxillary and mandibular dental midlines, and reproducibility of the patient's occlusion. This information will play a critical role in the reconstructive phase of any surgical treatment for malignancy provided.

The oropharynx is best examined directly by retracting the tongue anteriorly using tongue blades or gauze under adequate lighting. The tongue can be moved to bring the lateral aspects of the posterior third of the tongue into view. Palpation is used to assess this area for potential lesions. While retracting the tongue, visually inspect the soft palate, anterior and posterior tonsillar pillars, and uvula. A dental mirror can then be used to indirectly examine the lateral pharyngeal walls, the posterior pharyngeal walls, and the vallecula.

Larynx

An examination of the larynx is indicated if the patient presents with neck swelling concerning for nodal metastasis with no observable primary lesion or the patient who presents with changes in their voice, dysphagia, odynophagia, or airway obstruction. The examination can be done indirectly, using a dental mirror placed in the oropharynx, or directly with an endoscope.

To perform an indirect exam, have the patient sit in the exam chair, leaning forward slightly with chin tilted upward to allow for adequate illumination of the posterior oropharynx. As with examining the oropharynx, the tongue is retracted by grasping the anterior two thirds firmly with gauze. A warmed dental mirror is placed posteriorly with the backside gently retracting the uvula and soft palate superiorly. If the patient has a strong gag reflex, it is advisable to spray the soft palate with a topical anesthetic before performing the exam. Once the mirror is in place, it can be adjusted to bring the larynx into view.

A direct exam with a flexible endoscope provides improved visualization if the equipment is available. Similar to nasal endoscopy, after local anesthesia is

administered and the patient is positioned properly in a sitting position leaning slightly forward, a flexible scope is passed along the floor of the nasal cavity. The tip of the endoscope is placed above the epiglottis, from which the larynx should come into view.

The epiglottis, aryepiglottic folds, false vocal cords, ventricles, true vocal cords, and arytenoids should be studied for erythema, masses, or ulcerations. In addition, the true vocal cords should be examined while the patient is quietly breathing, breathing heavily, and phonating to assess for symmetry of cord adduction and abduction. If there is any incompetence of the vocal cords, such that the folds do not fully adduct, this should be noted.

The subglottic area can be viewed through the aditus while the true vocal cords are abducted. However, for a complete exam of the subglottic area, endoscopy under general anesthesia may be required.

Cranial nerves

A gross cranial nerve exam should be performed if the patient complains of any sensory or motor deficit. As mentioned above, any sign of paresthesia is highly suspicious of perineural invasion. For the initial consultation, the cranial nerve exam of the oral and maxillofacial pathology patient typically focuses on the sensory components of the trigeminal nerve and the motor components of the facial nerve. The distributions of the trigeminal nerve should be stimulated, looking for paresthesia or dysesthesia that can be localized. Similarly, the different branches of the facial nerve (temporal, zygomatic, buccal, marginal mandibular, and cervical) should be assessed to attempt to localize the nerve involvement. Details of the gross cranial nerve exam are described in Table 30.1.

In performing a focused history and physical exam for the head and neck pathology patient, it is essential for each clinician to establish a defined order so they can perform the history and physical exam systematically, in order to avoid inadvertently omitting any part. Also, the clinician should keep in mind the old adage that one cannot see what one does not know, and always keep considering the differential diagnosis. As soon as the patient-doctor interaction begins, the experienced clinician is already formulating a differential diagnosis and testing his or her hypotheses. This evolving differential is what drives the nature of further examinations and testing. Tables 30.2 and 30.3 outline the differential diagnosis for lesions within the oral cavity.

Conventional oral examination as a screening tool

The purpose of a screening tool is to detect disease in patients who are asymptomatic in the hopes of improving prognosis by early intervention. In order to be effective as a screening tool for oral cancer, the

Table 30.1 Focused examination of the cranial nerves.

	Cranial nerve	Examination
I	Olfactory nerve	Occlude one nostril, test the patency, then have the patient identify distinct smells, such as coffee or peppermint
II	Optic nerve	Check visual acuity of each eye separately using Snellen chart Check afferent limb of pupillary reflex by briefly shining a light into each pupil Perform the swinging light test to check for the consensual reflex
III	Oculomotor nerve	Check full range of extraocular muscle movements by having the patient follow your finger to six positions: right, right and up, right and down, left, left and up, left and down The oculomotor nerve is responsible for superior medial, superior lateral, medial, and inferior lateral gaze Check efferent limb of pupillary reflex as above
IV	Trochlear nerve	Check full range of extraocular muscle movements as above The trochlear nerve is responsible for inferior medial gaze
V	Trigeminal nerve	Test for sensation of the face in the three main branches of the trigeminal nerve by lightly touching the skin on each side of the face with the patient's eyes closed
VI	Abducens nerve	Check full range of extraocular muscle movements as above The abducens nerve is responsible for lateral gaze
VII	Facial nerve	Test for symmetrical activity of the muscles of facial expression in the upper, middle, and lower face Have the patient raise the eyebrows, tightly close the eyes, smile, and grimace
VIII	Vestibulocochlear nerve	Test gross hearing by gently rubbing strands of hair between your fingers adjacent to each ear and asking the patient if it is audible Vestibular function can be tested by performing the Dix-Hallpike test or Romberg test
IX	Glossopharyngeal nerve	Test for function of the stylopharyngeus muscle by having the patient phonate "Ah" and watch for symmetrical elevation of the soft palate and uvula
X	Vagus nerve	Test the function of the palatal and pharyngeal muscles along with testing the glossopharyngeal nerve, as described above
XI	Accessory nerve	Test the spinal accessory nerve by asking the patient to turn the head left and right against resistance and shrug the shoulders against resistance
XII	Hypoglossal nerve	Test by having the patient protrude the tongue and watching for deviation Have the patient press the tongue against each cheek against resistance from your hand against the patient's cheek

method must be sensitive enough to detect most tumors and find them early enough so that intervention improves survival. Furthermore, the tool must be specific enough that patients without cancer would not be subjected to unnecessary invasive testing and treatment. The screening tool should be safe and simple to perform. Finally, it is desirable that the screening tool leads to a reduction in mortality in the population that the tool is intended to target.

The conventional oral examination is the standard tool for screening for oral cancer and is the only such tool that has been properly studied for this purpose. The advantages of the conventional oral examination are that it can be easily performed by non-specialists, it is inexpensive, and it is non-invasive.

However, conventional oral examination effectiveness in detecting cancer when used by the generalist and its ability to reduce mortality in the population is controversial. Several studies do support its use. Jullien *et al.*, in a large oral cancer screening program that enrolled 2300 patients, demonstrated that general dentists were able to effectively identify suspicious lesions (defined as red, white, or ulcerative lesions of 2 weeks' duration) with a sensitivity and specificity of 74 and 99% respectively when compared to oral cancer specialists.⁷ The same group

performed a review of the literature, which included four studies evaluating dental auxiliaries as screeners, and found sensitivities and specificities ranging from 73–92% and 93–98% respectively.⁸ It is important to note that the lesions in the above studies were not confirmed by biopsies to be malignancies.

In terms of its ability to reduce mortality in the population, one large cluster-randomized controlled trial in Kerala, India, which enrolled 96517 patients over the course of 9 years, demonstrated an earlier stage at time of diagnosis and improved 3-year survival;^{9,10} as well as showing improved mortality rates in high-risk groups, namely male tobacco and alcohol users.¹¹ Although this study has been criticized for not addressing lead time bias in its analysis and not meeting standards set by the Cochrane Collaboration for cluster-randomized trials, its significance in demonstrating survival benefits by the use of a simple tool in an at-need population cannot be overlooked.

Although conventional oral examinations are effective in detecting suspicious lesions, only a minority of these lesions will ultimately turn out to be cancer. On the other hand, mucosa that appears clinically normal may actually have microscopic evidence of neoplasia. Thomson found that 36% of the patients in

Table 30.2 Differential diagnosis of oral lesions by appearance of lesion. The lesions are listed in order of frequency. The locations in the oral cavity are listed in order of frequency.

Diagnosis	Location in oral cavity
White lesions that can be wiped off	
White coated tongue	Dorsal tongue
Pseudomembranous candidiasis	Buccal mucosa, tongue, palate
Frictional hyperkeratosis	Buccal mucosa, lips, tongue
Thermal injury	Lip, palate, buccal mucosa
Chemical injury	Buccal mucosa, gingiva
Secondary syphilis	Tongue, lip, buccal mucosa, palate
Diphtheria	Oropharynx
White lesions that cannot be wiped off	
Linea alba	Bilateral buccal mucosa
Leukoedema	Bilateral buccal mucosa
Leukoplakia	Buccal mucosa, lip, gingiva, tongue, floor of mouth
Snuff dipper's pouch keratosis	Mandibular vestibule
Actinic keratosis	Lower lip
Lichen planus	Bilateral buccal mucosa, tongue, gingiva, palate, lip
Frictional hyperkeratosis	Buccal mucosa, lip, tongue
Nicotine stomatitis	Hard palate
Hairy leukoplakia	Lateral tongue
Hyperplastic candidiasis	Buccal mucosa
Lupus erythematosus	Buccal mucosa, palate, gingiva
Submucous fibrosis	Bilateral buccal mucosa
White sponge nevus	Bilateral buccal mucosa, tongue, lip, palate, gingiva, floor of mouth
Tertiary syphilis	Palate, tongue
White and red lesions	
Erythema migrans (geographic tongue)	Tongue
Erosive candidiasis	Buccal mucosa, tongue, palate
Erosive lichen planus	Bilateral buccal mucosa, tongue, gingiva, palate, lip
Erythroleukoplakia	Buccal mucosa, lip, gingiva, tongue, floor of mouth
Lupus erythematosus	Buccal mucosa, palate, gingiva
Scarlet fever	Tongue, pharynx
Red lesions	
Viral pharyngitis	Oropharynx
Bacterial pharyngitis	Oropharynx
Denture stomatitis	Palate
Erythematous candidiasis	Tongue, palate, alveolar mucosa
Erythroplakia	Buccal mucosa, lip, gingiva, tongue, floor of mouth
Atrophic glossitis	Tongue
Hemangioma	Any oral site
Plasma cell gingivitis	Gingiva
Blue, brown, black or pigmented lesions	
Ethnic pigmentation	Gingiva, palate
Varices	Floor of mouth, tongue, lips, buccal mucosa
Petechiae or ecchymosis	Any oral site
Amalgam tattoo	Gingiva, buccal mucosa
Mucocele or mucous retention cyst	Lower lip, buccal mucosa, ventral tongue, floor of mouth
Eruption cyst	Gingiva
Hairy tongue	Dorsal tongue
Hemangioma	Any oral site
Ranula	Floor of mouth
Melanotic macule	Lower lip
Smoker's melanosis	Gingiva
Melanocytic nevus	Any oral site
Blue nevus	Palate, any oral site
Malignant melanoma	Palate, gingiva

Adapted from Neville, Damm, Allen and Bouquot, *Oral and Maxillofacial Pathology*, 3rd edn. Saunders, Philadelphia, PA, 2009.

Table 30.3 Working differential diagnosis of oral lesions by location within the oral cavity and neck. The lesions are listed in order of frequency.

Ulcers of the tongue	Hodgkin's lymphoma Teratoma
Squamous cell carcinoma Traumatic ulcer Tuberculosis Primary syphilis Deep fungal infections	Congenital/developmental Thyroglossal duct cyst Dermoid cyst Epidermoid cyst Plunging ranula Laryngocele
Painful oral ulcer	Inflammatory Bacterial adenitis Viral adenitis Cat scratch disease (<i>Bartonella henselae</i>) Tuberculosis (scrofula)
Palatal mass	Mass in posterior neck
Pleomorphic adenoma Mucoepidermoid carcinoma Polymorphous low-grade adenocarcinoma Neurofibroma Schwannoma Non-Hodgkin's lymphoma Kaposi's sarcoma	Neoplastic Metastatic cancer – Posterior: nasopharynx and scalp primaries – Supraclavicular: gastric, pancreatic, lung primaries Non-Hodgkin's lymphoma Hodgkin's lymphoma Thyroid cancer
Parotid mass	Congenital/developmental Lymphangioma
Pleomorphic adenoma Warthin's tumor Mucoepidermoid carcinoma Adenoid cystic carcinoma Lymphoma Lipoma Mumps Bacterial parotitis Viral parotitis	Inflammatory Bacterial adenitis Viral adenitis Sialadenitis Cat-scratch disease (<i>Bartonella henselae</i>) Scrofula (tuberculosis)
Mass in lateral neck	Lips
Neoplastic Metastatic cancer – Upper jugular and jugulodigastric: oral cavity, oropharynx and laryngeal primaries – Lower jugular: hypopharynx and thyroid primaries – Submandibular gland: oral cavity, nasal, paranasal sinuses, and facial skin primaries Carotid body tumor Non-Hodgkin's lymphoma Hodgkin's lymphoma Hemangioma Schwannoma Salivary gland neoplasm	Dry vermillion Actinic changes Squamous cell carcinoma Keratoacanthoma
Congenital/developmental Branchial cleft cyst Thymic cyst Laryngocele Lymphangioma Hemangioma Congenital muscular torticollis Sialadenopathy	Wet mucosal surface Squamous cell carcinoma Minor salivary gland tumors
Inflammatory Bacterial adenitis Viral adenitis Sialadenitis Cat-scratch disease (<i>Bartonella henselae</i>) Scrofula (tuberculosis)	Buccal mucosa
Mass in the midline and anterior neck	Squamous cell carcinoma Minor salivary gland tumors Hyperkeratosis Lichen planus Proliferative verrucous leukoplakia
Neoplastic Thyroid cancer Benign thyroid neoplasm Non-Hodgkin's lymphoma	Floor of mouth
	Squamous cell carcinoma Sublingual gland tumors
	Hard palate in the area of the greater palatine foramen
	Mucoepidermoid carcinoma Adenoid cystic carcinoma Pleomorphic adenoma
	Retromolar trigone
	Squamous cell carcinoma Mucoepidermoid carcinoma Adenoid cystic carcinoma
	Soft palate
	Squamous cell carcinoma Dysplasia Squamous papilloma Minor salivary gland tumors

his study, who had been diagnosed with oral cancer, also had biopsy-proven dysplasia or cancer in the same anatomic location on the contralateral side, despite having normal appearing mucosa.¹² Thus, conventional oral examinations may lead to an increased amount of negative biopsies and miss some potentially malignant lesions.

Despite this, it is the authors' opinion that the advantages of routinely performing head and neck examinations on every patient at every visit greatly outweigh the drawbacks. It can be effortlessly and quickly performed, and has been demonstrated to be cost effective.^{13,14} Although it may detect many lesions that are benign, this low positive predictive value is acceptable given the minimal cost and morbidity of tissue biopsy in the oral cavity. In recent years, dental organizations have actively promoted the use of head and neck exams in oral cancer screening. Anecdotally, this has had a significant effect on the senior author's clinical practice. The majority of patients referred to the Department of Oral and Maxillofacial Surgery at the University of California, San Francisco diagnosed with oral cancer have been from providers within the dental community. Perhaps as a result of this, the authors have observed that patients present to their clinic at much earlier stages than reported in the literature. Holmes *et al.*¹⁵ and Bae and Hirsch¹⁶ have echoed this experience.

Imaging studies

Once the initial history and physical exam are completed, the next step in the clinical evaluation is often the ordering of imaging studies. There are a number of modalities available to the oral and maxillofacial surgeon, each with their own strengths, limitations, and varying degrees of invasiveness. This section aims to briefly highlight some of the advantages and disadvantages of each of these tools. As with any study, it is important for the surgeon requesting the imaging to have specific questions to be answered. When requesting imaging, the surgeon aims to: (1) better characterize the location, composition, and behavior of the lesion to assist in a diagnosis; (2) delineate the full extent of the lesion and its proximity to adjacent vital structures to aid in prognosticating as well as treatment planning; and (3) in the case of malignant disease, determine whether there is metastatic spread to the neck lymphatics.

Plain radiographs

Plain radiographs are two-dimensional images created by projecting X-rays through a patient on to a film or digital sensor. These include cephalometric radiographs, chest radiographs, dental panoramic tomograms, and dental radiographs. In general, there is very little utility in the use of cephalometric radiographs to evaluate oral and maxillofacial pathology. Tumors are usually quite extensive before they are

visible on cephalometric films. Furthermore, the anatomic extent and tumor composition are better visualized using other imaging techniques. Soft tissues are poorly imaged with these studies. Cephalometric films are not useful for detecting nodal metastasis. However, they still may be of some use when planning reconstruction, for presurgical preparation of rigid fixation plates, and for postoperative evaluation of hard tissue reconstruction.

A chest radiograph to screen for metastasis or synchronous lung primaries is part of the standard comprehensive evaluation of a patient diagnosed with head and neck cancer. Many of the risk factors that predispose a patient to developing oral cancer are also risk factors for other pathology. As many as 10% of oral cancer patients have chest metastases and 5% of patients have synchronous lung carcinomas that are detected on chest imaging.¹⁷

Dental radiographs, such as periapical, bitewing, and occlusal films, are sometimes helpful in the evaluation of lesions of the dentate segments of the mandible and maxilla. Although limited by the size of the area being visualized, the resolution of the images is far greater than other modalities. Dental radiographs are particularly useful in evaluating smaller odontogenic lesions as they can better show key features that help distinguish different odontogenic tumors or cysts from each other. These details include association with teeth, cortication, and presence of calcifications within the lesion. Furthermore, using Clark's rule (SLOB: same – lingual, opposite – buccal) is a quick and inexpensive way to determine a lesion's location in a buccal–lingual dimension.

The dental panoramic tomogram (orthopantomogram or Panorex) is a film that is commonly used by dentists and oral and maxillofacial surgeons. Much like other dental radiographs, it is quick and inexpensive. It also has the advantage of being able to effectively image the maxilla and mandible. For these reasons, it is routinely part of the initial assessment of a patient presenting with pathology of the oral and maxillofacial region, and can be very helpful in diagnosing tumors and cysts of the jaws. They may or may not be associated with teeth. The location of the lesion also helps narrow the differential. Any lesion that is located inferior to the inferior alveolar nerve canal is almost certainly not odontogenic in origin. The panoramic tomogram can also be used to look for erosion of bone by malignant soft tissue tumors, although its sensitivity for this is less than that of computed tomography (CT) or magnetic resonance imaging (MRI). It is particularly useful in treatment planning for lesions of the mandible.

Computed tomography

In 1972, Godfrey Hounsfield invented CT, a giant step forward in imaging diagnosis. By using computers to mathematically assemble multiple X-ray projections, CT is able to create cross-sectional images of the

human body. CT, therefore, has the great advantage of being able to eliminate the obscuring of deep structures by superficial structures. It allows the surgeon to appreciate a tumor's location and size in three dimensions, thus accurately defining a tumor's relationship to important vital structures, such as the great vessels, skull base, and orbit. In addition, CT is far superior in appreciating differences in soft tissue planes and vasculature. Fat, muscle, and water can be differentiated fairly easily. This ability is further enhanced by contrast. This has also allowed surgeons to screen for nodal metastasis with reasonable sensitivity.

There are a few disadvantages associated with the use of CT. Although the cost of a CT scan is decreasing as accessibility increases, the study still remains expensive. The effective dose of radiation from a head and neck CT is relatively small when compared to annual exposure to natural radiation. But, at around 1000–4000 μSv , it is not insignificant.^{18,19} Furthermore, this is still a greater effective dose than that of plain radiography, and may be of concern to some patients, especially when serial CT scans are required. Another drawback of the use of CT in the maxillofacial region is the obscuring of the image from artifacts arising from high-density materials such as dental filling or surgical hardware. Oral cancer most commonly affects older patients, who are more likely to have dental restorations that make CT imaging difficult.

Since 1972, several advances in CT technology have been introduced that have made CT faster, yield images with improved resolution, and have made it more accessible. Recently, the cone-beam CT is an iteration of the technology that has been increasingly adopted for use in the oral and maxillofacial region, in particular for use in prosthodontic and orthodontic treatment planning, but also for use in diagnosing pathology as well as surgical and radiotherapy treatment planning.^{20,21} Cone-beam CT uses an X-ray beam in the shape of a cone that rotates and scans the entire volume of interest, as opposed to a conventional fan-beam CT, which uses a fan-shaped X-ray that rotates around and advances along the volume of interest. Because of this, cone-beam CT decreases the effective dose to roughly around 23–52 μSv .^{22,23} In addition, cone-beam CT is less susceptible to dental artifacts. The spatial accuracy is comparable to conventional CT, with its relative geometric error measured in tenths of a millimeter.²⁴ However, some of the drawbacks of the cone-beam CT are its inferior soft tissue visualization, which limits its use in diagnostics for oral and maxillofacial pathology and staging of the neck, and that it is limited in the total volume that can be visualized with a single scan.

Another recent advance in surgical planning has been the use of conventional CT to create stereolithic models and customized surgical reconstruction plates. The authors have used these custom tools to simplify and expedite the surgical reconstruction,

especially when reconstructing mandibular defects. Although custom reconstruction plates certainly make the surgeon's work easier, they are an additional expense and whether they result in a clinically significant improvement in the surgical outcome remains to be studied.

Magnetic resonance imaging

MRI is a technology that is based upon the science of nuclear magnetic resonance. In simple terms, the system works by placing a patient in a magnetic field that aligns the protons of hydrogen atoms (most commonly found in water) in the direction of the field. A second magnetic field is then momentarily introduced that causes protons to be deflected from this axis and, in the process, absorb some of its energy. Once this second field is released, the protons that were previously deflected return to the initial axis of the first magnetic field and release the absorbed energy as a radiofrequency. The radiofrequencies are then detected by the MRI scanner and are used to determine the position of the respective protons. Several of these additional fields are used along the volume of interest and the information is compiled into a three-dimensional image that can be viewed in sections. Gadolinium, a paramagnetic material, is used to enhance areas of inflammation or increased vascularity.

As with CT, the MRI creates spatial imaging that allows the surgeon to visualize a tumor in three dimensions and appreciate the tumor's relationship to adjacent structures. In addition, MRI has several advantages over CT. It has superior soft tissue resolution, and, as such, is the preferred imaging of choice of neurologic pathology. It is minimally affected by artifacts from high-density materials. MRI also does not use ionizing radiation and is considered non-invasive. In addition, the use of certain protocols, namely magnetic resonance angiography (MRA), allows for the imaging and detection of blood flow, which is useful in the preoperative assessment of vessels for microvascular reconstruction.

As the technology uses a strong magnetic field, implanted paramagnetic materials, such as pacemakers, cochlear implants, deep brain stimulators, or older vascular aneurysm clips, are an absolute contraindication for MRI. These can cause motion and thermal injury to surrounding tissues. MRI scanners are much slower than CT scanners, with scanning times up to 40 minutes on older machines. Because of this, motion artifacts are a problem and patients who are unable to lie still (such as children) may require anesthesia to have an MRI. Furthermore, patients who suffer from claustrophobia may also find undergoing an MRI difficult.

MRI is the anatomic imaging study that is preferred by the authors for evaluating patients with oral cancer. It has superior soft tissue imaging when compared to CT, which is helpful for determining extent

of soft tissue invasion as well as nodal metastasis, and is not plagued by dental artifacts.

Positron emission tomography and PET/CT

Positron emission tomography (PET) is an imaging modality that highlights areas of the body with increased metabolic activity. Radionuclides are bound to molecules that are important to human metabolism. The most common tracer used in PET is ^{18}F -fluoro-2-deoxy-D-glucose (FDG), which is a radioactively labeled glucose. FDG is administered intravenously and becomes sequestered in cells that are actively taking up glucose. Malignant cells that are more mitotically and metabolically active would then have relatively greater uptake than surrounding non-malignant cells. Once sequestered in cells, the FDG undergoes radioactive decay to a more stable molecule, emitting positrons in the process. These positrons combine with electrons within the adjacent tissues and annihilate, resulting in a release of gamma rays 180 degrees from each other. These gamma rays are then picked up by detectors within the PET scanner and triangulated to determine the anatomic location of the metabolically active tissue.

PET is effectively used in the detection of distant metastases in cancers for which metastasis is common, such as cancers of the lung and colon. In addition, PET scans may be used to evaluate the patient with nodal metastasis and an unknown primary. The detection rates for unknown primaries are greater than MRI or CT, at 29% compared to 15–20%.^{25–27} However, these rates are still relatively low. On occasion, PET may also detect synchronous primaries in other locations.

One of the limitations of PET is the lack of anatomic detail displayed by the images. In order to remedy this, computer software registration has been developed to combine the biochemical imaging of PET with the anatomic detail afforded by CT. The combination of these two modalities is called PET/CT. Initially, this technique was quite labor-intensive, as technicians would have to manually combine images from different scanners taken at different time points. However, integrated PET/CT scanners have simplified the task of obtaining these composite images and have improved consistency and reliability of producing quality images.

The use of PET/CT for evaluation of lymph node metastases and recurrence of tumors is well established.²⁸ In addition, it has been studied for use in evaluating the clinically node negative neck. PET/CT is superior to CT or MRI for detecting occult lymph node metastases, although the difference may not be clinically significant.^{29–31} Nahmias *et al.* found that the use of PET/CT did not change the decision to perform an elective neck dissection in the patients studied.³⁰ Because of this, it is generally not recommended as part of the initial workup of head and neck cancer.

For head and neck cancer, the indications for PET are: (1) part of the assessment for recurrence in symptomatic patients or in patients with suspicious but non-specific findings on MRI or CT; (2) part of the assessment of a patient with an unknown primary; and (3) staging for patients with advanced cancers. In the senior author's practice, PET scans are not routinely used in the initial workup of a patient with oral and maxillofacial pathology.

Ultrasound

Ultrasound images are created from the recording of the amplitudes of reflected high-frequency sound waves. The primary advantage of this tool is that anatomy can be visualized dynamically in real time by the sonographer. Other advantages are that ultrasound is extremely safe and relatively inexpensive.

By varying the frequency of ultrasound used, the sonographer can also image objects of varying depths. Higher frequency ranges, 5–10 MHz, are used in the head and neck to visualize more superficial soft tissue structures. Calcifications are also well imaged by ultrasound, which is useful in diagnosing sialoliths, phleboliths, and atherosclerosis. However, tissues containing air and bone reflect nearly all ultrasounds. Thus anatomy deep to bony structures, such as the angle of the mandible, or posterior to air-filled objects, such as the trachea, are not well visualized. In addition to the standard gray-scale ultrasound images obtained, Doppler ultrasound records shifts in reflected frequencies to image moving objects. Clinically, this allows for the imaging of blood flow.

Ultrasound has several indications. It is useful in evaluating superficial nodules of the neck, salivary glands, and thyroid gland. Ultrasound is very effective in identifying cystic lesions, which appear as well defined hypoechoic (dark) masses. Benign tumors of the salivary and thyroid glands are similarly well defined, lobular, and hypoechoic. Lesions that are less well defined are suspicious for malignancy and should prompt the clinician to order additional studies.

Ultrasound is also highly effective in the evaluation of the lymphatic system of the neck, able to determine the presence, quantity, size, shape, and internal characteristics of lymphadenopathy. Most of the lymphatic chains clinically relevant to oral cancer are superficial and are well imaged by ultrasound. One exception is the retropharyngeal lymph nodes that can be involved with cancers of the pharynx and larynx. Because ultrasound allows for dynamic imaging, it is used by cytopathologists for guiding fine-needle aspiration biopsy (FNAB) of smaller, impalpable lesions.

Some practices have adopted the use of ultrasound to serially monitor and expectantly manage patients with clinically node negative (N0) necks.³² Although one group has published on the utility of this method, other groups have not replicated their success.^{6,33}

Part of the problem is that the reliability of this technique is highly dependent on the experience and skill of the sonographer in this specific application. As such, the authors do not use ultrasound for expectant management of the N0 neck.

Considerations for radiation exposure

With the exception of ultrasound and MRI, the imaging tools mentioned above expose patients and providers to varying degrees of radiation. The estimated effective doses for common imaging studies are listed in Table 30.4. Although the dosages from dental or head and neck radiography are hundredths of lethal doses and the risk of tissue injury or carcinogenesis is exceedingly small, the risk still exists. Therefore, imaging should not be haphazardly ordered. The risk of harm to the patient should always be weighed against the potential benefit from the information collected.

However, the risk of radiation exposure is not an absolute contraindication. One scenario that is exceedingly common is the pregnant patient with oral and maxillofacial pathology. It is important to

remember that the American College of Obstetricians and Gynecologists has published guidelines for the ordering of imaging in pregnant patients.³⁴ These are listed in Tables 30.5 and 30.6.

Obtaining a tissue diagnosis

Much information can be gathered from taking the history, performing the physical exam, and acquiring the appropriate imaging studies. However, histologic analysis is absolutely essential for a definitive diagnosis of oral pathology. For suspicious lesions of the soft or hard tissues of the oral cavity, a tissue biopsy is the sole method to obtain this.

There is often reluctance, mostly on the part of the practitioner, to perform biopsies. Deferring a biopsy is a reasonable approach if the clinical features of the lesion are consistent with a process that is entirely benign and is not treated by excision. However, if the lesion does not respond to the appropriate intervention for the initial diagnosis, a biopsy is absolutely indicated. Therefore, it is critical for the surgeon, if the plan is to treat empirically, that the patient be closely observed and followed. It is also crucial for the surgeon to be able to formulate a working differential diagnosis, in order to guide the decision of whether to perform a biopsy or not. Failure to perform a biopsy or make a referral for a biopsy due to a clinician's lack of experience with treating oral lesions is inexcusable. If a malignant disease or disease that is treated by wide excision is on the differential diagno-

Table 30.4 Effective dosages of radiation for common radiographic studies.

Study	Effective dose (μSv)
Full mouth dental radiography (20 films) ^a	17
Dental panoramic tomogram ^b	26
Cephalometric plain films ^c	10–30
Conebeam CT of maxilla and mandible ^d	23–52
CT of the maxilla	104 ^e –1202 ^f
CT of the mandible	761 ^e –3324 ^f
Abdominal plain film (kidney–ureter–bladder) ^c	530–700
Abdominal CT ^c	10 000
Chest plain film (posterior–anterior) ^c	20
Chest plain film (lateral) ^c	40
Chest CT ^c	8000
Annual exposure to natural radiation	3000

^a White SC. Assessment of radiation risks from dental radiography. *Dentomaxillofac Radiol* 1992; 21: 118.
^b Frederiksen NL, et al. Effective dose and risk assessment from film tomography used for dental implant diagnostics. *Dentomaxillofac Radiol* 1994; 23: 123.
^c Wall BF, et al. Revised radiation doses for typical X-ray examinations. *Br J Radiol* 1997; 70: 437–9.
^d Ludlow JB, et al. Dosimetry of two extraoral direct digital imaging devices: NewTom cone beam CT and Orthophos Plus DS panoramic unit. *Dentomaxillofac Radiol* 2003; 32: 229–34.
^e Frederiksen NL, et al. Effective dose and risk assessment from computed tomography of the maxillofacial complex. *Dentomaxillofac Radiol* 1995; 24: 55.
^f Scaf G, et al. Dosimetry and cost of imaging osseointegrated implants with film-based and computed tomography. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1997; 83: 41.
Adapted from White and Pharaoh, *Oral Radiology: Principles and Interpretation*. Mosby, St Louis, MO, 2009.

Table 30.5 American College of Obstetrics and Gynecology guidelines^a on the diagnostic imaging in the pregnant patient.

1. Women should be counseled that X-ray exposure from a single diagnostic procedure does not result in harmful fetal effects. Specifically, exposure to less than 5 rad has not been associated with an increase in fetal anomalies or pregnancy loss.
2. Concern about possible effects of high-dose ionizing radiation exposure should not prevent medically indicated diagnostic X-ray procedures from being performed on a pregnant woman. During pregnancy, other imaging procedures not associated with ionizing radiation (e.g. ultrasonography, MRI) should be considered instead of X-rays when appropriate.
3. Ultrasonography and MRI are not associated with known adverse fetal effects.
4. Consultation with an expert in dosimetry calculation may be helpful in calculating estimated fetal dose when multiple diagnostic X-rays are performed on a pregnant patient.
5. The use of radioactive isotopes of iodine is contraindicated for therapeutic use during pregnancy.
6. Radiopaque and paramagnetic contrast agents are unlikely to cause harm and may be of diagnostic benefit, but these agents should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

^a ACOG Committee Opinion. Number 299, September 2004 (replaces No. 158, September 1995). Guidelines for diagnostic imaging during pregnancy. *Obstet Gynecol* 2004; 104: 647–51.

Table 30.6 Fetal exposure due to common radiographic imaging studies as reported by the American College of Obstetrics and Gynecology (ACOG)^a. Note that current ACOG guidelines recommend that fetal exposure be kept under 5000 mrad.

Study	Fetal exposure (mrad)
Chest plain film (two views)	0.02–0.07
Abdominal film	100
Intravenous pyelography	≥1000
Hip film	200
Mammography	7–20
Barium enema or small bowel series	2000–4000
CT of head or chest	<1000
CT of abdomen or lumbar spine	3500
CT pelvimetry	250

^a ACOG Committee Opinion. Number 299, September 2004 (replaces No. 158, September 1995). Guidelines for diagnostic imaging during pregnancy. *Obstet Gynecol* 2004; 104: 647–51.

sis, than a biopsy should be performed. Or, if the initial impression consists of a disease whose treatment is morbid, such as long-term corticosteroid use for autoimmune disease, than a biopsy is necessary to confirm that diagnosis.

Once the decision is made to perform a biopsy, the surgeon must determine: (1) whether the biopsy should be excisional or incisional; (2) what is the setting that the biopsy should be performed under; (3) what type of instrumentation should be used; and (4) decide where the biopsy specimen should be taken from.

Excisional and incisional biopsy

For an excisional biopsy, the entire lesion is removed. This approach is indicated if the suspicious lesion is likely to be benign, likely to be cured by local excision, and small enough that local excision is feasible. For an incisional biopsy, only a sample of representative tissue is taken from the patient, leaving the rest of the lesion *in situ*. This is indicated if the lesion is suspicious for malignancy or the area involved is larger than can be excised.

Deciding between an excisional biopsy and an incisional biopsy should be relatively simple. If a surgeon is seriously considering a malignant lesion as part of the differential diagnosis, even if it is not the most likely diagnosis, an incisional biopsy should be performed. If the surgeon is only considering benign diseases on the differential diagnosis and the lesion is localized, an excisional biopsy should be performed.

However, it is important to remember that the primary advantage of performing an excisional biopsy over an incisional biopsy is that, with excisional biopsies, benign lesions can be both diagnosed and treated with a single procedure. Unfortunately, the drawback of performing excisional biopsies is

quite significant. If the surgeon mistakenly excises an oral lesion that is malignant, that surgeon has obscured the margins of resection for the definitive treatment. Unless the oncologic surgeon is the same surgeon performing the biopsy, it will be difficult for the oncologic surgeon to appreciate the original size and extent of the lesion. The ultimate consequence of excision of a malignant lesion is that the ability to adequately perform an oncologic resection is greatly compromised. Thus, it is safer to perform an incisional biopsy if one is uncertain.

Setting for performing the biopsy

Asking the pathologist for a histologic diagnosis is the same as asking any other specialist for a consultation. The request needs to be a specific question framed in an accurate and thorough clinical picture. The first half of the required information is the history, clinical exam, and the surgeon's impression. The second half is providing the pathologist with an adequate specimen to analyze and perform the necessary tests. In other words, it is critical for the surgeon to work very closely with the pathologist to maximize the team's ability to come to an accurate diagnosis.

An often overlooked step in ensuring an adequate biopsy is choosing the appropriate setting for performing the biopsy. Many lesions are amenable to biopsy in the clinic. These include lesions of the skin, lesions of the lip, and mucosal lesions of the oral cavity. However, large lesions of the oral cavity that extend into the oropharynx or oral lesions with an intraosseous component may be more efficiently sampled in the operating room under general anesthesia. Having the patient under general anesthesia affords the surgeon the opportunity to take the time to adequately expose and visualize the lesion and sample enough tissue in enough locations, without being limited by concern for keeping an awake patient comfortable. In addition, if the plan is to perform an excisional biopsy, frozen section analysis of incisional biopsy specimens can be performed initially to verify that a lesion is, in fact, benign and not in need of an oncologic resection.

Too frequently, the desire to minimize the morbidity from a large biopsy or to avoid the hassles of scheduling time in the operating room compromises the adequacy of the biopsy specimen. If the surgeon does not feel confident that the lesion can be comfortably and thoroughly sampled in the clinic, there should be no hesitation to do so under general anesthesia. A misdiagnosis or delay in diagnosis is much more troublesome than the short postanesthesia recovery period from a proper biopsy.

Instrumentation for performing the biopsy

A biopsy should be done with a sharp cutting instrument. A scalpel, fine scissors, a biopsy punch, or a combination of these can be used. The authors feel

that electrocautery or lasers do not have a place in the biopsy armamentarium. The use of electrocautery will destroy the tissue structure along the edges of the biopsy specimen. This not only reduces the available tissue for analysis, but it may obscure important prognostic markers such as margin status and depth of invasion. The use of a laser for vaporization does not leave any specimen for pathologic analysis.

The punch biopsy is particularly useful in sampling friable lesions. In these lesions, it may be difficult to adequately grab the tissue, as it often crumbles under the pressure of the tissue forceps. A punch can be used to cut a cylindrical piece with downward pressure, and in the same motion can be scooped out to cut the base and deliver the specimen. In addition, by driving the biopsy punch to the hub of the cutting blade, it is quite simple to obtain a specimen of an adequate depth.

Deciding where to sample with the biopsy

When performing an incisional biopsy, much depends on selecting a representative sample of the lesion to be diagnosed. The portion of the lesion that yields the most information will vary depending on the nature of the lesion and what is suspected to be the most likely diagnosis.

Local anesthesia should involve nerve blocks and local infiltration around the lesion of interest. Take care not to administer anesthetic directly into the lesion itself so as to not distort the tissue architecture.

In ulcerative lesions that are suspicious for malignancy, the biopsy should be performed on the ulcer itself, near the edge of the ulcer. Normal mucosa does not need to be removed, as it does not provide any meaningful information. The tissue near the edge of the ulcer will give a cellular picture that is least clouded by tissue necrosis or inflammation. The biopsy should have a minimum depth of 5 mm to enable the pathologist to view the depth of invasion. Ulcers that are suspicious for an infectious process are performed in the same way.

If the ulcerative lesion more closely fits an autoimmune process, a biopsy of the normal tissue should be performed. In vesiculobullous diseases, such as pemphigus vulgaris or erythema multiforme, the tissue from the ulcer itself does not provide helpful information, as it is the epithelium that is affected and ulcers are denuded of epithelium. Furthermore, the ulcer may be colonized by oral flora that can cloud the histologic picture. The surgeon should take more than one biopsy specimen so that one can be processed and stained with the standard hematoxylin and eosin stains and the other can be processed for direct immunofluorescence.

Biopsies in the oral cavity do not necessarily require closure, particularly if it is an incisional biopsy. However, if the surgeon is performing an excisional biopsy, and the incisions and tissues facilitate this, closure can help with hemostasis, comfort, and

improved healing. Biopsies of the lip are amenable to this and can be facilitated if the incisions are made vertically in the shape of an ellipse. When closing larger tongue excisions, the surgeon should take care to not tether the tongue to the floor of mouth. It may be better to allow the tongue to heal by secondary intention or to close the wound with a skin graft.

The differential diagnosis for intraosseous lesions includes odontogenic cysts or tumors, non-odontogenic cysts or tumors, fibro-osseous diseases, epithelial neoplasms that have eroded bone, vascular lesions, infectious processes, and metastatic diseases. Before an intraosseous lesion is accessed, it is advisable to rule out a vascular lesion, as cutting into a high-flow vascular malformation could lead to catastrophic hemorrhaging. Exam findings, such as a pulsatile lesion, audible bruits, or palpable thrills would be suggestive of a vascular lesion. Often there are associated soft tissue changes. The overlying mucosa may appear expansile and blue in color. In high-flow lesions affecting the dentoalveolar segments, there is frequently gingival hypertrophy with bleeding from the gingiva sulci. The increased vascular flow may also cause ipsilateral maxillary or mandibular hypertrophy. A Doppler examination may also be helpful in making this determination. If the lesion is likely a vascular malformation, then the lesion should be embolized by interventional radiology before surgical excision. Regardless, it is recommended that intraosseous lesions that cannot be ruled out as vascular be aspirated before they are entered. If blood is aspirated, the syringe should be removed from the needle. Pulsatile flow from the hub of the needle is consistent with a high-flow vascular malformation. In this situation, the needle should be withdrawn and pressure should be applied to achieve hemostasis. Blood that is not pulsatile and stops quickly, or aspirate that is non-bloody are signs of a lesion other than a vascular lesion.

Basic surgical principles apply when accessing the lesion of interest. When planning the incision, the design should allow for a closure that lies over solid bone and not a cavity. The length of the incision should allow for adequate retraction to fully visualize the extent of the lesion. If necessary, incorporate releasing incisions. If the lesion involves the mucosa, the affected mucosa should be sampled along with the bony lesion.

Once the area involved is visualized, a window in the bone may be created if the lesion has not already perforated the bone cortex. This will allow the lesion to be seen grossly. If the lesion is cystic, take note of the nature of the cystic contents, documenting the consistency, color, and whether it is characteristic for certain pathology. Generously sample the cyst lining. If the lesion is solid or solid with cystic components, sample tissue from the most central part of the lesion.

At the University of California, San Francisco, it is a common practice, if the lesion is obviously cystic, to marsupialize the cyst and place a drain that starts

within the deepest portion of the cystic space and exits through the soft tissue flap into the oral cavity. In this way, if the lesion ultimately is diagnosed as either a keratocystic odontogenic tumor (formerly known as odontogenic keratocyst) or a dentigerous cyst, it has also been simultaneously treated.

When an excisional biopsy or resection is performed, the specimen must be properly oriented with sutures. Two sutures in different dimensions, along with a proper description including location and laterality, allow the pathologist to orient the specimen. The surgeon's description should also include the lesion characteristics, such as condition of the overlying mucosa, texture, size, consistency and infiltration. For osseous lesions include a radiograph. If the margins are positive for atypical cells then proper orientation and a detailed description allow the surgeon to accurately perform definitive surgical excision.

Fine-needle aspiration biopsy

Fine-needle aspiration biopsy (FNAB) is a technique in which a fine needle is passed into a mass, cells are aspirated, and these cells are then prepared on slides and analyzed by a cytopathologist. The use of aspiration biopsy has been practiced for well over a century. Initially, the technique involved the use of a large-bore needle under local anesthesia, but it has since evolved to be used with fine-gauge needles. At most institutions, this is performed by specialized cytopathologists. Having a physician experienced in performing this procedure and diagnosing diseases of the head and neck is essential, as it can be difficult to obtain an adequate sample and make the correct diagnosis based upon cytology.

Indications

FNAB can be used as the method for initial diagnosis for almost any mass in the head and neck region, particularly in disease processes that are not clearly infectious. The differential diagnoses for neck masses are outlined in Tables 30.3 and 30.7. This technique is

helpful in distinguishing neoplastic processes from those that are inflammatory or reactive. However, the most common indication for the use of FNAB is a mass located in the neck, thyroid gland, or salivary glands. Fig. 30.3 shows an algorithm for the evaluation of a patient with a neck mass.

There are no absolute contraindications to FNAB. Relative contraindications include any medical comorbidities that would preclude any minor surgical procedure, in particular significant coagulopathies or thrombocytopenia. In addition, lesions that are suspicious for high-flow vascular malformations should not be subjected to FNAB. Masses overlying or underneath important anatomic structures, such as carotid body tumors, may be difficult to sample. In the past, there has been concern over tumor seeding along the track of the fine needle. However, tumor seeding has not been a problem with this procedure.³⁵

Technique

The procedure should begin by proper positioning of the patient. This may require the use of a shoulder roll to assist in extension of the neck or rotating the head of the patient to expose the side of interest. The area to be biopsied should be then cleaned with an alcohol swab. The area to be biopsied can be anesthetized with local anesthetic, however this is usually not necessary as the anesthetic needle is the same gauge as the biopsy needle. Have an empty syringe ready with a 23–27 gauge needle attached. The plunger should be drawn back to allow 1 ml of air in the syringe.

The mass is then palpated and stabilized with the non-dominant hand. The needle is then introduced into the mass. The patient should be instructed to not move or swallow while the needle is inserted. The plunger is pulled back from the syringe to create a vacuum. The needle should be deliberately pushed in and out of the mass with short 2–3 mm vibratory strokes. If the mass is cystic, the fluid should be allowed to drain first. To do this, first insert the needle with the syringe with the plunger removed. After draining, the needle can be used to sample any remaining solid material or the cyst lining. Once the biopsy specimen has entered the needle, the needle can be withdrawn. This process should be repeated a minimum of four times.

The aspirate is then deposited on to two separate slides. One slide should be air dried while the other slide is fixed. The second slide can be wet-fixed with 95% ethanol or spray-fixed. The needle is then rinsed with balanced salt solution.

If the person performing the FNAB is not the cytopathologist that will be reviewing the specimen, then it is critical that adequate documentation is sent with the biopsy. As mentioned above, the documentation should include the patient's information, relevant history, description of clinical findings, description of

Table 30.7 Differential diagnoses of neck masses stratified by age.

0–15 years
Inflammatory
Congenital/developmental
Neoplastic (malignant > benign)
16–40 years
Inflammatory
Congenital/developmental
Neoplastic (benign > malignant)
>40 years
Neoplastic (malignant > benign)
Inflammatory
Congenital/developmental

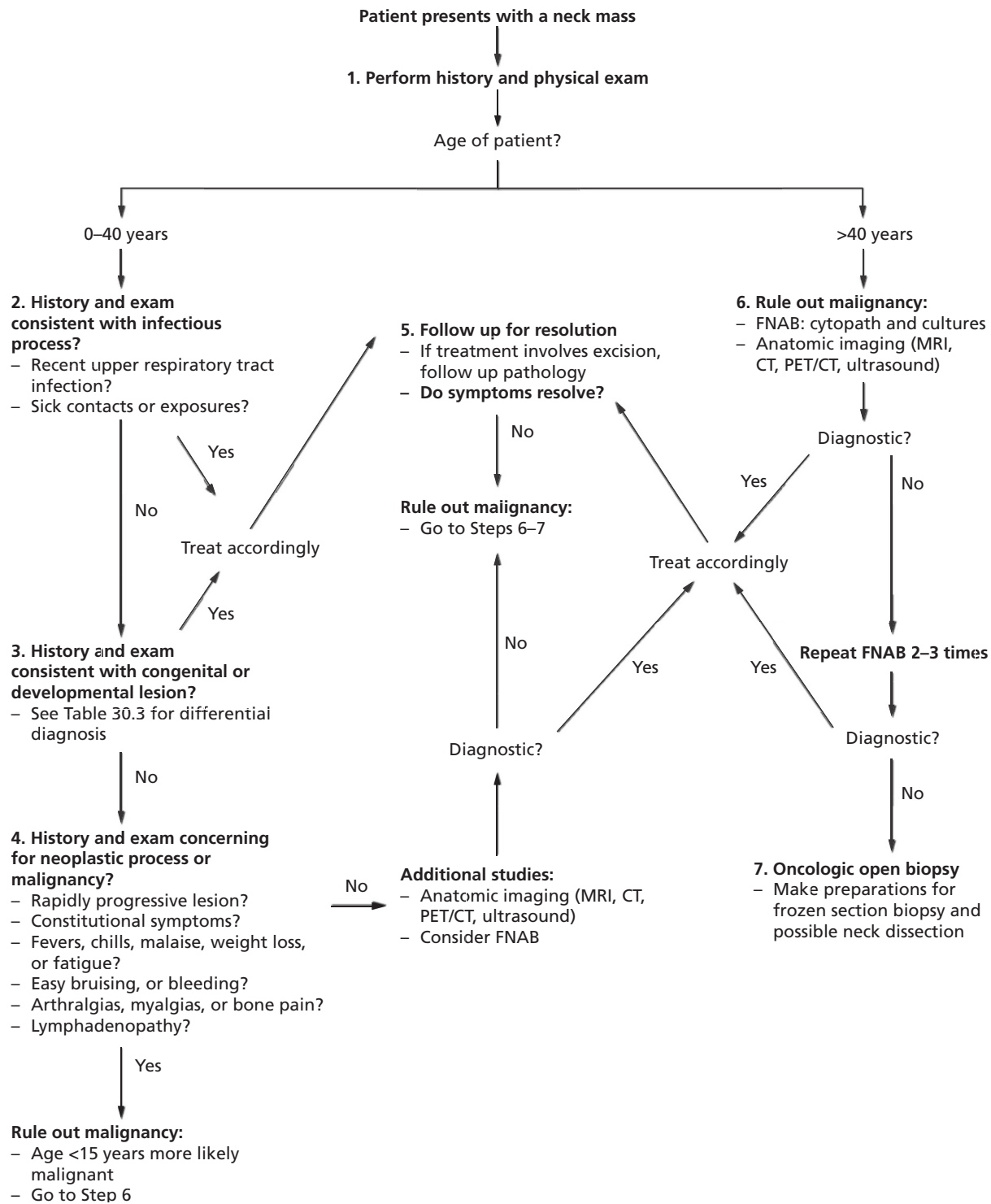


Fig. 30.3 Algorithm for the evaluation of neck masses.

the biopsy procedure, and the surgeon's clinical impressions. The description of the biopsy procedure should include the number of needle passes, consistency of the mass being biopsied, volume of fluid aspirated, and the characteristics of the aspirate.

Lymph nodes

Lymphadenopathy is a common presentation of disease among patients with head and neck pathology. As discussed above, the differential diagnosis

includes metastatic diseases, reactive lymphadenopathy, lymphoma, and inflammatory processes, both infectious and non-infectious. In patients older than 40 years of age, lymphadenopathy should be considered malignancy until proven otherwise. Features that are suggestive of malignancy include a chronic history, unexplained etiology, lymphadenopathy that is fixed to overlying or underlying tissues, induration, and lack of findings more consistent with inflammatory processes, such as redness, fluctuance, warmth, or purulent drainage. The presence or his-

tory of head and neck malignancy is highly concerning for metastatic or recurrent disease.

FNAB is the part of the initial diagnostic workup of lymphadenopathy. One of the primary indications for performing FNAB is to decipher the presence of metastatic disease or lymphoma. Several studies have demonstrated that FNAB can determine a specific diagnosis for lymphadenopathy 82–96% of the time.^{36–38} FNAB is especially useful for distinguishing cystic lesions from inflammatory lesions and benign lesions from malignant lesions. In this way, FNAB can be a cost-effective, minimally invasive means for evaluating neck masses in patients for whom the provider has a low index of suspicion for malignancy.

FNAB is particularly useful in diagnosis of metastatic disease or recurrent lymphoma. However, the utility for FNAB in the initial diagnosis of lymphoma is controversial. The use of several analytical techniques such as flow cytometry to detect specific cell surface antigens, immunophenotyping of immunoglobulins, and cytogenetics to detect specific chromosomal rearrangements can increase the diagnostic power of this technique.^{39–41} Despite this, if non-Hodgkin's lymphoma is suspected, an open biopsy remains the definitive diagnostic modality as it provides more ample tissue to perform the above studies.

If the lymphadenopathy is suggestive of infectious processes, the aspirate should be sent for microbiologic testing. Bacterial, mycobacterial, fungal, and viral organisms can all manifest as lymphadenopathy.

On occasion, metastatic disease can manifest as lymphadenopathy in the absence of an obvious primary tumor. The diagnosis of neck disease with an unknown primary should be followed with anatomic imaging and panendoscopy with biopsies.

A negative result from FNAB of lymphadenopathy, like all diagnostic tests, should be interpreted in context of the clinical picture. If the history or exam is at all concerning and the clinical suspicion for malignancy remains high, the surgeon should push the diagnostic evaluation further, including repeat FNAB, panendoscopy, and open excisional biopsy of the lymphadenopathy.

Salivary glands

FNAB is part of the initial evaluation of swellings of the major salivary glands that cannot be ruled out as neoplastic in etiology. The benefit of using FNAB in diagnosis of salivary gland masses is that the thin caliber of the needle avoids injury to critical related structures, namely the facial nerve that is intimately associated with the parotid gland. FNAB is able to effectively distinguish between salivary gland neoplasms, soft tissue neoplasms, parotid lymph nodes, lymphoepithelial cysts, and sialadenitis with good sensitivity and specificity, 90% and 80% respective-

ly.^{42,43} More importantly, it is helpful in distinguishing benign lesions from malignant salivary gland neoplasm and lymphomas, a distinction that would alter treatment.

Like all of pathology, the quality of results depends a great deal on the experience and skill of the cytopathologist. The differential diagnosis for salivary gland masses, as already mentioned, is quite broad. In addition to the potential diagnoses already mentioned, diseases of adjacent tissues may mimic an intraparotid lesion. Secondary alterations of tissues surrounding a disease process, such as chronic inflammation, cystic changes, clear cell changes, sebaceous differentiation, mucin production, and oncocytic changes can further confuse the picture. Finally, because FNAB samples cells rather than tissues, diagnoses may be extremely difficult to distinguish from each other. For example, a pleomorphic adenoma may manifest with plasmacytoid and lymphocyte-like myoepithelial cells, a finding that can easily be confused for a plasmacytoma or malignant lymphoma.⁴⁴ As such, it is critical that the surgeon is able to communicate well with the pathologist and be able to interpret the pathology report to make certain that it is consistent with the rest of the patient's clinical picture.

Some authors have argued that FNAB of salivary glands is an unnecessary step in the workup of parotid masses, given that the majority (80%) are benign and 80% of these masses are pleomorphic adenomas. Some surgeons argue that the majority of lesions, as well as several malignant lesions, may be diagnosed and treated simultaneously by a superficial parotidectomy. The minority of malignant neoplasms that would require more extensive treatment would be able to be distinguished by a careful history and examination.⁴⁵ Although this logic is valid, it is the practice within the authors' department to take a more cautious approach. Given that undergoing FNAB is well tolerated, fairly quick, and minimally invasive, it does not seem justified to proceed to major surgery without a tissue diagnosis. Anecdotally, there have been a small number of patients in the senior author's practice whose examinations were deceiving, and ultimately benefited from having a tissue diagnosis prior to surgery.

Thyroid nodules

In the management of thyroid nodules, FNAB has been shown to be a highly accurate and useful tool, decreasing the number of unnecessary surgeries and increasing the rate of diagnosis of thyroid malignancies in several studies.⁴⁶ It has been recommended as part of the initial workup of a thyroid nodule.^{47,48} The indications for FNAB of the thyroid are: (1) a solitary thyroid nodule; (2) a multinodular goiter with a growing nodule; and (3) new nodules in patients with Hashimoto's thyroiditis.³⁵ Once performed, the cytopathologist will typically report the findings as

unsatisfactory, benign, indeterminate, suspicious, or malignant. The findings for these reports are 17% for unsatisfactory, 69% for benign, 10% for indeterminate or suspicious, and 4% for malignant.⁴⁷ FNAB is limited in its ability to distinguish follicular adenomas and Hurthle cell adenomas from their malignant counterparts, as this distinction can only be made by identifying invasion in a tissue sample. Otherwise, the sensitivity and specificity for diagnosing thyroid malignancy has been reported as 83% and 92% respectively. False-negatives can occur 1–5% of the time and are commonly associated with aspiration of cysts greater than 3 cm or nodules less than 1 cm.^{35,49} False-positives can occur around 1% of the time, usually associated with inflammatory thyroid diseases, such as Grave's disease or Hashimoto's thyroiditis, or a toxic thyroid nodule.

A FNAB that is reported as unsatisfactory should be repeated. Similarly, indeterminate biopsies should also be either repeated or followed with a more definitive biopsy, as 25% of these are malignant.⁴⁷ Benign nodules can be excised, followed expectantly, or treated medically if appropriate. Only 1% of benign nodules are ultimately cancer. Suspicious nodules should undergo further workup, as 20% of these are cancer.³⁵ The workup can include additional imaging, such as a radionuclide scan, or excisional biopsy. Radionuclide scans that are cold should be diagnosed surgically. However, given that the majority of nodules are cold, the utility of the scan might be questioned. Malignant lesions should be treated with oncologic resection, with chemotherapy and radiation therapy as dictated by the specific diagnosis.

Limitations

In addition to the indications mentioned above, some institutions have published reports of the use of FNAB to diagnose benign intraosseous pathology of the maxilla and mandible. Specifically, FNAB has been used by some to definitively diagnose odontogenic tumors such as ameloblastoma.^{50,51} Generally speaking, the primary question that FNAB intends to answer is whether a lesion is likely to be malignant or benign. In some instances, the cytologist may be able to determine the predominant cell type of the aspirate. However, definitive diagnoses of odontogenic tumors of the neck require tissue that can only be obtained by surgical biopsies. Having a definitive diagnosis is critical as the surgical treatment can vary from a simple enucleation to wide resection. Unfortunately, only a paucity of case reports support the use of FNAB for diagnosis of odontogenic tumors. Therefore, the authors do not recommend the use of FNAB for this indication.

Open biopsy for lymphadenopathy

An open lymph node biopsy is indicated when FNAB is suggestive of lymphoma, has ruled out metastatic

disease, or is inconclusive after multiple attempts. Proper diagnosis of lymphoma requires sampling of tissue, as mentioned above. In the case of non-specific or inconclusive findings, obtaining adequate tissue facilitates the pathologist's ability to reach the correct diagnosis.

It is important to remember that open biopsy for lymphadenopathy should not be part of the initial evaluation of the neck swelling. Inadvertent violation of fascial planes by open biopsy of a metastatic node risks seeding of malignant cells throughout the neck and has been demonstrated to significantly increase disease-related mortality.⁵² Although some recent studies have challenged this dogma, suggesting that inappropriate open biopsy may not be as poor a prognosticator if followed by a radical neck dissection and radiation therapy, it is generally regarded to be below the standard of care to proceed to open biopsy without first obtaining an FNAB.^{53,54}

When performing an open biopsy for lymphadenopathy, the surgeon should approach the surgery as if performing a neck dissection by maintaining fascial planes and removing the fibroadipose-lymphatic tissue in continuity. Once removed, the surgeon should have the pathologist examine frozen sections to confirm that the diagnosis is not metastatic head and neck cancer. The surgeon should be prepared to perform a completion neck dissection if the frozen sections reveal that the lymphadenopathy is indeed metastatic cancer.

Once the lymph node is appropriately removed, the tissue should be submitted for testing related to the differential diagnosis. Cultures and sensitivities are sent if the disease is potentially infectious in etiology. The specimen should be sent for pathology to rule in or out metastatic disease. If the pathology is suggestive of lymphoma, additional studies will be required.

Summary of the approach to the oral and maxillofacial pathology patient

The evaluation of the oral and maxillofacial pathology patient begins with a focused history and physical examination. An experienced clinician will develop a differential diagnosis early on and will test and refine his or her hypotheses as they interview and examine the patient. Imaging studies are selected and ordered to answer specific questions and based upon specific indications, as each type of study has its strengths and weaknesses. If a neoplastic process is suspected, it should be confirmed with a tissue diagnosis. FNAB is extremely helpful as a first-line evaluation tool for neck or salivary gland masses. For most diseases, a surgical biopsy is necessary for a definitive diagnosis. If a neoplastic disease is suspected, the clinician should not hesitate to perform a biopsy or refer to an appropriate specialist. Fig. 30.4 shows a basic algorithm for evaluation of the oral and maxillofacial pathology patient.

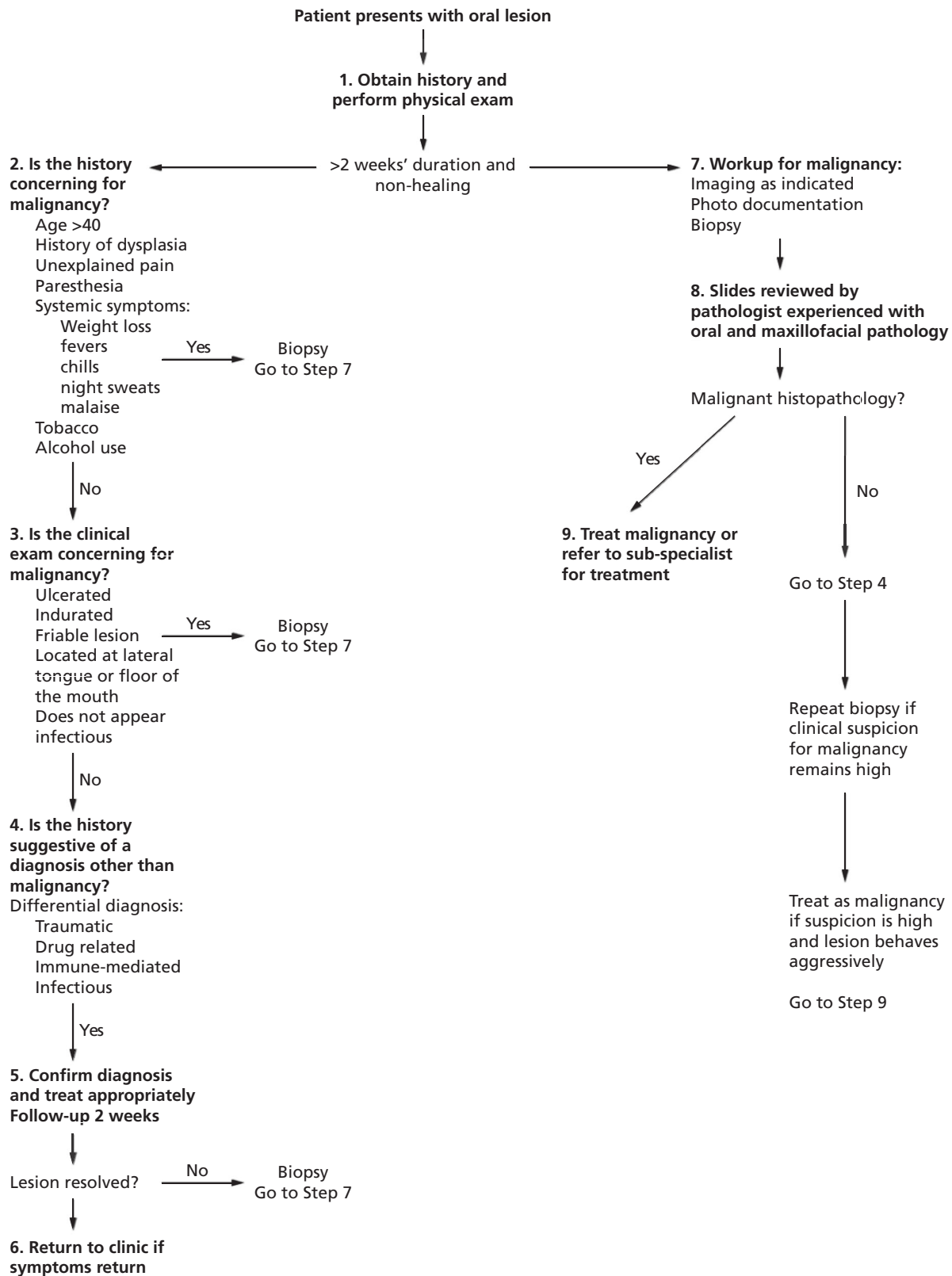


Fig. 30.4 Algorithm for evaluation of the oral and maxillofacial pathology patient.

Adjunctive diagnostic tools

Many premalignant and malignant lesions are small and are difficult to appreciate. Even when they are visualized, providers who do not frequently deal with diagnosis of diseases of the oral and maxillofa-

cial region are, understandably, reticent to aggressively biopsy all suspicious lesions. Both of these scenarios are especially true when the lesions are relatively innocuous appearing. As a result, it is common for the generalist to dismiss more serious diseases from the differential diagnosis, such as oral

cancer, because they are rare or the patient's demographics do not necessarily fit the disease. Unfortunately, this is a fallacy, as it has been demonstrated that innocuous lesions can indeed be cancer and that there is an increasing incidence of oral cancer in younger adults who have never used tobacco or abused alcohol.⁵⁵

Therefore, the ability to perform a minimally invasive test to detect inconspicuous lesions or determine the malignant potential of borderline suspicious lesions would be a highly attractive tool. The following section will discuss the most commonly available adjunctive diagnostic tools that have been used to fill this niche, the relevant literature evaluating their utility, and the authors' opinions regarding their places in the oral and maxillofacial pathology workup.

Toluidine blue

Toluidine blue is a vital dye that stains nuclear material. As a consequence, it preferentially stains tissues with high rates of cellular turnover, namely neoplastic mucosa.

Technique

To perform the stain, have the patient first rinse his or her mouth with 1% acetic acid for 20–30 seconds. Following this, the patient should rinse with water twice, again for 20–30 seconds each time. Next, have the patient rinse with 1% toluidine blue solution. Follow this with 20–30 seconds of rinsing with 1% acetic acid and 20 seconds of rinsing with water.

Dark blue staining indicates a positive result, pictured in Fig. 30.5, which represents the dye binding to nuclear material. Pale blue staining is more representative of benign lesions, which show no nuclear stains on histology. The papillae of the dorsal tongue also tend to retain toluidine blue.

Indications

Toluidine blue has been demonstrated to be an effective adjunctive screening tool in identifying premalignant lesions or oral cancer recurrences in high-risk populations, particularly those who have already been diagnosed with having oral dysplasia or

oral cancer. Gray *et al.* published an extensive review of the literature evaluating the use of toluidine blue as an adjunctive screening tool.⁵⁶ The 14 studies included in the review examined the use of toluidine blue in aiding specialists in detecting lesions that were not readily visible with a conventional oral examination. Overall, the sensitivity for detecting oral cancer ranged from 40–100% and the specificity from 31–92%. From this series, Barrellier *et al.* found six oral cancers that were not detected by conventional oral examination alone, supporting its use as an aid to the conventional oral examination for detecting lesions.⁵⁷ However, although highly sensitive for detecting oral cancer, it has its limitations. Warnakulasuriya and Johnson noted that 50% of oral lichen planus lesions stained positively with toluidine blue, highlighting its potential for false-positives.⁵⁸ In addition, Martin *et al.* found that only 42% of dysplasias stained positively, underscoring an important limitation in the dye's abilities in lesion detection.⁵⁹ Therefore, the surgeon must be careful not to overextend the utility of this tool. Although the tool is highly sensitive, the literature does not support the use of toluidine blue to rule out malignancy.

Recent studies have also suggested that toluidine blue might be used to predict degree of risk for transformation in oral dysplasia. Zhang *et al.* found that toluidine blue preferentially stained lesions that had high-risk histologic features. More importantly, it predicted risk of transformation in lesions with no histologic features of dysplasia and the staining was correlated with patient outcome.⁶⁰ However, the degree of staining can be subjective, and thus should not be the determining factor for not performing a biopsy.

In addition to the above indications, the use of toluidine blue has been also been suggested as a potential screening tool. Unfortunately, there is currently no evidence to support the use of toluidine blue to screen for oral cancer in the general population.

Tissue fluorescence

Autofluorescence is a property of cells whereby specific wavelengths of light are absorbed by molecules within cells, causing them to move into an excited state. As the molecules return back to their resting state, the energy from the excitation is released in the

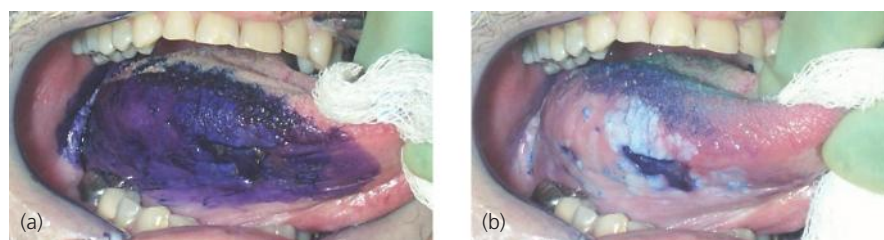


Fig. 30.5 (a) Staining of the oral tongue by toluidine blue prior to decolorization. (b) Tongue stained with toluidine blue after decolorization. The area of dysplasia retains the vital stain and remains dark blue. Note that the filiform papilla of the dorsal tongue stains a pale blue.

form of fluorescence emissions. There are several types of molecules that exhibit autofluorescence, but most commonly exist in the mitochondria and lysosomes of the cell. Porphyrins in erythrocytes also contribute to autofluorescence.

It has been observed that cancer cells have different autofluorescence emission patterns than normal tissues.⁶¹ This has led to the development of technologies that aim to highlight the differences in autofluorescence in order to aid in detection of pathologic oral lesions.^{62–76}

VELscope is an example of an adaptation of this technology that uses a handheld device that shines high-intensity blue excitation light (400–460 nm wavelengths) into the oral cavity while the provider can visualize the different fluorescence patterns. The following discussion will focus on the VELscope, pictured in Fig. 30.6.

Technique

After performing a thorough history and physical exam under normal incandescent lighting, repeat the intraoral examination while shining the VELscope light into the oral cavity. Keep the light approximately 5 cm away from the entrance of the oral cavity to maximize the area to be illuminated. While shining the light, examine the entire oral cavity through the filter that is attached to the handheld light, taking care to retract the tongue to view the lateral tongue and floor of mouth.

Under the VELscope's blue light, normal tissue fluoresces green. Malignant or dysplastic tissues will appear dark, as pictured in Fig. 30.7. However, normal tissue can be dark as well. Attached mucosa, such as the gingiva, is typically darker, as are the tonsillar pillars. Inflammation will also appear darker under the VELscope. On the other hand, hyperkeratosis appears brighter. To differentiate normal from

pathologic, malignant or dysplastic lesions are generally much darker than the aforementioned normal tissues, sharply demarcated, and are in areas at higher risk for oral cancer.

Areas that are suspicious should be palpated. Blanching of tissues by applying pressure will cause areas of inflammation that appear dark to fluoresce like normal tissue.

It is important to correlate what is seen under the VELscope with the conventional oral examination. If, after considering all the information gathered the lesion remains suspicious, then the area should be biopsied.

Indications

The VELscope, as recommended by the manufacturer, is meant to be an adjunctive screening tool, to aid in the detection of suspicious oral lesions that may require a surgical biopsy when performing a conventional oral examination. In addition, the manufacturer suggests that it may also be used to delineate the full extent of a lesion when determining margins of resection.⁷⁷

Certainly, one study of 44 patients reported that the VELscope has a sensitivity and specificity of 98 and 100% for identifying dysplasia or carcinoma verified by surgical biopsy.⁷⁸ It is important to point out that all of these lesions were visible with standard incandescent lighting and the majority of them were clinically suspicious. Unfortunately, whether the VELscope is useful in detecting suspicious lesions that are not visible, or for highlighting areas of concern within larger innocuous lesions, has not been determined. Although one small case series of three non-consecutive patients and one study screening for dysplasia in normal appearing mucosa in patients already diagnosed with oral cancer show promising results, no rigorous studies evaluating the efficacy of the VELscope in increasing early diagnosis and improving survival have been done.⁷⁹

Furthermore, it cannot be overemphasized that the VELscope was designed only to serve as an adjunctive diagnostic aid, and is not intended to



Fig. 30.6 VELscope device. Photograph reproduced from LED Dental Inc. with permission.



Fig. 30.7 Darkening of oral tongue due to difference in tissue fluorescence highlighted by VELscope. Photograph reproduced from LED Dental Inc. with permission.

replace the surgical biopsy for diagnosing oral lesions. Therefore, it should not be used to rule out malignancy in visible lesions.

Tissue reflectance

Tissue reflectance, or chemiluminescence, is an adjunctive screening tool that has been used for detection of premalignant or malignant lesions of the cervical mucosa. Recently, this method has been applied to the identification of suspicious oral lesions as well. Two such systems are currently available, ViziLite Plus and MicroLux DL, pictured in Figs 30.8a and 30.8b respectively. Tissue reflectance is a tool meant to enhance the visibility of pathologic lesions, as shown in Figs 30.8c, 30.8d, and 30.8e. Theoretically, the increased nuclear to cytoplasmic ratio of squamous neoplasms causes increased light reflectance relative to normal epithelium, an observation that these tools attempt to take advantage of.

Technique

After performing a thorough history and head and neck examination, have the patient rinse with a 1% acetic acid solution for 30 seconds. Next use the light source of the available system to inspect the mucosa of the oral cavity, taking special care to examine areas at higher risk for oral malignancy such as the floor of mouth and tongue. The ViziLite Plus system uses a chemiluminescent light that is disposable, where as the MicroLux DL is battery operated. In either case, the manufacturer recommends that the light be kept about 0.5cm from the area of epithelium being inspected. Suspicious lesions will appear “aceto-

white”. Surgical biopsy is necessary to confirm a diagnosis of dysplasia or cancer.

Indications

Tissue reflectance technology is intended to be an adjunctive screening tool to the conventional oral examination and requires confirmation by surgical biopsy. However, it is debatable whether or not the use of these instruments provides any added benefit over the conventional oral examination under standard incandescent lighting. Although the sensitivity for highlighting potentially pathologic lesions is high, it has been observed that many obviously benign lesions, such as leukoedema and traumatic ulcers, test positive.⁸⁰ In the majority of the studies evaluating its efficacy, lesions detected by tissue reflectance were also visible under incandescent lighting.⁸⁰⁻⁸⁴ Furthermore actual sensitivity and specificity are difficult to report, as surgical biopsies were not used to diagnose all detected lesions in any of the available studies. One study by Oh and Laskin reported that the use of ViziLite actually made visualizing lesions more difficult due to the distracting highlights it created.⁸³

Brush cytology

The brush biopsy (Oral CDx from CDx Laboratories) is a technique developed as a bridge to the surgical biopsy. It is intended for oral lesions that appear innocuous and would normally not be subject to a surgical biopsy. In these situations, rather than wrestle with the pitfalls of taking the “watch and wait” approach in a high-volume practice not specialized in

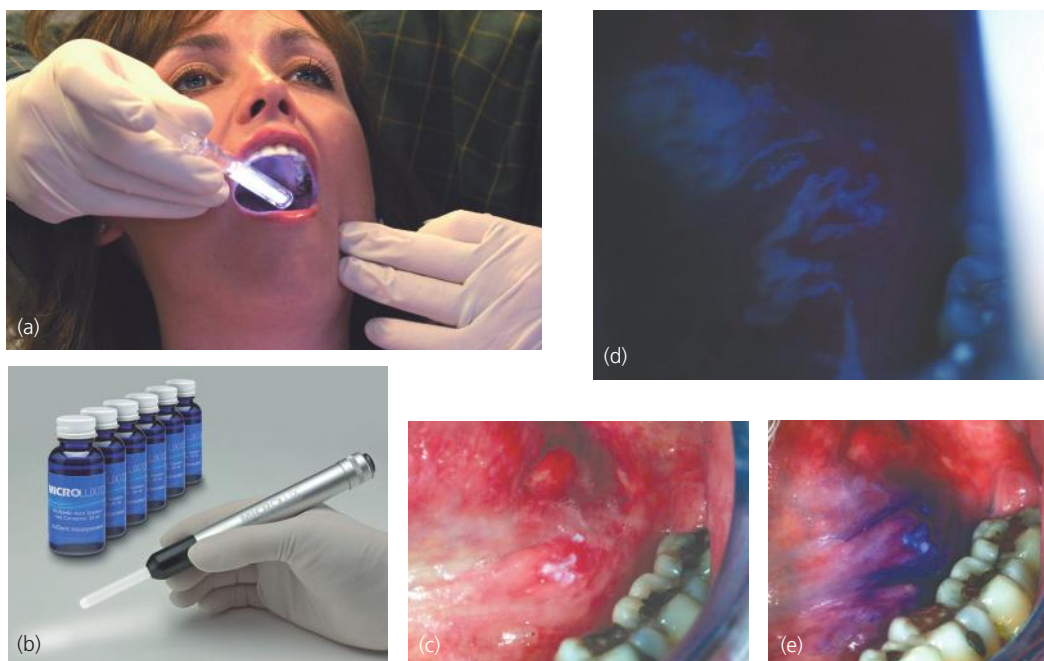


Fig. 30.8 (a) ViziLite device. Photo reproduced from Zila Pharmaceuticals, Inc. with permission. (b) MicroLux/DL device. Photograph reproduced from AdDent, Inc. with permission. (c) Tongue with carcinoma *in situ*, viewed under standard incandescent lighting. (d) The same lesion viewed with ViziLite chemiluminescent lighting. (e) The same lesion viewed after staining with toluidine blue.

following oral and maxillofacial pathology, the provider can perform a brush biopsy to assess the malignant potential of the lesion.

Technique

The bristled brush that is supplied by the manufacturer is applied to the lesion in question and used to scrape and disrupt the epithelium. The brush should be rotated several times over the lesion. This should be done with enough force to cause a small amount of bleeding, to ensure that the all layers of the mucosa as well as portions of the submucosa are sampled. Next, the cells need to be transferred to a glass slide by rotating the brush over the length of the slide. Following this, the manufacturer-supplied slide fixative should be applied directly to the entire slide. This should be allowed to dry for 20 minutes. This slide is then placed into a container and shipped to the CDx laboratory.

Once received by the laboratory, the harvested cells are analyzed by a computer, which selects out cells that are potentially malignant. These cells are then reviewed by staff pathologists, using both digital images and standard microscopy. The results are reported to the provider as “positive”, “atypical”, “negative”, or “incomplete”. Positive and atypical lesions are then subjected to a surgical biopsy. Lesions reported as incomplete do not have all of the cell layers. A repeat brush biopsy is recommended by the manufacturer.

Indications

The brush biopsy is intended to be an adjunct diagnostic tool, as opposed to a screening tool. In other words, in lesions that have been detected, the brush biopsy is meant to assist in establishing a diagnosis.

Because the brush biopsy is targeted towards those lesions that clinicians might not necessarily biopsy, it must be highly sensitive for detecting malignancy in lesions that are clinically innocuous. In order to accurately demonstrate this advantage, the population investigated must have lesions that are not already highly suspicious for malignancy, and all lesions in the population must be subjected to the gold standard: surgical biopsy. Unfortunately, the majority of available studies evaluating the brush biopsy are not selective for the target population and include likely or known-to-be malignant lesions.^{85–91} In addition, in most of the studies, lesions that were reported as “negative” based on the brush biopsy were not confirmed by a surgical biopsy. Only in one study by Scheifele *et al.* did all lesions have both a brush and surgical biopsy.⁹² The sensitivity and specificity reported by this paper were 92.3% and 94.3%, respectively. However, lesions that were highly suspicious for malignancy were included in this analysis. Therefore, the actual sensitivity may be lower due to spectrum bias. Regardless, a false-

negative rate of 7.7% is unacceptably high for an adjunctive diagnostic tool.

It is the authors’ opinion that the current literature does not support adding the brush biopsy to the diagnostic armamentarium. At best, it leads to a delay in the definitive surgical biopsy. At worst, it has a nearly 8% false-negative rate, leading the patient and provider to the mistaken impression that the patient is disease free. Some specialists have argued that it may have a role in the surveillance of patients with a history of oral cancer or dysplasia that might otherwise require multiple biopsies.⁹³ However, it is the authors’ opinion that it is exactly in this population that the threshold for biopsy should be lowered as they have the highest risk of malignant transformation.

Management of premalignant disease

In oncology, it is a commonly held tenet that cancers that are detected early can be treated more effectively and with less morbidity, thus increasing a patient’s chances for survival. It has been theorized that cells that ultimately become malignant must go through a succession of genetic changes that allow them to be derailed from the usual growth restrictions of normal cells in tissues, a process described as multistep carcinogenesis.^{94,95} Thus, if a cancer can be detected early, before it has acquired all of the mutations necessary for invasion and metastasis it may be possible to successfully treat the disease locally. Certainly, this has been shown to be true in several cancers including colorectal cancer and cervical cancer.

However, in order for the same to be true for oral cancer requires an understanding of the natural history of premalignant oral lesions. Furthermore, the natural history of the disease must be such that the premalignant lesion progressively becomes more dysplastic, becomes invasive, and only then does it have the ability to metastasize. Recent studies in breast cancer indicate that this paradigm may not be true and that metastasis can occur before invasion is seen on histopathology.⁹⁶ Regardless, there also must be an intervention that can remove the premalignant disease in such a way that improves survival.

For some time, it has been observed that certain oral lesions, specifically leukoplakias and erythro-leukoplakias, have the potential for malignant transformation. Unfortunately, the natural history of oral cancer and leukoplakia is convoluted. Not all oral squamous cell carcinoma are preceded by leukoplakias, not all leukoplakias progress to oral cancer, and some leukoplakias are much more aggressive than late-stage cancers. The following sections will briefly summarize what is known about the natural history, the surgical treatment, and medical treatments for premalignant disease.

Natural history of premalignant disease

Several lesions within the oral cavity have been said to be premalignant. They include leukoplakia, erythroplakia, lichen planus, and submucous fibrosis, among others. However, only leukoplakia and erythroplakia contribute significantly to the incidence of premalignant lesions and oral cancer.⁹⁷

Leukoplakia is a descriptive term for a white patch or plaque that is adherent to the oral mucosa and cannot be wiped off. It is a term used only when other diagnoses with similar appearances, such as lichen planus, candidiasis, white sponge nevus, frictional keratosis, and leukoedema, have been ruled out. Similarly, erythroplakia and erythroleukoplakia are descriptive terms for red lesions and mixed red and white lesions.⁹⁸

The worldwide prevalence of oral premalignant disease is estimated to be 1–5%^{99–101} and the prevalence of oral leukoplakia is calculated to be 2.6%.¹⁰² Naturally, prevalence varies significantly from region to region, depending on local risk factors.^{103–107} It is most commonly found in patients 30–70 years old, although younger patients are affected as well. Men are more commonly affected than women, although the ratio between the genders varies greatly depending on the population.

Overall, the buccal mucosa is the most common location for leukoplakia to occur.^{103,108–110} However, the site of the leukoplakia seems to be correlated to types of tobacco habits practiced by the patient population of interest. For example, parts of India where pan (betel nut with tobacco and slaked lime) chewing is common, 65% of leukoplakias are on the buccal mucosa. On the other hand, regions where reverse smoking is more prevalent, leukoplakias are more commonly found on the palate.¹⁰³ In addition, although buccal mucosa is the most common location, leukoplakias of the tongue and floor of mouth are most at risk for malignancy.^{110–113}

Partly because the definition of leukoplakia is one of exclusion, it includes a heterogeneous group of lesions with different clinical appearances and histologic findings. As such, the etiology of leukoplakia is multifactorial. The vast majority of leukoplakias have been correlated to tobacco use.^{101,105,108,114–116} As mentioned above, these are commonly found on the buccal mucosa or mandibular vestibule (snuff dipper's pouch) and are often reversible with cessation of the tobacco habit.¹¹⁷ However, 4–38% of leukoplakias are idiopathic.^{108,118–120} It is precisely these idiopathic leukoplakias that have the greatest potential for malignant transformation. As many as 5% of idiopathic leukoplakias are squamous cell carcinoma on initial biopsy, and another 10–15% are at risk for transformation.¹¹⁰ Erythroplakia and erythroleukoplakia carry even greater risk for developing malignancy.¹²¹

In addition, Silverman *et al.* have described a unique form of leukoplakia that has very high rates of

malignant transformation called proliferative verrucous leukoplakia (PVL).¹²² These lesions tend to appear initially as hyperkeratosis. However, they aggressively spread to include large areas of the oral cavity and often are multifocal. Although slow growing, they are persistent, and invariably recur after being excised. Over time, they can become exophytic and verrucous in appearance. Even as these lesions become progressively malignant in behavior, the histopathology can often remain as benign hyperplasia. Therefore, these lesions should be evaluated by their clinical behavior and not their pathology. Malignant transformation is expected if patients are followed for enough time, with rates of transformation at 60–100%.^{110,120,122,123}

It has been suggested that the human papilloma virus (HPV) may also play a role in leukoplakia and premalignant disease in the oral cavity, as is the case with oropharyngeal cancer.¹²⁴ Whether certain HPV subtypes increase the risk of developing oral cancer remains a contentious issue. Recent studies using polymerase chain reaction (PCR) assays to detect HPV DNA in oral cancers have not shown a relationship between oral squamous cell carcinoma and HPV positivity.^{125–127} However, other studies have shown a relationship.^{128–132} One of the difficulties with establishing a relationship between HPV and oral cancer is confusion with the nomenclature. The distinction between oral cancer, oropharyngeal cancer, and head and neck cancer is often not made explicit or is simply disregarded. In addition, PCR assays for HPV in oral cancer are not nearly as sensitive as they are for cervical cancer. At the present time, the general consensus is that HPV is not strongly associated with the development of oral cancer.¹³³

On histopathologic analysis, leukoplakias are also heterogeneous in presentation. Biopsy findings can be variable, demonstrating hyperkeratosis, varying grades of dysplasia, carcinoma *in situ*, or invasive squamous cell carcinoma. The discovery of dysplasia may provide some diagnostic benefit, as patients with biopsy-proven dysplasia are at higher risk for malignant transformation.¹³⁴

The grading of dysplasia is also helpful in predicting risk, because as many as 36% of severe dysplasias become invasive cancer.^{110,119,135} Although there are a number of dysplasia grading systems that have been described, the most commonly used system is as follows. Mild dysplasias have architectural changes confined to the basal third of the full thickness of epithelium. Moderate dysplasias are up to two thirds the full thickness of epithelium. Severe dysplasias are greater than two thirds of the full thickness, but without invasion through the basement membrane. Consideration is then given to the degree of cellular atypia. These features include increased nuclear cytoplasmic ratios, increased or abnormal mitoses, or pleomorphism of nuclei.

Unfortunately, oral squamous cell carcinoma does not always follow a natural progression from mild

dysplasia to severe dysplasia to invasive carcinoma. Cancer can also be found in hyperplasia or mild dysplasia. One group found that patients with mild dysplasia had the same transformation rates as those with severe dysplasia.¹³⁶ Also, the grading of dysplasia can be difficult, and there is significant inter-examiner and even intra-examiner variability. Several studies have found poor agreement between pathologists.^{137–140} As such, using presence or grade of dysplasia to quantify risk for transformation is not straightforward. An understanding of the biology of oral cancer development is necessary.

Genetic progression to oral cancer

Epithelial tissues, whether they are oral mucosa or skin, are able to renew themselves as they continuously slough off cells. These tissues are able to do so due to a tightly regulated system of self-renewal involving stem cells that reside in the basal layer of the epithelium. Each of these stem cells, when appropriately stimulated, divides to produce identical cells called daughter cells. One of these daughter cells undergoes further divisions and differentiates into mature squamous cells. The other of the daughter cells remains in the basal layer as a stem cell. Once the tissue is renewed, the system autoregulates and the stem cells become dormant.

Because these epithelial stem cells remain in the basal layer of the epithelium rather than being sloughed off, they are potentially exposed to carcinogens or other genetic insults for the lifetime of the patient. It is believed that genetic injury to epithelial stem cells leads to loss of this tight tissue regulation.¹⁴¹ This loss of regulation results in a clonal expansion of epithelial stem cells with the ability to self-renew, that ultimately creates a field of potentially premalignant cells that are susceptible to further mutations, the so-called “field cancerization” phenomenon described by Slaughter *et al.*¹⁴² Amongst this field, further genetic injury will lead some of these cells to become more derailed from regulatory mechanisms as they acquire mutations that promote proliferation, inhibit tumor suppressor genes, stimulate angiogenesis, and allow invasion.

It is estimated that a minimum of five mutations are required for carcinogenesis to occur.¹⁴³ Califano *et al.* proposed a model for progression of head and

neck squamous cell carcinoma defining early and late mutations that lead to carcinogenesis, which is shown in Fig. 30.9.¹⁴⁴ Since Califano’s article, a plethora of work has been devoted to identifying other common genetic mutations that predispose to transformation and invasion.¹⁴⁵ However, how they fit into the natural history of carcinogenesis and whether the knowledge will affect clinical practice is yet to be determined.

At times, the progressive accumulation of genetic injury leads to microscopic and macroscopic changes, such as leukoplakia. Unfortunately, these genetic alterations can also occur without any phenotypic changes. This significantly handicaps the surgeon’s ability to fully excise a lesion or tumor. Certainly, it has been known for some time that even histologically clear margins demonstrate mutations that ultimately lead to increased rates of recurrence.^{146–152} In the setting of premalignant disease, excision of a leukoplakia may only be removing a small portion of the field of defective cells. This makes oral cancer screening and surveillance difficult. This may also explain the frequency of local recurrences or second primary tumors in contiguous areas.

Surgical management of dysplasia

The most common method for managing biopsy-proven dysplasia of the oral cavity is local surgical excision. However, it is important to recognize that there is currently no evidence that surgical excision is effective in removing all premalignant cells. Nor is there any evidence that suggests excision prevents recurrence of the dysplasia, prevents transformation, or reduces mortality. In actuality, nearly 30% of dysplasias recur in the same location after excision.¹⁵³ A recent Cochrane review underscored this lack of evidence of therapeutic benefit, noting that there is not a single randomized controlled trial evaluating the efficacy of surgical treatment of dysplasia.¹⁵⁴ The majority of the literature on the subject is retrospective, does not use controls, and has conflicting results. One retrospective study that does have a control group found no significant difference in rates of transformation.¹⁵⁵ Furthermore, excision of dysplasia is like any other surgery and has associated morbidity and risks.

Despite this, there are certainly reasons to recommend surgical excision. The primary reason is that

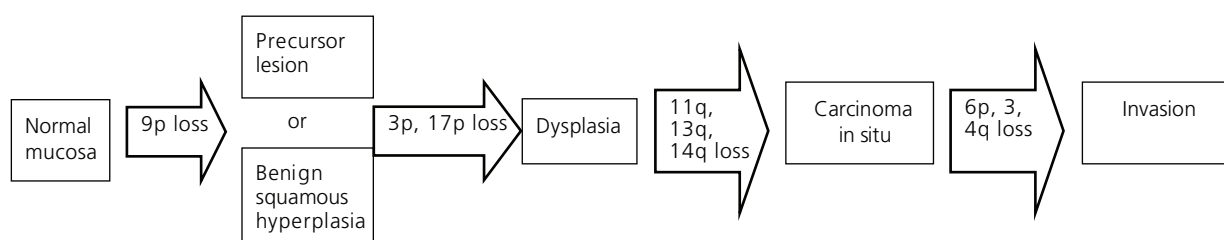


Fig. 30.9 Genetic progression of head and neck cancer. Adapted from Califano *et al.*¹⁴⁴

excision of a dysplastic lesion provides a valuable histologic diagnosis.¹⁵⁶ As mentioned above, 5% of idiopathic leukoplakias already have invasive cancer at the initial biopsy.¹¹⁰ In addition, incisional biopsies are subject to sampling error, and dysplasia or carcinoma can be easily missed. Some studies have reported that over 10% of lesions diagnosed by incisional biopsy as dysplasia demonstrated invasive carcinoma after excision.^{157,158}

Medical management of dysplasia

Given that surgery is probably ineffective in treating dysplasia, especially in the setting of field cancerization, the use of systemic or topical chemopreventive measures is particularly attractive. Several agents have been studied for this purpose, including vitamin A and retinoic acid, taken systemically or applied topically, systemic beta-carotene, systemic lycopene, topical ketorolac, and topical bleomycin. Although beta-carotene, lycopene, vitamin A, and retinoids showed a small amount of benefit, they were associated with adverse effects and had high recurrence rates with cessation of the therapy. A recent Cochrane review concluded that no currently studied chemopreventative measures are of any benefit for treating premalignant disease or preventing malignant transformation.¹⁵⁴

Summary of management of dysplasia

The authors recommend, therefore, that all moderate to severe dysplasias be excised, if it is anatomically feasible. This should be done using a scalpel, as the use of lasers or electrosurgery eliminates the ability to perform a histopathologic analysis. Patients with mild dysplasia should be counseled that their risk for transformation is likely to be less, but still present. These patients can undergo expectant management and should be educated to self-monitor for phenotypic changes or the development of concerning symptoms, such as pain, paresthesia, or ulceration. Regardless of the therapy provided, a patient with biopsy-proven dysplasia should be followed by clinical exam indefinitely.

Margin assessment

The primary aim of surgical resection of cancer of the head and neck is to remove all macroscopic and microscopic tumor cells. In doing so, the hope is that either all of the cells with potential for invasion have been removed, or that only a small number of cells with premalignant or malignant characteristics remain such that the patient's own immune defenses may eliminate them. The completeness of a surgical resection is typically assessed by measuring the margin clearance.

Margin assessment involves the histologic measurement of a cuff of normal tissue beyond the most invasive portion of the tumor. For oral squamous cell carcinoma (SCC), a clear margin is conventionally defined as 5 mm of normal tissue beyond all tumor margins, a close margin as less than 5 mm but without tumor cells involving the margin, and positive if tumor cells are seen at the margin.^{159–161} However, these designations are arbitrarily defined.¹⁶² Patients with close or positive surgical margins have been shown to have worse local control of disease, and adjunctive radiation therapy or additional surgical resection is generally offered.^{159–161,163} Thus, close or positive margins are regarded as poor prognosticators, with 5-year survival decreasing from 60% to 52% and local recurrence rates increasing from 18% to 36%.¹⁶¹ However, recent studies have suggested that close or positive margins may not actually be indicators of lack of surgical completeness, but rather it may represent an increased invasiveness of a particular tumor.¹⁶⁴

Frozen section analysis

The pathologist performs the margin assessment on the permanent specimen after the ablative surgery. However, it is desirable to assess the adequacy of a resection intraoperatively, so that the surgeon may decide whether to increase the volume of resection if allowable. Frozen section analysis is used for this end.

Frozen section analysis is typically performed in the following way. Intraoperatively, after the main tumor specimen has been resected, the surgeon cuts sections off of the mucosal and deep margins, especially in areas of the surgical bed that are concerning for remaining tumor cells. These specimens are submitted to the pathologist for frozen section analysis. The pathologist will freeze the specimens, and then slice them into 5–7 µm sections with a microtome. These sections are then adhered to glass slides, treated with fixatives, and stained with hematoxylin and eosin. These slides are then reviewed to verify if any tumor cells remain in the margin.

There are two main drawbacks to this form of analysis. The first is that it is subject to sampling error. Because the surgeon cannot sample the entire margin of the tumor resection bed, whether a positive margin can be detected intraoperatively depends entirely on whether the surgeon can predict where the tumor cells remain. The second shortcoming is that close margins will not be detected.

For these reasons, some surgeons argue that frozen section analysis is not beneficial and simply prolongs the length of the operation. Ord and Aisner reported that, although frozen sections were 99% accurate in diagnosing SCC when compared to permanent sections, only 1 of 49 patients (2%) had a positive margin diagnosed by frozen section that led to extending the resection margin.⁴⁵ This group argued

that the routine use of frozen section analysis might not be worthwhile.

However, given the lack of a viable alternative to frozen section analysis for intraoperative assessment of margin status, the authors still recommend the routine use of frozen section analysis, keeping in mind the limitations with this technique. When sampling tissue for frozen section analysis, the surgeon should take care to select areas where tumor invasion is most likely to have compromised the margin. This invariably will occur at the deep margin. When treating tumors of the retromolar trigone or posterior maxilla, it is often the most posterior and deep margin that is most difficult to clear and, because of this, is the most appropriate area to sample. The surgeon should also keep in mind the tissue planes that are most susceptible to rapid tumor invasion. SCC does not uniformly invade with a broad front radiating from an epicenter. Rather, SCC will invade irregularly, along the path of least resistance. For resections involving bone, this may be the bony margin or marrow space. For tongue cancers, this is often along skeletal muscle. If there is nerve associated with the tumor, invasion may be perineural.

Tissue shrinkage in margin analysis

One major hurdle with using clear margins as a treatment goal is that margin lengths are subject to tissue retraction and shrinkage. Therefore, when a surgeon measures and resects a 1 cm surgical margin from the edge of the tumor, this length will invariably shrink to a shorter margin on pathology. Johnson *et al.* reported in a dog model a mean shrinkage of 34.5% in deep tongue margins, with most of the shrinkage occurring immediately after resection.¹⁶⁵ Mistry *et al.* reported similar results in their study in humans.¹⁶⁶ Furthermore, the shrinkage that occurs is not uniform and, as one would expect given the complex anatomy of the oral cavity, varies from location to location. In a retrospective study, our group has reported that tumors of the retromolar trigone and mandible have the greatest discrepancy between the surgical and pathologic margin.¹⁶⁷

Because of this variable margin shrinkage, it can be difficult to achieve clear margins predictably. Even when a surgeon includes a generous cuff of healthy tissue around the tumor, tissue shrinkage can result in a close margin. Some have suggested that the fact that margin shrinkage occurs should be reassuring to the surgeon in that a close margin after shrinkage occurs is, in actuality, a clear margin. However, the authors strongly disagree with this interpretation, recalling that all of the literature relating margin status to local control of disease and outcomes are based on the final pathologic margin, presumably after any margin shrinkage would have occurred. Therefore, rather than being reassuring, the presence of tissue shrinkage should prompt the surgeon to be more careful in planning the surgical resection, being more

generous in areas where the surgeon feels shrinkage is more likely to occur. Naturally, this is exceptionally difficult, given that even the most experienced head and neck surgeons report rates of close or positive margins at 33–43%.^{45,159,168}

Molecular techniques in margin analysis

Surgical resection with a margin of normal tissue is important because cells that appear normal on histopathology may still harbor mutations that predispose the cell towards malignant behavior. The goal, then, is to surgically remove as many of these normal appearing malignant cells when resecting the tumor. Brennan *et al.* elegantly demonstrated that mutations in TP53, a tumor suppressor gene that is commonly mutated, could be found in margins that are clear.¹⁴⁶ These mutations increased the risk for local recurrence in the same location. Similarly, Nathan *et al.* demonstrated that overexpression of the translation factor eIF4E is an early step in malignant transformation. Furthermore, detection of eIF4E in histopathologically clear margins using immunohistochemistry is correlated with high recurrence rates.^{148,150,169} Therefore, the development of molecular techniques to detect these mutations in normal appearing tissue would be of great value in aiding surgical completeness when possible.

However, a number of difficulties have prevented routine use of molecular testing outside of a research setting. The most important hurdle is that the majority of studies using molecular markers only test one or a handful of genetic mutations. Unfortunately, oral SCC is heterogeneous. Even mutations of TP53, the most common mutation associated with oral SCC, are only present half of the time.^{170,171} To address this problem, the use of microarrays for molecular margin analyses are actively being researched to simultaneously look for aberrations in expression or copy numbers across the entire genome. Although the use of these molecular techniques is still in its infancy, the potential is substantial. Chung *et al.* reported that, using microarrays to profile head and neck SCC, they were able to predict risk of neck metastases in 80% of their patients, but only after excluding patients with oral SCC, underscoring the heterogeneity of this disease.¹⁷²

Summary

Surgical pathology remains the gold standard for diagnosis of tumors of the head and neck. However, in order to fully utilize the expertise of our pathology colleagues, the surgeon must be able to effectively communicate with the pathologist, provide the pathologist with an adequate and appropriately oriented specimen, and understand the limitations of pathology and how to interpret results. Pathologists

have several tools with which they can analyze tissues; which tools they choose depends on the clinical question that the surgeon presents to them. Depending on the context, the same slide can be interpreted differently. Surgeons often rely on frozen sections to preliminarily assess the completeness of a resection, but the efficacy of this tool depends on the surgeon's ability to select the appropriate specimens for the pathologist to analyze. Pathologic processing can cause dimensional changes in tissue specimens. The surgeon must understand and consider this in order to adequately remove tumor. The next frontier for pathology is the use of molecular techniques on tissue to improve margin analysis and stratify risk for recurrence, metastasis, and, more importantly, prognosis. As these technologies develop and become readily available, surgeons and pathologists must learn to incorporate them into the diagnosis and treatment of their patients.

Case presentations

The case of the inappropriate use of a technique

Patient A was a 56-year-old woman who was seen in our department for a fifth opinion regarding a possible diagnosis of ameloblastoma of her left lower jaw. She reported a 5-month history of a painless swelling at the angle of her left lower jaw. Thinking that this was a dental infection, she went to see her dentist, who noted an unusual radiolucency on dental radiographic exam. The dentist consequently referred her to see a specialist. She saw a number of different specialists, and ultimately underwent an FNAB of the left mandibular swelling. The cytopathologist noted "malignant appearing epithelial cells", offering a differential diagnosis of ameloblastoma or myoepithelial tumor. She was then offered mandibular resection and reconstruction with a fibular free flap as treatment for this lesion by the specialist at the academic center nearest to where she lived. Seeking another opinion, she was referred to the University of California, San Francisco.

Outside of the swelling, she denied any appreciable symptoms, such as pain, paresthesia, dysphagia, odynophagia, otalgia, voice changes, weight loss, fevers, chills, or malaise.

Her past medical history was only significant for systemic lupus erythematosus, for which she took Plaquenil. Her surgical history only included third molar extractions in the distant past. Her family history was significant for her mother being diagnosed with lung cancer in her fifties with no history of tobacco use. The patient was also a never smoker. She drank alcohol occasionally. She denied any history of illicit drug use.

On examination, the patient appeared well. However, she had obvious facial asymmetry involv-

ing her left mandible, as shown in Fig. 30.10. A mass measuring approximately 4×3 cm was clearly visible at the angle of her left mandible. The overlying skin was entirely normal. The lesion was solid, fixed to the surrounding tissues, and non-tender. There was no palpable thrill or audible bruit. On bimanual palpation, with one examining finger placed intraorally, a clear demarcation could be appreciated between the mandible and the lesion. No buccal expansion of the mandible was appreciated. The rest of her oral exam was unremarkable.

A series of radiographs ordered by her previous consultations were reviewed. As shown in Fig. 30.11, her dental panoramic tomogram shows a poorly defined osteolytic lesion at the angle of her left mandible, inferior to the inferior alveolar nerve canal. Fig. 30.12 shows selected views from her cone-beam CT. One can clearly see that the lesion was not associated with her dentition.

Having reviewed the patient's history, exam, radiographic studies, and cytopathology report, it was clear that a number of things did not make sense, particularly with her diagnosis of ameloblastoma. Ameloblastomas are benign odontogenic tumors. Although epithelial in origin, they typically are not



Fig. 30.10 Facial photograph of patient A. Note the swelling of the left angle of mandible.

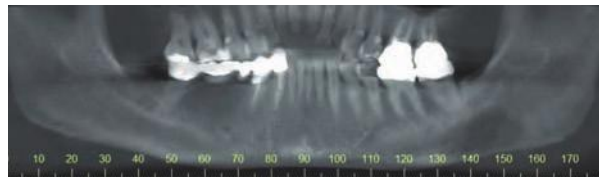


Fig. 30.11 Initial dental panoramic tomogram of patient A. Note the poorly defined radiolucent lesion located at the angle of the left mandible, inferior to the inferior alveolar nerve canal.

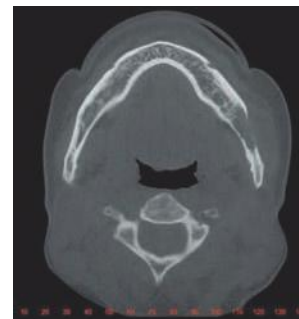


Fig. 30.12 Selected images of the cone-beam CT of patient A.

described as containing “epithelial” cells and certainly are not “malignant”. Very rarely, a patient may present with ameloblastic carcinoma, which is malignant. However, being odontogenic, it would not be located inferior to the inferior alveolar nerve canal. Therefore, the patient’s diagnosis seemed implausible.

Our differential diagnosis would include metastatic cancer, salivary gland neoplasms, metastatic lymphadenopathy, and lymphoma. The most common cancers to metastasize to bone in women are breast, lung, liver, renal, and colon cancers. This diagnosis fits well, given the appearance of the lesion, the location at the angle of the mandible, which is the most common site for bony metastasis in the maxillofacial region, and the family history of lung cancer in her non-smoking mother. For salivary gland neoplasms, it would be unusual for benign tumors to cause osteolysis of the mandible. Our differential diagnosis for malignant salivary gland neoplasms would include mucoepidermoid carcinoma, adenoid cystic carcinoma, polymorphous low-grade adenocarcinoma, and, given the cytopathology report, epithelioid myoepithelial carcinoma. The most common cancer to metastasize to the cervical lymph nodes would be head and neck SCC. However, the exam did not fit this diagnosis well. Much lower on the differential we might consider infectious processes, such as chronic osteomyelitis, tuberculosis, cat scratch disease; however, the exam also did not seem to fit well with any of these.

Regardless, it was clear that the next step in the evaluation would be to perform an incisional biopsy to obtain an adequate tissue sample for diagnosis. Given the difficult location and the intraosseous component, she was scheduled for this procedure under general anesthesia in the operating room.

Frozen sections taken at the time of the operation demonstrated an epithelial neoplasm. The initial review of the permanent specimen showed an infiltrating glandular malignancy, consistent with a metastatic adenocarcinoma. To determine the origin, a battery of stains was performed. Pancytokeratin stains were positive, confirming the epithelial nature of the malignancy. A positive CK7 and negative CK20 profile was suggestive of lung, thyroid, salivary gland, breast, and gynecologic tumors. TTF-1 positivity narrowed the diagnosis to thyroid or lung. Negative mammaglobin and ER argued against breast cancer. Finally, negative synaptophysin and chromogranin argued against a neuroendocrine carcinoma. These results were most consistent with a diagnosis of metastatic lung adenocarcinoma. Given that the patient already had metastasis to a distant site, she was diagnosed with a Stage IV adenocarcinoma of the lung.

A CT of the chest was performed, pictured in Fig. 30.13, which demonstrated a 2.6×1.8 cm spiculated lesion of the right upper lobe with a 6–7 mm perivascular lymph node. She was subsequently

referred to a cardiothoracic surgeon and a medical oncologist. She ultimately underwent treatment with chemotherapy. She had complete resolution of both the mandibular and right upper lobe lesions. Unfortunately, soon after, she was found to have local recurrence of her lung adenocarcinoma.

There are a number of learning points that should be highlighted with this case. The first is that the history and physical exam are critical. The family history of a non-smoking mother diagnosed with lung cancer should alert the clinician to include metastatic cancer in the differential diagnosis, especially given the location and clinical findings. The examination findings were not consistent with an odontogenic tumor, so it should not be included on the differential diagnosis. Second, one must choose the appropriate test for the patient. An FNAB is not the diagnostic test of choice for an osteolytic lesion in the mandible. It should not be used to definitively diagnose ameloblastoma. Third, the surgeon must be able review the pathology report, interpret the description in the report, and place the pathology in context of the patient. Although the pathologist is the one reading the slide, the responsibility to come to the correct diagnosis and treatment depends solely on the surgeon. A surgeon reading the word “malignant” on a report with a diagnosis of ameloblastoma should immediately begin to question whether that report is valid. If any diagnosis appears on a report that does not fit well with the initial differential diagnosis, it should be considered suspect and the pathology should either be repeated or reviewed again. This also emphasizes the necessity of excellent communication with the pathologist and using a pathologist with experience in interpreting head and neck diseases. Although this mistake probably did not affect the patient’s prognosis, she would have needlessly undergone a major debilitating surgery if the mistake had not been corrected.

The case of the inadequate specimen

Patient B was a 93-year-old woman referred to the University of California San Francisco’s Department of Oral and Maxillofacial Surgery for evaluation of a large exophytic verrucous mass on her hard palate. She initially noticed a lesion in her upper jaw approximately 1 year prior when her maxillary denture was

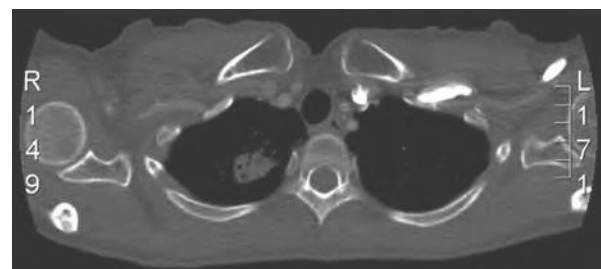


Fig. 30.13 Chest CT of patient A. Note the 2.6×1.8 cm spiculated lesion of the right upper lobe and the 6–7 mm perivascular lymph node.

not fitting as well. She went to see her dentist to see if her denture could be adjusted and the dentist noticed the verrucous mass on her left hard palate, part of which was underneath the maxillary denture. She was then referred to her local surgeon for a biopsy. She underwent a biopsy in the clinic. The specimen was reviewed by an outside pathologist as “papillomatous hyperkeratosis” with “no evidence of malignancy”. The surgeon, reassured, diagnosed her with a benign papilloma and sent her back to her dentist to have her denture remade. However, her general dentist was still quite concerned, and asked the surgeon to repeat the biopsy. Again, the surgeon performed a biopsy in the clinic, and again, the diagnosis came back as a benign papilloma. Unconvinced, the general dentist referred the patient to the University of California, San Francisco.

She denied any pain, paresthesia, dysphagia, odynophagia, otalgia, voice changes, weight loss, fever, chills, or malaise. She did report that she had occasional bleeding from the area. Her main concern was her ill-fitting denture.

Her past medical history was significant for a history of colon cancer treated by partial colectomy, hypertension, hypercholesterolemia, stable angina treated with nitroglycerin, and bronchitis. Her surgical history was significant for her partial colectomy

and cataract surgery. She was taking Zocor, nitroglycerin, and Benicar. She denied ever smoking and she only rarely drank alcohol. She denied any history of illicit drug use. Her family history was non-contributory.

On examination, she had no cervical or submandibular lymphadenopathy. Her facial exam revealed no asymmetries or skin lesions. Her oral exam, with her dental prostheses removed, demonstrated a large exophytic verrucous lesion, approximately 4×6 cm in size, which extended from the left hard palate and maxillary alveolus posteriorly into her oropharynx, as shown in Fig. 30.14. In addition, there was a 2×1 cm verrucous lesion on her left mandibular gingiva in the area of the left retromolar trigone, within an intervening area of normal but telangiectatic mucosa between the mandibular lesion and the maxillary lesion.

A dental panoramic tomogram (Fig. 30.15) was taken that did not show any evidence of bony involvement in the mandible. The maxilla was atrophic, and it was difficult to appreciate whether any bony invasion had occurred.

On review of the pathology reports, which diagnosed the lesion as “benign polypoid and papillary squamous mucosa with acute inflammation and reactive changes, consistent with squamous papilloma”, it was noted that the dimensions of the first biopsy specimen were “ $0.7 \times 0.5 \times 0.3$ cm” and of the second specimen were “ $0.5 \times 0.4 \times 0.3$ cm”.

To summarize, Patient B was a 93-year-old woman with a large aggressive verrucous lesion of the maxilla extending into her oropharynx and mandible. Her previous diagnosis of benign papilloma, or “oral wart”, was not consistent at all with her clinical presentation, with papillomas being typically much smaller. Our differential diagnosis included oral SCC, verrucous carcinoma, or proliferative verrucous leukoplakia.

Recalling that the previous biopsy specimens were both only 0.3 cm in depth, short of the minimum 5 mm depth required, we made the determination that more tissue would be needed for an adequate diagnosis. In the clinic, under local anesthesia, the



Fig. 30.14 Intraoral photograph of patient B. Note the large exophytic verrucous lesion of the left maxilla.



Fig. 30.15 Initial dental panoramic tomogram of patient B.

patient had three surgical biopsies using a punch biopsy that was carried to the hub of the cutting edge and down to the bone of the maxilla. These specimens were then sent to our oral pathologists for review, and the patient was diagnosed with oral SCC.

The patient was presented at the University of California, San Francisco Head and Neck Tumor Board and was staged with a T4N0Mx SCC of the left maxilla and mandible. The head and neck CT is shown in Fig. 30.16. She was taken to the operating room and underwent a left selective neck dissection, left maxillectomy, left mandibular gingiva excision, and left oropharyngeal excision. She was reconstructed with a split-thickness skin graft and surgical obturator.

The first learning point from this case is that, as mentioned in the previous case, the surgeon must interpret the pathology report in the context of the patient's clinical presentation. A diagnosis of an oral wart is not consistent with a 4 × 6 cm lesion extending into the pharynx and mandible. This should prompt either a repeat biopsy or having the pathology reviewed again. The second learning point is that the surgeon needs to give the pathologist adequate material to work with. A surgical biopsy needs to be a minimum of 5 mm in depth. With exophytic verrucous lesions, this depth should be greater considering that much of the verrucous portion of the lesion is keratinized epithelial tissue and the pathologist needs to see invasion into the submucosa to establish the diagnosis of cancer. The outside surgeon, reticent to aggressively incise into a lesion on a 93-year-old woman, only performed an epithelial shave of the lesion. Although this was certainly more comfortable (a fact that was confirmed by the patient, who reported that our biopsy was much "deeper"), it delayed the correct diagnosis for this patient.

The case that benefited from an oral pathology consult

Patient C was a 7-year-old girl who was referred to the San Francisco General Hospital, Department of Oral and Maxillofacial Surgery for evaluation and treatment of an incidental finding of an osteolytic lesion in her left mandible. The lesion was first dis-

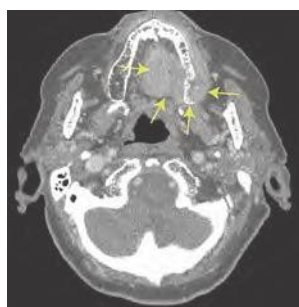


Fig. 30.16 Head and neck CT of patient B. Note the large enhancing lesion in the area of the left maxilla with evidence of bony invasion, as highlighted by the yellow arrows.

covered during a routine dental visit on a dental radiograph. At that time, a dental panoramic tomogram was taken that demonstrated a well defined radiolucent lesion in the body and ramus of the left mandible. This lesion was causing the displacement of her tooth #19 (Universal numbering system) or 4–6 (FDI) and the developing follicle of tooth #18 (Universal numbering system) or 4–7 (FDI). She was subsequently referred to San Francisco General Hospital.

The patient was entirely asymptomatic. She did not complain of any pain, paresthesia, dysphagia, odynophagia, dyspnea, otalgia, weight loss, fever, or chills. Her past medical history, past surgical history, social history, and family history were unremarkable.

On examination, she was a well appearing young girl in no acute distress. She had no cervical or sub-mandibular lymphadenopathy. She did not have any visible facial asymmetry or cutaneous lesions. However, on clinical examination, she was found to have bony hard expansion of the body of the left mandible. On examination of her oral cavity, her tooth #19 (Universal numbering system) or 4–6 (FDI) was somewhat displaced. On bimanual examination, her buccal expansion was hard, with no evidence of cortical perforation.

Her dental panoramic tomogram was reviewed (not pictured) which demonstrated a 3 × 2 cm multilocular radiolucency in the body and ramus of the left mandible, as previously described.

The initial differential diagnosis for a slow growing expansile, well defined, osteolytic lesion of the mandible in a 7-year-old girl would include dentigerous cyst, keratocystic odontogenic tumor, odontogenic myxoma, and ameloblastic fibroma. Ameloblastoma might also be included, but given the patient's age it would be much lower in probability. Given the indolent nature of the patient's history, we were reassured that it was unlikely to be malignancy. As all of these lesions are diagnosed and treated surgically, the patient was planned for an incisional biopsy in the operating room under general anesthesia. The decision to go to the operating room and use general anesthesia was made because of the lesion's location as well as the patient's age. In addition, given that the patient would already be intubated and anesthetized, she also was to have a CT performed following the biopsy.

The frozen sections were reassuring, consistent with a benign pathology. The final pathology diagnosed the lesion as an ameloblastoma. The CT (Fig. 30.17) agreed with the previous radiologic findings, showing a 1.7 cm osteolytic lesion in the left mandible associated with the crown of the developing tooth #18.

However, we felt that it would be prudent to get a second opinion on the pathologic diagnosis. Ameloblastoma was much lower on our differential, given the patient's age. Ameloblastic fibroma, which could easily be confused for ameloblastoma, was

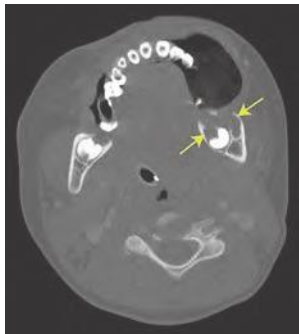


Fig. 30.17 Head and neck CT of patient C. Note the osteolytic lesion causing cortical expansion in the area of the developing left mandibular second molar, as highlighted by the yellow arrows.

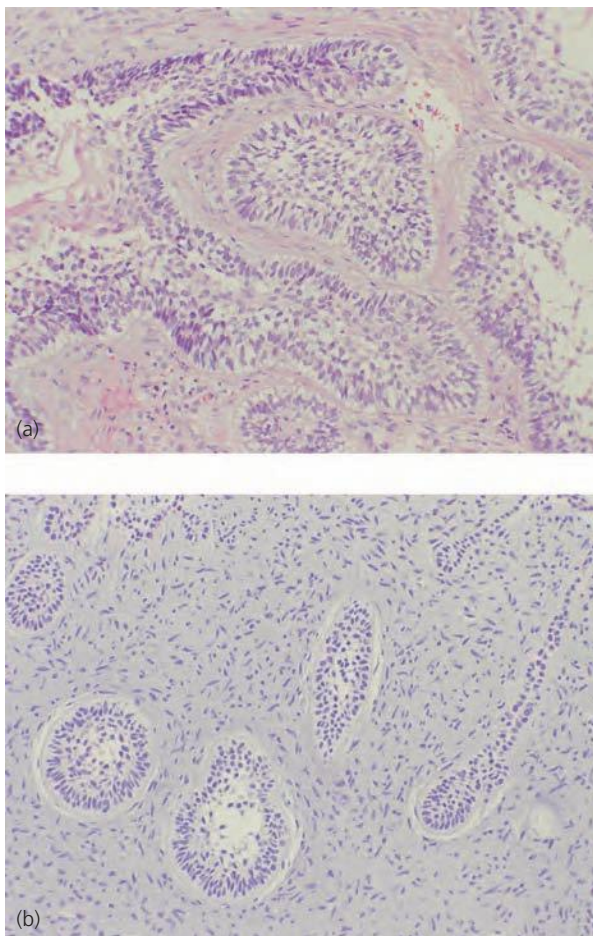


Fig. 30.18 (a) Micrograph of ameloblastoma. (b) Micrograph of ameloblastic fibroma. Note the significant myxoid connective tissue component.

much more likely. Fig. 30.18a shows a representative micrograph of an ameloblastoma compared to Fig. 30.18b, an ameloblastic fibroma. The distinction is critical, as ameloblastoma is treated with a wide resection, whereas ameloblastic fibroma can be effectively treated with enucleation. Consequently, we had the slides reviewed a second time by our colleagues in oral pathology.

On the second review, it was noted that although there were numerous strands of odontogenic epithelium very similar to ameloblasts, there was also a

prominent myxoid connective tissue component within the tumor as well. Given that ameloblastoma is an epithelial tumor in origin and ameloblastic fibroma is a mixed epithelial and mesenchymal tumor, the diagnosis of ameloblastic fibroma was made.

The patient was then treated with extraction of tooth #19 and enucleation and curettage of the ameloblastic fibroma. The patient recovered uneventfully. She was seen for regular follow-up visits and continues to remain disease free. A follow-up dental panoramic tomogram (not pictured) demonstrated near complete resolution of the lesion.

Upon reviewing this case, it is important to take away two learning points. The first is that if there is any question with regard to the diagnosis, one should not hesitate to consult a second opinion. The second point is that, given the rarity of these lesions, it is critical that the specimen be reviewed by a pathologist with experience in this area. This is especially true when the correct diagnosis can be the deciding factor between a minor surgical procedure and a mandibular resection.

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Chapter 31

Cystic Lesions of the Jaws

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This chapter describes the range of cystic lesions, common and uncommon, of the hard and soft tissues of the jaws and neck that present to dentists and oral and maxillo-facial surgeons. Cystic lesions associated with the salivary glands and those from odontogenic tumors are considered in their respective chapters. For each condition the etiology, presentation, radiographic findings, and diagnosis are presented along with the accepted treatment for each condition.

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Cystic lesions of the jaws are common pathologic lesions. This group of lesions is comprised of a number of different conditions either odontogenic or non-odontogenic in origin.

Odontogenic cysts are the most frequent, and of these the radicular cyst is seen most often. Odontogenic cysts arise from the epithelial remnants of tooth for-

mation, and consequently they are epithelially lined and develop in the tooth-bearing areas of the jaws. These lesions are benign, but very rare instances of associated malignancy or malignant change in the cyst wall are found in the literature.

Non-odontogenic cysts were classically thought to arise from epithelial remnants from the fusion of the maxillary and frontonasal processes and mandibular processes in the embryo. Aneurysmal and solitary bone cysts, however, are non-epithelial lined non-odontogenic cysts. These theories have, however, been challenged and most are now felt to be odontogenic or respiratory in origin.

Many of these lesions are slow growing and asymptomatic, often found as incidental findings at dental check-ups or with the use of panoramic radiographs for dental assessment. Accurate diagnosis and appropriate treatment are required, as these lesions will generally continue to grow and may affect adjacent teeth and other structures and even result in pathologic fracture.

The small group of developmental soft tissue cysts will be considered at the end of this chapter.

Odontogenic cysts

Apical or radicular cysts

Radicular cysts are the commonest jaw cysts, comprising half to two-thirds of all such lesions. The cyst is most commonly found associated with the apex of a tooth, but lateral periodontal cysts may be found associated with lateral canals and residual cysts persist and enlarge following extraction of the causative tooth leaving cystic remnants behind. The cyst usually arises following the development of periapical granuloma from the necrotic remnants of the dental pulp. Chronic inflammation of this tissue initially stimulates the cell rests of Malassez, resulting in epithelial proliferation. This initiation phase is then followed by a phase of cyst development, followed by cyst growth. Radicular cysts are fluid-filled lesions

that expand in the jaw by osmotic pressure and cytokines cause local resorption of bone.

Radicular cysts are uncommon in children, and become more frequent in adolescents but are most often seen in adults of all age groups. They are reported to be more prevalent in men than women, although this may be due to women taking greater care of their teeth and visiting the dentist more often than men. The maxilla is more commonly involved than the mandible. Most radicular cysts present as slow-growing, painless swellings associated with a non-vital or root-treated tooth or as incidental findings on panoramic radiographs. Less frequently, radicular cysts become infected and present as a facial abscess or cellulitis.

At presentation the oral soft tissues are often normal, but careful examination may reveal a firm, hard, smooth swelling at the apex of the tooth. As the cyst enlarges the overlying bone may become resorbed leaving a soft swelling or even a thin layer of bone that may be felt to give way on palpation (egg shell crackling). As the cyst is palpated the patient may report pain. The causative tooth will be found to be non-vital and other teeth involved as the cyst enlarges may also become devitalized.

Investigation is by vitality testing of the associated teeth and periapical or panoramic radiographs. The radiographic features are of a smooth, rounded, unilocular radiolucency with a radiopaque outline associated with the apex of a non-vital tooth. Adjacent structures may be displaced by the expanding cyst (Fig. 31.1).

Treatment is by removal of the cause of the inflammation either by extirpation of non-vital dental pulp by orthograde root filling in the first instance or by extraction of the causative tooth. Small cysts are reported to regress, however larger lesions will require enucleation, complete removal of the cyst lining usually facilitated by using a curette. In teeth previously root treated with a good periapical seal, or those restored with a post crown where the post is large and threatens root fracture if an attempt is made



Fig. 31.1 Radicular cyst arising from non-vital roots of lower left first molar tooth. Note displacement of inferior dental canal by cyst posteriorly.

to remove it, apicectomy needs to be performed if the tooth is not extracted.

Following enucleation, the soft tissue overlying the bony cavity is closed and the resulting hematoma ossifies. Some surgeons prefer to fill large cavities with corticocancellous bone chips to accelerate bone regeneration, but there are no studies to show that this practice has benefit. More recently the use of bone morphogenic protein has been described to improve rates of bone regeneration. In mandibular lesions where removal of the cyst threatens to cause a postoperative pathologic fracture, miniplates can be placed prior to closure to strengthen the weakened area of jaw. Patients with very large lesions and those who are severely medically compromised and unable to withstand a prolonged oral surgical procedure may be best treated by marsupialization (decompression) of the cyst allowing bony regeneration. The cyst is decompressed and the lining sutured to the oral mucosa. The cyst cavity should gradually regress, but the neck of the cyst needs to be held open using a pack or a bung. This process is often slow, taking many months for resolution of the cyst, and a further procedure may be required to remove the residual lining.

Residual cysts

Residual cysts develop from residual periapical infection or from cyst fragments left following extraction of a non-vital tooth. They present with similar features to the radicular cyst and radiographically are found as isolated, circumscribed, unilocular radiolucent lesions in the alveolar process but without an obvious causative tooth (Fig. 31.2). They require enucleation or marsupialization as above.

Lateral periodontal cysts

These lesions are usually asymptomatic and identified radiographically between the roots of teeth often in the mandibular canine and premolar area. The adjacent teeth are vital and the cysts are thought to arise from the cell rests within the periodontal ligament. The botyroid odontogenic cyst and glandular odontogenic cyst are variants of the lateral periodontal cyst and are both uncommon. Botyroid odontogenic cyst is found in adults of 50 years and older,

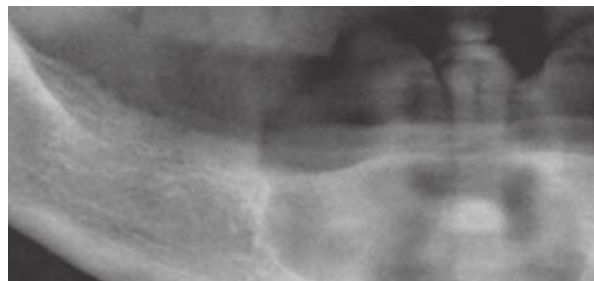


Fig. 31.2 Residual cyst from lower second premolar.

and involves the bicuspid area of the mandible. The glandular odontogenic cyst may be multilocular and is reported to be prone to recurrence. Treatment is usually by enucleation, although on occasion extraction of the involved tooth may be required.

Dentigerous cysts

Dentigerous or follicular cysts account for approximately one sixth of dental cysts. They are a fluid-filled expansion of the dental follicle and are attached to the crown of the tooth at the cemento-enamel junction.



Fig. 31.3 Dentigerous cyst arising from lower left third molar. Note displacement of the tooth and the inferior dental canal by the enlarging cyst.

As the cyst expands it prevents the eruption of the tooth and may even displace the tooth into the jaw. The most commonly involved teeth are reported to be the mandibular third molar and maxillary canine.

Dentigerous cysts are more common in men than women and usually these cysts present as an asymptomatic swelling, found when non-eruption of a tooth is investigated or incidentally with panoramic survey radiographs. Infection is uncommon, but it may present as a dentoalveolar or facial abscess.

Radiographically, a circumscribed, unilocular, radiolucent area is seen with a thin radiopaque lamina dura associated with the crown of the involved tooth. The tooth and adjacent structures may be displaced (Fig. 31.3). Cysts within the maxilla may occupy the maxillary sinus (Fig. 31.4).

Treatment is by removal of the causative tooth and enucleation of the cyst lining and the cavity is managed as described for radicular cysts. In those with very large cysts who are infirm, marsupialization may be the treatment of choice. If the tooth is to be retained for orthodontic reasons, it may also be advantageous to marsupialize the cyst.

Eruption cyst

This soft cystic swelling, usually blue or purple in color, is seen in children. It is found on the alveolus overlying an erupting tooth. The cyst most likely arises from the enamel organ. They are uncommon



Fig. 31.4 Dentigerous cyst of maxilla involving maxillary sinus: occipitomeatal radiograph and MRI.

and many burst spontaneously. Should the cyst not resolve quickly, the overlying tissue may need to be removed to allow the tooth to erupt. Radiographs are usually unnecessary, but if taken may show a lucent lesion at the alveolar margin in association with the crown of the tooth.

Gingival cysts

Two varieties of gingival cyst are found. In newborns small nodules are frequently seen on the alveolar crest. These lesions arise from proliferation of the cell rests of Serres. In the majority of cases they resolve spontaneously but persistent lesions may require local excision or curettage. In adults, usually in the fifth decade, soft tissue cysts can occasionally be found overlying the alveolus. These may cause local resorption of bone resulting in small saucer-shaped depressions in the alveolar plate. These cysts are treated by local excision.

Odontogenic keratocyst (primordial cyst, keratocystic odontogenic tumor)

The World Health Organization reclassified the odontogenic keratocyst as a benign tumor with the new title of keratocystic odontogenic tumor in 2006. It is discussed under this title in Chapter 32.

Calcifying epithelial odontogenic cyst

This rare cyst, first described by Gorlin *et al.* in 1962, may be found at any age and in either gender. It may be intraosseous or be found within the gingiva eroding the jaw. The radiographic appearance may have little calcification within it in early lesions, giving a similar appearance to other jaw cysts, but with increasing calcification may have the appearance of an odontome or calcifying odontogenic tumor. It has a benign behavior but can be aggressive and recur. Initial treatment is usually by enucleation and curettage.

Malignancy within odontogenic cysts

This is very rare but a few cases are reported of malignancy in the wall of dental cysts. Such lesions should be managed as primary malignant tumors once the diagnosis is established.

Non-odontogenic cysts

Fissural cysts

Fissural cysts were originally described to classify cystic lesions found in areas of what were believed to

be embryonic fusion. With more detailed studies these lines of fusion are believed not to exist and close examination of previous cases shows that most of these cysts actually have an odontogenic cause. However, the names are occasionally used for descriptive purposes only.

Globulomaxillary cysts

The term globulomaxillary cyst is an umbrella term used for cystic lesions appearing between the upper lateral incisor and canine tooth. It was originally felt to arise from epithelium due to embryonic fusion of the maxillary process with the nasal process, but this version of embryonic fusion has no supporting evidence. Many reported cases are in fact apical cysts related to non-vital lateral incisors or other cystic lesions. Treatment is by enucleation.

Median mandibular cyst

This is a term for cystic lesions in the midline of the jaw. Original proposals for this being of embryonic origin have now been disproved and, rather like the globulomaxillary cyst, most cases are in fact of odontogenic origin.

Nasopalatine cysts

Nasopalatine or incisive canal cysts arise from the epithelial remnants of the embryonic nasopalatine ducts. This occurs during the creation of the incisive canal by the fusion of the premaxilla with the palatine processes of the maxillary bones. The etiology of cyst formation may be local inflammatory processes such as infection and trauma. Nasopalatine cysts are more common in males than females and in the fourth to sixth decades. The cyst presents as a symmetrical swelling behind the upper incisors in the midline. Rarely, a fistula can be found leading to the incisive canal. Radiographs show a midline round or oval radiolucent lesion which may displace the roots of the central incisors laterally (Fig. 31.5). Accurate vitality tests are essential to avoid misdiagnosis. It is difficult to differentiate a small nasopalatine cyst from a large incisive canal, but it has been proposed that an



Fig. 31.5 Nasopalatine cyst.

incisive canal of less than 6mm on occlusal radiograph is within normal limits provided the patient has no symptoms nor abnormal clinical findings. Treatment is by enucleation.

Non-epithelially lined cysts

Stafne/static bone cyst

This classic radiolucency is found in the mandible as an incidental finding on survey radiographs such as orthopantomograms and lateral obliques. A small, smooth outlined lesion is found beneath the second and third molar teeth and below the inferior dental canal. The lesion is asymptomatic and is not a true cyst. It is an invagination of the medial aspect of the mandible usually containing salivary tissue, although lymphoid tissue has been reported. Occasional cases are reported of Stafne-type lesions occurring more anteriorly in the mandible. In difficult cases radiographic assessment can be made with either fine cut coronal computed tomography (CT) scan to determine the bony anatomy and the soft tissue signal within the cavity or by magnetic resonance imaging (MRI) scan. Biopsy and exploration are not indicated.

Aneurysmal bone cysts

This non-epithelially lined cyst-like lesion occurs occasionally in the jaws. It is more common in the mandible than maxilla. The etiology is unknown, but essentially it is a vascular malformation that arises from a prior unrelated lesion. It presents in adolescents and young adults as a swelling, which is painful in 50% of cases, or as an incidental finding on a panoramic radiograph. Radiographically it may be a uni- or multilocular lesion with irregular outline and occasional displacement of tooth roots. Histopathologically, the lesion contains many giant cells and can be indistinguishable from central giant cell granuloma. This feature has led to the idea that aneurysmal bone cysts are a vascular variation of central giant cell granuloma. Treatment is by curettage although the lesions can recur. Resection and adjuvant cryotherapy have been proposed for large or recurrent lesions.

Solitary bone cyst

Solitary bone cysts have been given many titles including simple bone cyst, traumatic bone cyst, hemorrhagic bone cyst, and latent bone cavity. All these terms refer to the same lesion, a non-epithelially lined space that may be found to contain a small amount of straw-colored fluid but may have no fluid within it. It is found most often in the long bones and rarely in the jaws. Its etiology is unknown, but trauma has been proposed, following which there is intramedullary bleeding and clot formation. However,

instead of the normal process of resorption of the clot and bone formation, the clot liquefies leaving a cavity. It is then proposed that the liquid content decreases with time and the lesion becomes filled with gas. The oxygen from this is absorbed into the blood leaving nitrogen that has been found on analysis. It is most commonly found in adolescents as a painless swelling or incidental finding on radiographs affecting the mandible. Females are more commonly affected than males (3:2). It has rarely been described in association with florid cemento-osseous dysplasia. Radiographs show a radiolucency that extends between the roots of involved teeth. One explanation for their rare appearance after adolescence is that these lesions spontaneously resolve. In longstanding or large lesions treatment is by curettage of the cavity, which results in clot formation and complete bony infill.

Soft tissue cysts

Nasolabial cysts

This is a rare cyst of the soft tissues with unknown etiology. Like the globulomaxillary cyst it has been proposed to arise from embryonal fusion but this has no evidence to support it. This cyst is commonest in the fourth and fifth decades and is more common in females (male:female 1:4). It is found as a swelling at the nasal fold and can become quite large before being detected causing distortion of the alar rim and local resorption of the maxilla. Treatment is by excision. It can be lined by squamous or respiratory epithelium.

Sublingual dermoid and dermoid cysts

Sublingual dermoid cysts are soft tissue cysts found in the midline beneath the tongue and as far down as the hyoid bone. These cysts are believed to arise from epidermal cell rests resulting from the fusion of embryonal processes. Unlike mucous retention cysts they are deeply placed and filled with keratin, giving a much firmer feel. Initially they are asymptomatic but as they increase in size they start to affect speech and swallowing if above the mylohyoid, where they bulge into the floor of the mouth. Those cysts arising below mylohyoid appear as a swelling in the neck. Treatment is by excision.

Other small dermoid cysts may be found within the skin of the head and neck and implantation dermoid cysts can arise from local trauma where skin is driven into the subepithelial layers. These lesions are treated by surgical excision.

Branchial lymphoepithelial cysts

Branchial cysts arise from incomplete obliteration of branchial clefts or epithelial inclusions within lymph

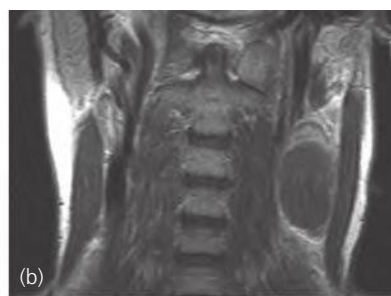
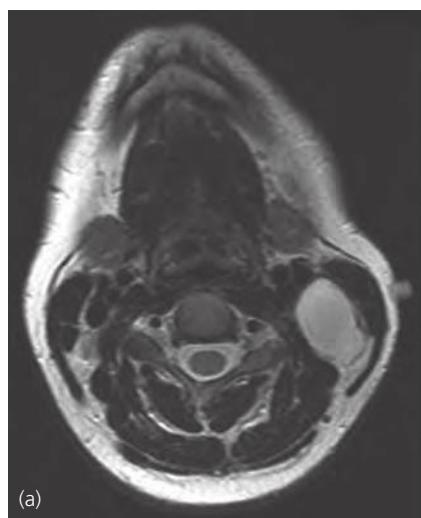


Fig. 31.6 Branchial cyst: axial and coronal MRI views showing cyst deep to sternomastoid.

nodes. Lymphoepithelial cysts are found in the floor of the mouth and posterior tongue. Branchial cysts usually present as asymptomatic swellings in the lateral aspect of the neck at the anterior border to the sternomastoid. Rarely, they present with acute infection. Branchial cysts are commonest in children and young adults, but can occur in older patients. In those over 40 years, cysts presenting in this fashion should be investigated for tonsillar or tongue base carcinoma as cystic metastasis for such lesions is the commonest cause of lateral neck cysts in this age group. Rare cases of primary branchial cyst carcinoma are reported in the literature.

In those with simple branchial cyst the neck should be imaged with MRI scan (Fig. 31.6) and the cyst excised. Any track to the pharynx should also be excised.

Thyroglossal cysts

These cysts arise from the thyroglossal duct, which should break down following the migration of the thyroid gland from the foramen caecum in utero. These cysts present as midline swellings anywhere from the tongue base to the thyroid gland. They are usually asymptomatic. Due to the attachment of the tract to the hyoid bone and the tongue, the cysts move on swallowing and protrusion of the tongue. Investigation is by MRI scan and fine-needle aspiration. Thyroid scans will occasionally show these cysts to be active, but usually patients have adequate other functioning tissue. Solid lesions may, however, represent all of the patient's functioning thyroid tissue and a thyroid scan is required before the removal of such a lesion. Cases of carcinoma within thyroglossal cysts are reported. Treatment for uncomplicated thyroglossal cyst is by the Sistrunk procedure, which involves excision of the cyst and the central portion of the hyoid bone.

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Chapter 32

Odontogenic and Non-odontogenic Tumors of the Jaws

R. Bryan Bell

This chapter will cover the classification, frequency of occurrence, etiology, molecular biology, histogenesis, treatment, and prognosis for odontogenic and non-odontogenic tumors:

- Benign epithelial odontogenic tumors include ameloblastomas, keratocystic odontogenic tumors, calcifying epithelial odontogenic tumors, and adenomatoid odontogenic tumors.
- Benign mixed odontogenic tumors include ameloblastic fibroma, ameloblastic fibro-odontoma, odontomas, odontoameloblastomas, calcifying cystic odontogenic tumors, and dentinogenic ghost cell tumors.
- Mesenchymal odontogenic tumors include odontogenic fibromas, odontogenic myxomas, and cemento-blastomas.
- Non-odontogenic tumors include the ossifying fibroma, fibrous dysplasia, osseous dysplasia, giant cell lesions, and simple bone cysts.
- Malignant odontogenic tumors include the ameloblastic carcinoma, primary interosseous squamous cell carcinoma, clear cell odontogenic carcinoma, ghost cell odontogenic carcinoma, and odontogenic sarcomas.
- Non-odontogenic malignant tumors include osteosarcoma, chondrosarcoma, Ewing sarcoma, malignant peripheral nerve cell tumors, and metastatic tumors.

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Introduction

Odontogenic and non-odontogenic tumors of the jaws are a relatively rare and heterogeneous group of

benign and malignant neoplasms, hamartomas, and other bone-related lesions that demonstrate great variability in etiology, biologic behavior, and clinical significance. The single commonality amongst these

cellular proliferations is that they may occur in the jaws. Odontogenic tumors, by definition, are derived from tooth-related tissues and, although they have been reported in tooth-bearing structures such as dermoid cysts and teratomas, originate only in the maxilla or mandible. The majority of these disorders are benign, and while they may be locally aggressive, only very rarely do they have the ability to metastasize. Non-odontogenic jaw tumors, on the other hand, develop from the epithelium and/or mesenchyme of a wide variety of tissues in the body, often originate in non-tooth-bearing facial bones, and may develop in other sites outside of the head and neck. Some of these are of little clinical significance and require nothing more than observation or local excision, while many of the malignant variants mandate multimodal therapy and portend a poor prognosis for survival.

Odontogenic tumors

Odontogenic tumors (OT) are derived from tooth-forming elements and for the purposes of this chapter include lesions that are not definitively neoplastic. Even using this broad definition, OTs are rare lesions that account for <2–3% of all oral and maxillofacial specimens sent for diagnosis to oral pathology services.¹ It has been estimated that if viewed as a percentage of all tumors in the human body, this figure may only be 0.002–0.003%.²

Odontogenesis occurs through a complex process involving the enamel organ, the dental follicle, and the dental papilla (Fig. 32.1). The enamel organ is an epithelial structure that is derived from oral ectoderm. The dental follicle and dental papilla are derived from neural crest cells and are therefore considered ectomesenchymal in nature. Odontogenic tumors demonstrate varying inductive interactions between the odontogenic epithelium and odontogenic ectomesenchyme and are typically subclassified by their tissue of origin.

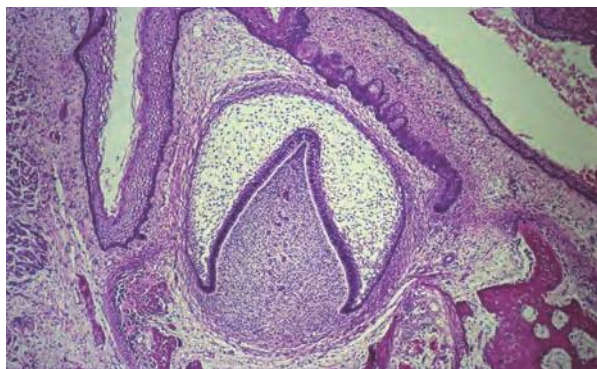


Fig. 32.1 The “bell stage” of tooth development. (Courtesy of F. James Kratochvil III, DDS.)

Classification

The first significant attempt to classify odontogenic tumors probably occurred in 1869 when the French physician and professor of pathology and clinical surgery, Pierre Paul Broca,³ suggested that tumors arising from the dental formative tissues (he termed them odontomes) were organized according to the stage of development of the tooth when abnormal growth commenced. Later modified by Malassez in 1885,⁴ neither of these classification schemes gained much traction in the international scientific community. In 1888, Bland-Sutton⁵ provided what might be considered the foundation for our current system of taxonomy by basing his classification on the particular cells of tooth germ from which the tumor arose. Gabell, James, and Payne⁶ further modified Bland-Sutton’s classification by recognizing three distinct groups of odontomes: epithelial, composite, and connective tissue. In the years to follow, the term odontome was gradually replaced by terms more compatible with general pathological use.⁷

In 1946 Thoma and Goldman⁸ described our modern classification of odontogenic tumors based on the tissue cell of origin using contemporary terminology. Subsequently revised by Pindborg and Clausen⁹ and Gorlin and colleagues,¹⁰ these classifications provided a structured means to catalog and study lesions but did not provide the clinician with a useful guide to clinical decision making. In 1971, under the leadership of Pindborg and Kramer, the World Health Organization¹¹ (WHO) published a classification that distinguished between lesions and tumors of the odontogenic apparatus, neoplasms and other lesions of bone. Furthermore, it subclassified these disorders according to their biologic behavior as being benign or malignant. The WHO classification was revised in 1992 by Kramer, Pindborg, and Shear¹² as a reflection of increased knowledge of the origin and inductive interaction of odontogenic tissues, but almost immediately evoked persistent criticism.^{13–15}

A better understanding of the pathogenesis and progression of odontogenic tumors is currently being developed as a result of chromosomal and molecular genetic analyses. Ultimately, this information can be used to better predict the biologic behavior of a particular tumor, properly select the appropriate treatment, and develop novel forms of therapy directed against specific genetic alterations. Such studies are underway for various odontogenic cysts and tumors that, in part, led Philipsen and Reichart in 2002¹⁶ to suggest another classification change.

The new WHO classification of odontogenic tumors was published in July 2005 (Table 32.1)¹⁷ and made a number of important changes to terminology, benign or malignant classifications, and assignment to relevant subgroups.¹⁸ Previous WHO classifications described only tumor histology. The new publication includes other information such as definition,

Table 32.1 WHO histological classification of odontogenic tumors. Adapted from Barnes L, Eveson JW, Reichart P, Sidransky D *World Health Organization Classification of Tumours, Pathology and Genetics of Head and Neck Tumours*. Lyon: IARC Press, 2005; 284.

MALIGNANT TUMORS
Odontogenic carcinomas
Metastasizing (malignant) ameloblastoma
Ameloblastic carcinoma – primary type
Ameloblastic carcinoma – secondary type (dedifferentiated), intraosseous
Ameloblastic carcinoma – secondary type (dedifferentiated), peripheral
Primary intraosseous squamous cell carcinoma-solid type
Primary intraosseous squamous cell carcinoma derived from keratocystic odontogenic tumor
Primary intraosseous squamous cell carcinoma derived from odontogenic cysts
Clear cell odontogenic carcinoma
Ghost cell odontogenic carcinoma
Odontogenic sarcomas
Ameloblastic fibrosarcoma
Ameloblastic fibrodentino- and fibro-odontosarcoma
BENIGN TUMORS
Odontogenic epithelium with mature, fibrous stroma without odontogenic ectomesenchyme
Ameloblastoma, solid/multicystic type
Ameloblastoma, extraosseous/peripheral type
Ameloblastoma, desmoplastic type
Ameloblastoma, unicystic type
Squamous odontogenic tumor
Calcifying epithelial odontogenic tumors
Adenomatoid odontogenic tumor
Keratocystic odontogenic tumor
Odontogenic epithelium with odontogenic ectomesenchyme, with or without hard tissue formation
Ameloblastic fibroma
Ameloblastic fibrodentinoma
Ameloblastic fibro-odontoma
Odontoma, complex type
Odontoma, compound type
Odontoameloblastoma
Calcifying cystic odontogenic tumor
Dentinogenic ghost cell tumor
Mesenchyme and/or odontogenic ectomesenchyme with or without odontogenic epithelium
Odontogenic fibroma
Odontogenic myxoma/myxofibroma
Cementoblastoma
Bone-related lesions
Ossifying fibroma
Fibrous dysplasia
Osseous dysplasias
Central giant cell lesion (granuloma)
Cherubism
Aneurysmal bone cyst
Simple bone cyst

epidemiology, etiology, localization, clinical and imaging features, macroscopy, histopathology, tumor genetics, and prognosis.

Odontogenic cysts were not considered in the new 2005 classification, which means that the 1992 classification is still relevant for cysts of the jaws and maxillofacial region, with one notable exception: the odontogenic keratocyst (OKC) was renamed “keratocystic odontogenic tumor” (KOT) and was added to the odontogenic tumors category. The change in terminology is a reflection of contemporary clinical, molecular, and immunohistochemical data suggesting that the KOT is a benign neoplasm rather than a cyst. The nevoid basal cell carcinoma syndrome (NBCCS) or PTCH-gene has been mapped to chromosome 9q23.3-31 where it probably acts as a tumor suppressor.¹⁹ A two-hit mechanism in the pathogenesis of NBCCS and sporadic KOT demonstrated allelic loss of 9q22 with overexpression of bcl-1 and TP53 in NBCCS.²⁰ There is also evidence suggesting that the PTCH-gene is a significant factor in the development of sporadic KOT. Evolving research demonstrates that the loss of tumor suppressor genes may play an important role in the aggressive clinical behavior of KOTs, provides a partial explanation for their propensity to recur after conservative treatment, and supports the view that KOTs are indeed benign neoplasms.²¹⁻²³

Another important change in the 2005 WHO classification is related to malignant tumors of odontogenic origin. The metastasizing ameloblastoma was incorrectly described in the 1992 classification. It is now clarified in the 2005 document, which provides a clear distinction between the metastasizing ameloblastoma (containing benign histological features) and ameloblastic carcinoma. The diagnosis of an ameloblastic carcinoma is made in the presence of malignant histological features. In addition, the clear cell odontogenic carcinoma (CCOC), formerly clear cell odontogenic tumor, has been added to the list of malignant odontogenic carcinomas due to its newly recognized aggressive behavior.

The 2005 classification also recognizes the existence of variants, by using the plural term: ameloblastomas. Four ameloblastoma variants are now recognized: (1) solid/multicystic ameloblastoma; (2) extraosseous/peripheral ameloblastoma; (3) desmoplastic ameloblastoma; and (4) unicystic ameloblastoma. The demographics, clinical features, and biologic behavior of each of these entities are variable and probably contribute to the lack of consensus regarding optimal treatment.

The adenomatoid odontogenic tumor was formerly grouped with the “mixed odontogenic tumors” and has now been added to the benign tumors category with odontogenic epithelium and mature stroma without odontogenic ectomesenchyme.

The calcifying cystic odontogenic tumor is described with synonyms such as calcifying odontogenic cyst, Gorlin cyst, and calcifying odontogenic

tumor. Although there is no consensus regarding the continued separation of the cystic variants, it is possible that future revisions will lump this entity into a single term.

Two variants of the central odontogenic fibroma were described in the 1992 classification that is now termed “epithelial-poor” and “epithelial-rich”, based on histological findings.

The term “osseous dysplasias” has now been introduced which pools groups of bone-related lesions under one heading. The former terms of cementifying ossifying fibroma and cementifying osseous dysplasias have been changed to ossifying fibroma and osseous dysplasia respectively.

The genetic basis of head and neck tumors is complex, and knowledge of the mechanisms that drive neoplastic growth and development is in its infancy. As a result of chromosomal and molecular genetic analyses, a better understanding of the pathogenesis and progression of odontogenic tumors is being developed and will, no doubt, lead to future changes in the classification of these diverse cellular proliferations.

Molecular biology

Various genetic and molecular alterations appear to promote the development and progression of tumors via a series of complex processes. Recent studies have identified a number of molecular alterations that are responsible for the development and progression of odontogenic tumors and may provide further insight into their etiology, pathogenesis, and treatment (Table 32.2).²⁴

Oncogenes are normal cellular genes that contribute to neoplastic transformation through functions activated by gene amplification, translocation, or mutation. Many oncogenes have been described. Among other things, these oncogenes appear to function as growth factors, such as platelet-derived growth factor (PDGF) and fibroblast growth factor (FGF); growth factor receptors, such as epidermal growth factor receptor (EGFR), HER-2, and Ret; non-receptor tyrosine kinases (Src, Abl); serine/threonine kinases (Mos); signal transducers (Ras); and transcription factors (Myc, Fos). Ameloblastomas have been shown to be EGFR positive. Products encoded by Ras genes (p21Ras) have been shown to be preferentially expressed in the odontogenic epithelium of ameloblastomas, ameloblastic fibromas, and odontogenic myxomas.^{25,26} C-Myc oncoprotein is expressed predominantly in neoplastic cells neighboring the basement membrane of ameloblastomas.²⁷ Fos oncogene encodes a transcription factor participating in the control of cell proliferation and differentiation in ameloblastomas on cDNA microarray and subsequent real-time reverse transcriptase polymerase chain reaction, suggesting that these oncogenes play a role in the pathogenesis of odontogenic tumors via dysregulation of cell proliferation.²⁸

Table 32.2 Summary of molecules involved in the development and progression of odontogenic tumors. Adapted from Kumamoto M. Molecular pathology of odontogenic tumors. *J Oral Pathol Med* 2006; 35: 65–74.

Molecules possibly associated with tumorigenesis and/or tumor cell differentiation	
Oncogenes	<i>Ras, Myc, Fos</i>
Tumor-suppressor genes	<i>p53, APC</i>
Oncoviruses	HPV, EBV
Growth factors	TGF- α , - β , FGF-1, -2, HGF
Telomerase	Telomerase
Cell cycle regulators	Cyclin D1, P16 ^{INK4a} , p21 ^{WAF1/Cip1} , p27 ^{Kip1}
Apoptosis-related factors	Bcl-2 family, IAP family, Fas, TNF- α , p53
Regulators of tooth development	SHH pathway, Wnt pathway
Hard tissue-related proteins	Amdogenin, enamelin, ameloblastin, enamelysin, BSP, osteonectin, osteocalcin, osteopontin, BMP
Molecules possibly associated with tumor progression	
Cell adhesion molecules	E-selectin, ICAM-I, VCAM-I, E-cadherin, integrins, CD44
Matrix-degrading proteinases	MMP-I, -2, -9/IMP-I, -2, heparanase
Angiogenic factors	VEGF
Osteolytic cytokines	IL-I, -6, TNF- α , PTHrP, RANKL/OPG

HPV, human papilloma virus; EBV, Epstein–Barr virus; TGF, transforming growth factor; FGF, fibroblast growth factor; HGF, hepatocyte growth factor; IAP, inhibitor of apoptosis protein; TNF, tumor necrosis factor; SHH, Sonic hedgehog; BSP, bone sialoprotein; BMP, bone morphogenetic protein; ICAM, intercellular adhesion molecule; VCAM, vascular cell adhesion molecule; MMP, matrix metalloproteinase; TIMP, tissue inhibitor of metalloproteinases; VEGF, vascular endothelial growth factor; IL, interleukin; PTHrP, parathyroid hormone-related protein; RANKL, receptor activator of nuclear factor-KB ligand; OPG, osteoprotegerin.

Tumor-suppressor genes act as regulators of cell growth. Inactivation of these genes by mutations and/or loss of heterozygosity in both alleles results in tumor development. p53 gene is the most well studied, however other genes have been identified including retinoblastoma (RB), adenomatous polyposis coli (APC), ST-1, and patched (PTC) genes. Although several studies have shown p53, mutations are infrequent in benign ameloblastomas,^{29–31} p53 reactivity has been detected in ameloblastomas, malignant ameloblastomas, primary introsseous carcinomas, and ameloblastic fibrosarcomas.^{32–35} Furthermore, there is some evidence that hypermethylation of p16 may be involved in the malignant transformation of ameloblastoma.³⁶ More recent studies have demonstrated high levels of Ki-67, p53 and p63 in KOTs, thus favoring its tumorigenesis.^{37,38} Immunohistochemical reactivity for APC is lower in benign and malignant ameloblastomas than in tooth germs.³⁹ Although RB has not been investigated in odontogenic tumors, there is evidence to suggest an association between tumor-suppressor genes and the development of odontogenic tumors via aberrant control of cell proliferation.⁴⁰

A comprehensive review of the molecular biology of odontogenic tumors is well beyond the scope of this chapter. The reader is referred to other, more definitive reviews of the subject.²⁴ Suffice it to say, the characteristics of odontogenic tumors appear to depend on the molecular mechanisms associated with tooth development, bone metabolism, and the malignant potential of tumors. Future molecular studies are underway that are investigating the

genomic- and proteomic-based profiling that will hopefully lead to the development of new therapeutic modalities.

Treatment options for benign tumors of the jaws

Enucleation and curettage

Enucleation and curettage has been the traditional and time-honored method for managing odontogenic cysts and some jaw tumors. The technique offers the patient a minimally invasive procedure, with little associated morbidity and few complications. Most odontogenic cysts can be effectively removed by simple enucleation of the cystic lining and meticulous curettage of the bony cavity. However when used alone, this technique is usually inadequate for tumors with true neoplastic potential and its use in entities such as ameloblastoma or keratinizing odontogenic tumor should be accompanied by adjuvant treatment, such as peripheral osteotomy, cryotherapy, or chemical fixation with Carnoy's solution.

Wide exposure is necessary to allow complete access to the bony cavity (Fig. 32.2a). Every effort must be made to thoroughly remove the cyst lining as well as epithelial remnants that may be present between the cyst wall and overlying mucosa. One of the difficulties encountered when attempting to remove these cysts lies in the nature of the thin cystic lining which, at times, is readily removed *in toto*, but more often comes out in multiple soft tissue

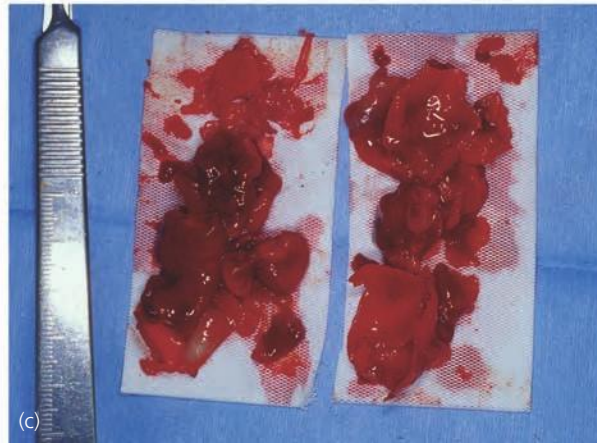


Fig. 32.2 Enucleation and curettage. (a) Exposure of the cyst to allow complete access to the bony cavity. (b) Complete, intact removal of the cyst. (c) Complete, fragmented removal of the cyst.

fragments (Fig. 32.2b, c). The inferior alveolar nerve can be routinely spared and root canal therapy is almost never indicated. Most of the time, the mucosa can be closed primarily, without the need for bone grafts or packing material. Removal of a large cyst will occasionally weaken the remaining bony integrity, placing it at risk for pathologic fracture. This can be managed with 6 weeks of intermaxillary fixation and/or placement of a reconstruction plate. Enucleation and curettage is a reasonable method for the primary treatment of small, unilocular cysts that are typically not biopsied prior to definitive treatment.

Enucleation and peripheral osteotomy

Enucleation with peripheral osteotomy is an extension of the curettage technique described above. It involves the use of a rotary instrument to remove bone adjacent to the cystic lining, theoretically facilitating removal of all residual epithelium and/or daughter cysts (Fig. 32.3). Some authors have described the use of methylene blue to aid in identification of appropriate bony margins.⁴¹ The technique can stand alone or include chemical or thermal fixation of the interior of the bony cavity. The advantage of peripheral osteotomy is that it provides an additional “margin” of bone removal during excision of the lesion and can potentially alleviate the need for adjunctive measures. The disadvantages are that it places other anatomic structures at risk of injury, i.e. teeth and the inferior alveolar nerve, and may further weaken the jaw structure. There are only limited published data comparing this technique with other tech-

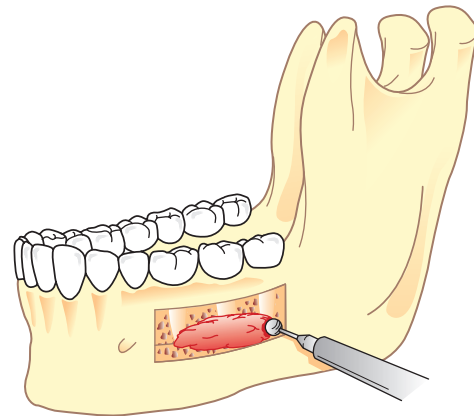


Fig. 32.3 Illustration of peripheral osteotomy.

niques, with or without chemical or thermal fixation, therefore recurrence rates for treatment of odontogenic tumors are not truly known. However, the use of peripheral osteotomy for treatment of a number of odontogenic tumors has merit in that it can facilitate a more “radical” surgery than curettage but is less morbid than resection.

Marsupialization

Marsupialization is a technique advocated by some authors that is designed to initially decompress and shrink the cyst or tumor prior to definitive removal, generally by enucleation and curettage several months later. The primary advantage of this method is to minimize the surgical defect that is created by removal of the cystic lesion. This technique has been observed to cause inflammation and subsequent thickening of the cystic lining which facilitates its ultimate removal. Various inflammatory mediators may play a role in cyst volume reduction. Recently,

marsupialization was shown to inhibit interleukin-1alpha expression within the lining of KOTs, thereby halting epithelial cell proliferation and decreasing the size of the cystic tumor.⁴² The technique is performed by “de-roofing” the cyst and either repeatedly packing the cavity with gauze or by simply placing a tube, catheter or drain in order to facilitate gradual decompression and shrinkage of the defect. The pack or drain is left in place for 2–3 months, depending on the size of the lesion, and is followed by an enucleation procedure.

Technique

The author favors removal of overlying bone and cyst roof, creating an opening into the cyst that is as wide as possible. A 1" (2.5cm) Penrose drain is cut to a length just long enough to reach the depth of the lesion. Its periphery is sutured to the gingival and alveolar mucosa around the defect with approximately eight 2-0 silk sutures (Fig. 32.4). The patient is instructed in wound irrigation with saline and the drain is allowed to spontaneously exfoliate, which usually occurs within 1–2 months. In cases of extensive mandibular destruction, an ipsilateral coronoidectomy may help avoid pathologic fracture.

Intralesional adjunctive therapy: chemical fixation

Remnants of the dental lamina have been hypothesized to play a role in the etiology of some odontogenic tumors.⁴³ The presence of epithelial islands within the mucosa overlying the cyst as well as the bony cavity has prompted the use of various surgical strategies to adjunctively treat the surrounding tissue in an effort to eradicate residual disease and minimize recurrence. The use of such adjuvant measures is perhaps best studied in the treatment of KOTs. Stoelinga, Voorsmit, and their colleagues from Denmark advocate excision of the overlying mucosa and have popularized the use of Carnoy’s solution as a chemical tissue fixative for treatment of KOTs.^{44–47} Carnoy’s



Fig. 32.4 Marsupialization of a keratinizing odontogenic tumor (KOT).

Table 32.3 Carnoy’s solution (Cutler and Zollinger).

Absolute alcohol	6ml
Chloroform	3ml
Glacial acetic acid	1ml
Ferric chloride	1g

solution is a mixture of absolute alcohol, chloroform, glacial acetic acid, and ferric chloride that penetrates bone to a predictable, time-dependent depth, without injuring the neurovascular structures (Table 32.3). A 5-minute application will penetrate bone to a depth of 1.54mm, nerve to a depth of 0.15mm and mucosa to a depth of 0.51mm (Table 32.4).⁴⁶ Because most residual cells and daughter cysts from locally recurrent lesions are adjacent to the main lesion, it is likely that fixation of vital bone need only extend for 2–3mm beyond the enucleated lesion. Theoretically, enucleation of the cyst wall, excision of the overlying mucosa, and treatment of the surrounding tissue with Carnoy’s solution should sufficiently remove the cystic lesion along with any epithelial remnants remaining in the area. Application of the Carnoy’s solution can be either prior to enucleation or afterwards. Voorsmit’s original description of the technique called for treatment of the cyst before enucleation which causes a “tanning effect” of the lesion thereby facilitating complete removal. The author’s experience, however, is that treatment of the bony cavity after enucleation allows for easier removal of the cyst lining and better identification of soft tissue remnants. There is no evidence in the literature to support one method being superior to the other. Furthermore, there is nothing to suggest that coating the neurovascular bundle with petroleum jelly or the like affects neurosensory outcome.

Technique

Following cyst enucleation through a generous access window and a peripheral osteotomy, the defect is filled with ¼" (6mm) or ½" (12mm) ribbon gauze (Fig. 32.5). Care is taken to protect the surrounding soft tissues with gauze sponges or towels (Fig. 32.6). A syringe and angiocath are used to administer the Carnoy’s solution, soaking the gauze thoroughly. After a period of 5 minutes the gauze mass is clamped with forceps and is manipulated within the cavity to ensure complete application to all walls. The gauze is then removed and the cavity irrigated with copious amounts of saline solution. Primary closure is achieved and strong consideration is made to perform a primary bone graft in medium and large-sized tumors.

Intralesional adjunctive therapy: cryotherapy

Cryosurgery is a method of treating disease by the production of freezing temperatures in tissue.

Table 32.4 Carnoy's solution: depth of penetration (mm). Adapted from: Voorsmit RACA. The incredible keratocyst: a new approach to treatment. *Dtsch Zahnarztl* 1985; 40: 641–4.

Period of application	Keratocyst wall	Mucosa	Nerve	Bone
	0.75*	1.64*	2.38*	4.57*
1 minute	0.18	0.13	0.08	0.72
5 minutes	0.75	0.51	0.15	1.54
60 minutes	>0.75	1.19	0.33	1.81

* Mean full-thickness of tissue in mm.

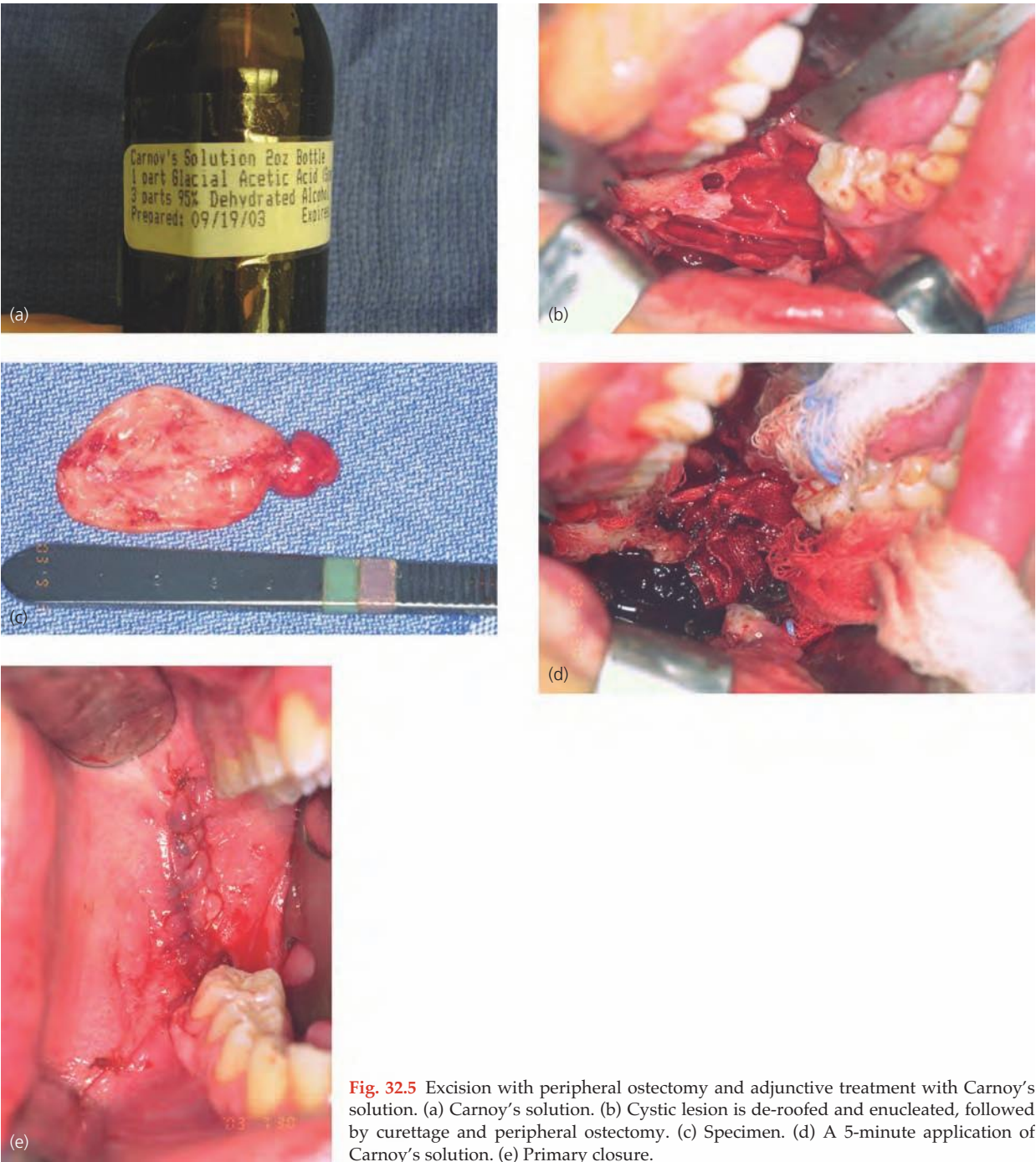


Fig. 32.5 Excision with peripheral ostectomy and adjunctive treatment with Carnoy's solution. (a) Carnoy's solution. (b) Cystic lesion is de-roofed and enucleated, followed by curettage and peripheral ostectomy. (c) Specimen. (d) A 5-minute application of Carnoy's solution. (e) Primary closure.



Fig. 32.6 Photograph of patient with Carnoy's burn to the lip. These burns typically heal without a scar, but can be avoided with proper protection.

Cryosurgical techniques were first described in 1850, modernized in the 1960s, and revitalized in the 1990s, and they are used for tumors in diverse sites.⁴⁸ Various authors have advocated the use of cryotherapy as an adjunct to the surgical treatment of benign cysts and tumors of the jaws over the last three decades.^{49–54} Cryosurgery for osseous lesions produces cellular necrosis in bone while maintaining the inorganic osseous framework.⁵⁵ Predictable cell lysis occurs in temperatures below -20°C and is caused by direct damage from intracellular and extracellular ice crystal formation, osmotic disturbances, and electrolyte imbalance.⁵⁶ Similar to chemical fixation with Carnoy's solution, cryosurgery allows for removal of the cyst or tumor by enucleation and curettage followed by treatment of the surrounding tissue. A single 1-minute freeze produces a depth of bone necrosis of 1–3 mm depending on the technique.⁵³ The techniques acceptable for oral cryosurgical use include the cryoprobe with water-soluble jelly and liquid nitrogen spray. The advantage of a cryoprobe with jelly is that it is possible to freeze irregular, gravity-dependent portions of the bony cavity. The disadvantage is that there is non-uniform freezing. The advantage of liquid nitrogen spray is potent, uniform freezing. The disadvantage is the potential for damage to surrounding tissues. Immediate bone grafting has been recommended for defects greater than 4 cm that are treated with cryotherapy.⁵⁷ Bone grafting is thought to decrease complications such as wound dehiscence and pathologic fracture, while simultaneously providing greater residual bone height and density, thus facilitating implant placement.

Technique

The preparation for cryotherapy begins with placing the patient on a fully adjustable operating table with the patient's head on a Mayfield horseshoe head holder, in order to allow intraoperative repositioning. An aggressive removal of overlying bone and any soft tissue that might harbor cyst epithelium is per-

formed. The cyst is then removed as thoroughly as possible, although cysts extending into the condyle often defy complete removal. In anticipation of wound closure following later bone graft placement, a removal of the superior quarter of the mandibular height is performed as in a piecemeal marginal mandibulectomy. Coronoidectomy may be performed to decrease bite force and avoid pathologic fracture. Malleable retractors are positioned so as to self-retain gauze sponges to protect and insulate the soft tissues (Fig. 32.7). If a cryoprobe is to be used, a portion of the defect is filled with water-soluble jelly. The nitrous oxide cryoprobe is activated once it is immersed in

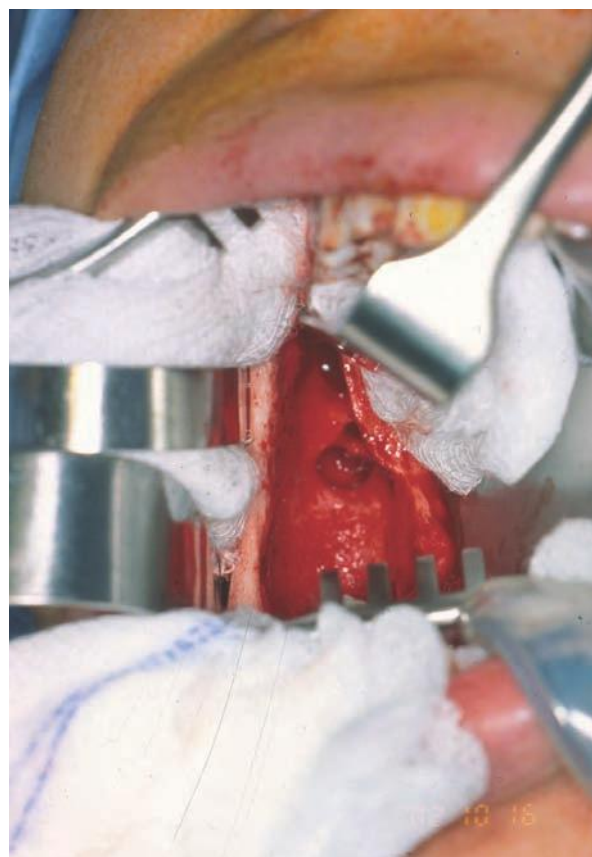


Fig. 32.7 Cryotherapy preparation. Malleable retractors are positioned so as to self-retain gauze sponges to protect and insulate the soft tissues.

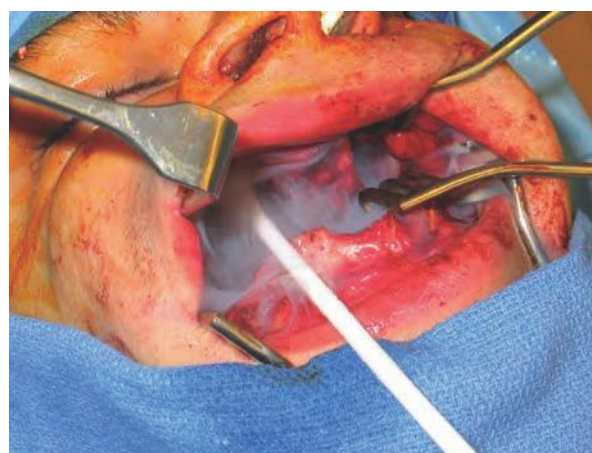


Fig. 32.8 Cryo-applicator in water-soluble jelly.



Fig. 32.9 Open funnel cryo-application system.

the jelly-filled cavity (Fig. 32.8). The freeze process is continued for 2 minutes and then the ice ball is allowed to thaw. The freeze process is repeated three times. A significant limitation of the cryoprobe technique is the limited area of freezing that occurs, therefore the technique should be used on small cavities.

Alternatively, a commercially available cryospray may be used, or an even simpler open funnel system may be assembled (Fig. 32.9). The operating table is then positioned until inspection of the meniscus of a trial filling of the defect with saline demonstrates that the entire defect is flooded with saline. Larger defects and those that involve both the mandibular body and ascending ramus may require two separate positionings and treatments. Using a ladle and funnel or a commercially available applicator, the defect is then carefully filled with liquid nitrogen. Following three freeze-thaw cycles, the defect is filled with autogenous bone graft material and an everted, multiple layer closure of the overlying soft tissue is performed.

Resection

The aggressive clinical behavior of some odontogenic and non-odontogenic jaw tumors is well recognized. Although rare, extension into vital anatomic regions occurs and can involve the skull base,^{58–62} infratemporal fossa,^{63,64} and/or orbit.^{65–69} Composite, segmental or marginal resection should be considered for some large primary or recurrent benign tumors, tumors involving the orbit, posterior maxilla, pteryopalatine fossa, skull base or infratemporal fossa, and malignant tumors.

Technique

The mandible or maxilla is skeletonized in a subperiosteal or suprapariosteal plane (depending on the histological diagnosis and extraosseous tumor extension), taking care to excise periosteum that might be contaminated by tumor perforation of cortical bone. The inferior alveolar nerve can be preserved if appro-

priate, and a segmental or marginal resection performed with care to maintain 0.5–1 cm tumor-free margins. For patients with recurrent lesions, periosteum and/or mucosa is resected with bone in areas of perforation and along previous incision lines. Lesions located at the retromolar trigone can often be marginally resected using a transbuccal approach (Fig. 32.10). Following soft tissue excision of the overlying mucosa, a stab incision is made in the cheek that facilitates placement of the reciprocating saw blade. The handpiece is then attached to the saw blade and the marginal resection performed. The soft tissue and bone are removed *en bloc*. Occasionally, the residual bone volume is insufficient to allow function and may be at risk for pathologic fracture, either intraoperatively or postoperatively. These patients may benefit from reinforcement with plate fixation.

If segmental resection is to be performed, a reconstruction plate should be adapted prior to resection, if possible, or a stereolithographic model used to prebend the plate. Following resection, the decision to perform delayed non-vascularized bone graft reconstruction or immediate reconstruction with free tissue transfer should be made depending on a number of patient-related, tumor-related, and defect-related factors.

Reconstruction

Ideally, benign or malignant ablation should be followed by immediate oromandibular or palatamaxillary reconstruction to re-establish function and achieve an acceptable cosmetic result. Immediate bony reconstruction of smaller defects can be performed by using non-vascularized autogenous bone grafts harvested from the ilium. Important factors that have been found to affect graft survival include the length of the mandibular defect, timing of the reconstruction (immediate versus delayed), pre- or postoperative radiation therapy, postoperative recipient site complications, malignant diagnosis, intraoral communication, estimated blood loss, the number of days on postoperative antibiotics, and the use of soft tissue flaps (Table 32.5).⁷⁰ In the primary setting, however, microvascular transfer of free osteocutaneous flaps from the fibula, iliac crest, radial forearm, or scapula seems to provide the most reliable means for reconstruction of composite or segmental defects. More than 15 years have elapsed since the introduction of microvascular surgery to head and neck reconstruction and a number of studies with large series of patients demonstrate that in experienced hands the success rate for re-establishing mandibular continuity is greater than 95%.^{71–80}

Length of the defect has been shown to be an important factor in bone graft survival for mandibular reconstruction. Foster *et al.*⁸¹ critically evaluated the use of non-vascularized grafts compared to microvascular composite free flaps in reconstruction of mandibular continuity defects and found that suc-

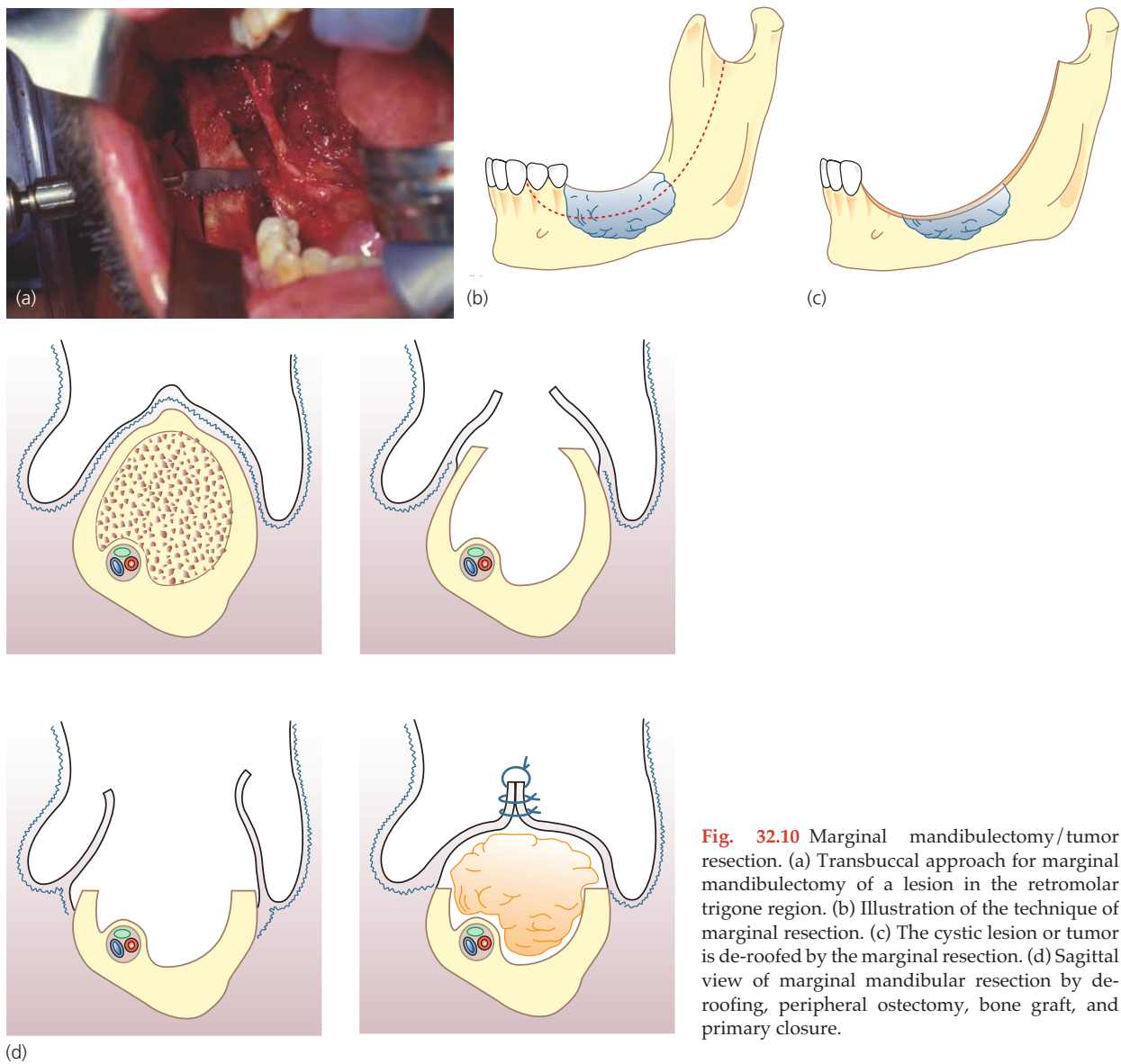


Fig. 32.10 Marginal mandibulectomy/tumor resection. (a) Transbuccal approach for marginal mandibulectomy of a lesion in the retromolar trigone region. (b) Illustration of the technique of marginal resection. (c) The cystic lesion or tumor is de-roofed by the marginal resection. (d) Sagittal view of marginal mandibular resection by de-roofing, peripheral osteotomy, bone graft, and primary closure.

Table 32.5 Factors affecting bone graft survival.⁷⁰

Timing
Length of defect
Radiation
Postoperative complications
Malignant diagnosis
Intraoral communication
Estimated blood loss
Number of days on antibiotics
Use of soft tissue flaps

cess was dependent in part on the length of the defect. Success rates in terms of osseous union using non-vascularized bone grafts decreased significantly in those patients with defects greater than 6cm. The overall success rate of non-vascularized bone grafts was approximately 69% while the overall success rate of vascularized bone flaps was 96%. Additionally, it took an average of 2.3 operations to complete the

reconstruction for those patients with non-vascularized bone grafts as opposed to only 1.1 operations for those patients with vascularized bone flaps. A follow-up study at the same institution by Pogrel *et al.*⁸² reported an increased rate of failure for continuity defects greater than 9cm and recommended that vascularized grafts be used in such cases to improve outcome (Fig. 32.11). These findings were further supported in a more recent study by August *et al.*⁷⁰ who reviewed a series of 70 cases of mandibular reconstruction performed at their institution over a 15-year period in an attempt to identify factors that affect the long-term outcome. All except two patients underwent reconstruction using corticocancellous bone grafts, occasionally combined with a sternocleidomastoid flap. Overall success, defined as wound closure, freedom from infection, bony continuity, and maintenance of bulk, was remarkably similar to that of Foster *et al.*, at 68.8%. The failure rate was highest in defects with an average length of 9.9cm, whereas the average length of successfully reconstructed defects was 7cm. For these reasons the author

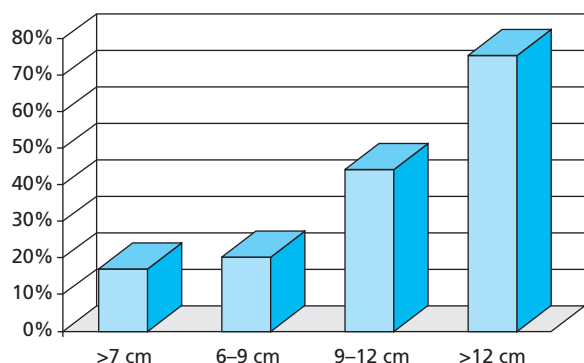


Fig. 32.11 The failure rate of non-vascularized bone grafts when related to the length of the graft. (Pogrel MA, Podlesh S, Anthony JP, Alexander J. A comparison of vascularized and nonvascularized bone grafts for reconstruction of mandibular continuity defects. *J Oral Maxillofac Surg* 1997; 55: 1200-6. Copyright © 1997 Elsevier.)

recommends vascularized free flaps when planning reconstruction for patients with continuity defects of the mandible greater than 6-9 cm. Non-vascularized bone grafts, generally harvested from the iliac crest, are reserved for delayed reconstruction of segmental defects less than 6 cm, in whom no radiation is planned and in whom there is adequate soft tissue. The exception is in the pediatric population in whom non-vascularized bone grafts appear to be much more predictable.⁸³ Corticocancellous bone may be placed into a resorbable crib along with a reconstruction bar for added stability and precision.

Surgical treatment of malignant tumors of the jaws

Malignant tumors typically require standard neck incisions in order to obtain proper access and facilitate reconstruction. The surgery is performed on a Mayfield-type head holder which allows both optimal head positioning as well as easy intraoperative repositioning. A tracheostomy may be needed depending on the location and extent of the planned surgical resection. A neck dissection is generally required and the incision can be designed as a straight line in a prominent neck crease or as a modification of the well known Shoberger or Conley incisions. The superior and inferior skin flaps are developed in a subplatysmal plane and sutured to the adjacent skin. Classically, the cervical lymphadenectomy precedes surgical resection of the primary tumor. A combined transoral/transcervical approach is generally used for all but the largest and most posterior tumors. Lip splitting incisions are reserved for inelastic, previously irradiated skin or very large primaries. A transbuccal trocar can be used in selected cases. The primary tumor is encircled by an incision that allows a margin of at least 1 cm of grossly normal oral mucosa and at least as much normal bone around the tumor edges. The author finds that prebending a reconstruction plate prior to surgery and application of the plate prior to mandibular osteotomies (if oncologically fea-

sible) facilitates accurate reconstruction. Classically, the resection should be performed *en bloc* with the neck dissection and the tumor delivered along with the lymph node-bearing tissue. The sites of mandibular osteotomy are exposed in the subperiosteal plane on both the medial and lateral aspects, and access is obtained transorally and through the neck. Mandibulotomy is accomplished with a reciprocating saw.

Benign tumors

Odontogenic epithelium with mature, fibrous stroma without odontogenic ectomesenchyme

Ameloblastomas

The ameloblastoma is a true neoplasm of odontogenic epithelial origin. The etiology of ameloblastoma is not known with certainty but the cells of origin are thought to be ameloblasts.^{84,85} Excluding odontomas, ameloblastomas are the most common odontogenic neoplasm and account for approximately 10% of all tumors that arise in the mandible and maxilla.⁸⁶⁻⁸⁹ Some authors have suggested significant epidemiological variation based on ethnic and/or geographical differences,^{1,2,90-95} however, the literature almost certainly contains bias due to differences in tumor sampling and whether the study was performed through a dental school pathology department or a medical hospital.⁹⁶ Larsson and Almeren⁹⁷ described an incidence of 0.3 cases per million people per year in Sweden. On the other hand, studies from Africa suggest that ameloblastomas may represent more than 60% of all odontogenic tumors in that part of the world.⁹⁸

Presenting symptoms may include a slow-growing submucosal mass, loose teeth, malocclusion, paresthesia, and pain. As many as 35% of patients are completely asymptomatic and the lesions are discovered as incidental findings on routine dental radiographs. The median age at presentation is 35 years (range, 4-92 years) and there is no gender predilection.⁸⁶⁻⁹⁵ Eighty percent of ameloblastomas arise in the mandible, usually in the ramus region, and are often associated with molar teeth.

Ameloblastomas have a distinctive histological appearance manifested by columnar, basally staining cells arranged in a palisaded pattern along the basement membrane (Fig. 32.12). Six histopathologic subtypes are recognized, including follicular, acanthomatous, plexiform, basal cell, desmoplastic, and granular cell variants. Most tumors show a predominance of one pattern, but mixtures of different patterns are commonly observed. Granular cell ameloblastoma was at one time thought to be a more aggressive variant; however, this is no longer the case.⁹⁹ Reichart *et al.*⁸⁶ reviewed a large series of ameloblastomas and suggested that histological sub-

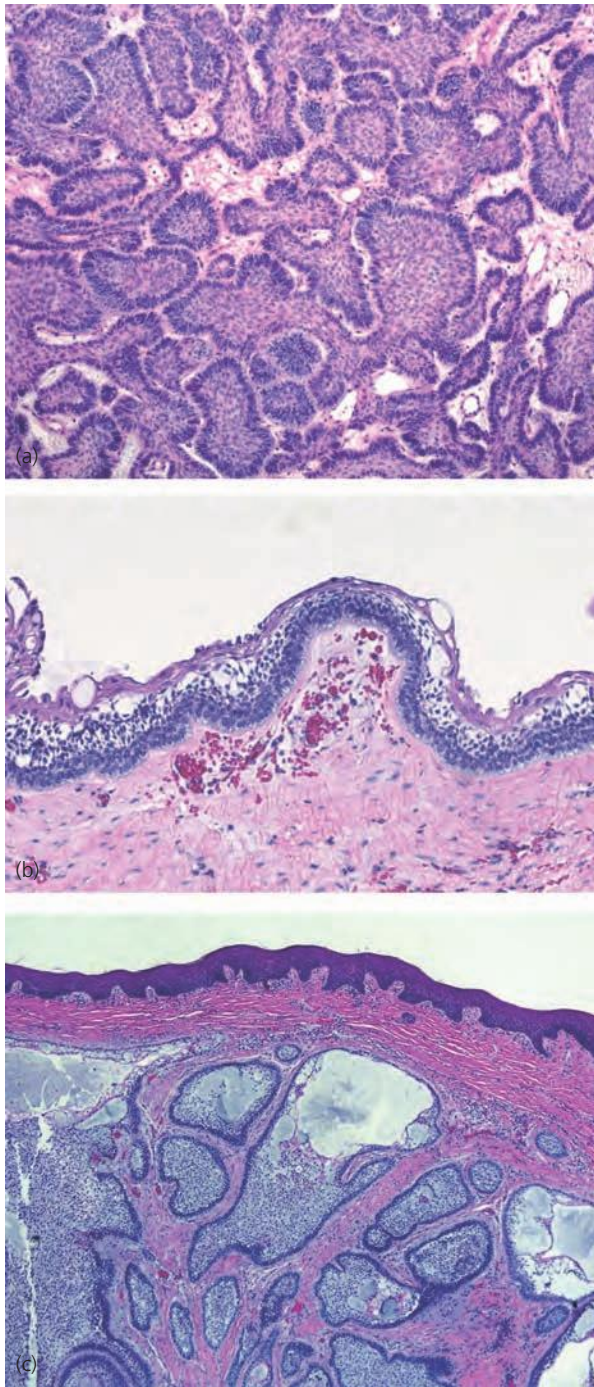


Fig. 32.12 Histologic appearance of ameloblastoma. (a) Solid/multicystic: note columnar, basally staining cells arranged in a palisaded pattern along the basement membrane. (b) Unicystic: note cystic architecture with typical ameloblastic changes confined to the cyst-lining epithelium. (c) Peripheral: note histological characteristics of intraosseous tumor. (Courtesy of Jeffery C. Stewart, DDS, MS.)

types may indeed have prognostic implications for recurrence. According to their study, the follicular type of ameloblastoma had the highest rate of recurrence at 29.5%, the plexiform subtype had 16.7% rate of recurrence, and the acanthomatous type had only a 4.5% recurrence rate. These differences were statistically significant. Too few cases of the other histological subtypes were included in the study for valid comparison, however, and there is still no consensus

as to the validity of these findings. Currently, most clinicians consider the histological subtypes to be of academic interest only and there is no convincing evidence suggesting a significant difference in biologic behavior. All ameloblastomas are benign but locally aggressive, will occasionally metastasize, and a few have malignant potential. Malignant ameloblastomas may be classified as metastasizing ameloblastoma or the more aggressive ameloblastic carcinoma.¹⁰⁰ It is thought that ameloblastic carcinoma may either arise *de novo* or as a transformation of benign ameloblastoma.¹⁰¹

As mentioned previously, the WHO has reclassified ameloblastomas into four types: (1) solid/multicystic ameloblastoma; (2) extraosseous/peripheral ameloblastoma; (3) demoplastic ameloblastoma; and (4) unicystic ameloblastoma. An entire body of literature has been devoted to explaining the various types of ameloblastomas and, unfortunately, there is still no uniform agreement on terminology. The WHO classification represents an important consensus of world leaders in the field of oral medicine, pathology, and surgery, and should be adopted in order to facilitate accurate communication and data evaluation.

Solid/multicystic ameloblastoma

Solid/multicystic ameloblastomas present as locally aggressive tumors that demonstrate inherent neoplastic cellular proliferation, are associated with high recurrence rates when inadequately treated, and represent the most clinically significant odontogenic tumor based on potential morbidity and prevalence. If left untreated, these tumors can grow to extreme sizes (Fig. 32.13). Enucleation and curettage has been associated with a recurrence rate of 60–80%.^{102–105} Neoplastic cells have been shown to be present several millimeters from the radiographic margin of the tumor,¹⁰⁶ which has led to the recommendation that resection be performed with care to maintain 1 cm tumor-free margins. This so-called radical resection generally results in local control rates greater than 90% (Table 32.6).^{102,103,105,107–110} Ghandhi *et al.*¹¹¹ recently compared the mode of presentation, treatment, and outcome of patients presenting at two different centers in different continents to investigate whether there were any significant differences in patient outcome based on geography. Fifty patients were included in the study and there were no significant differences in the clinical features on presentation (swelling, followed by pain, and altered sensation), the radiographic appearance (unilocular, 30%; multilocular, 70%), or treatment (enucleation and/or curettage, 50%; radical resection, 50%). Primary therapy by “conservative” enucleation and/or curettage led to a recurrence in approximately 80% of cases and the authors recommended “aggressive” treatment for both solid/multicystic tumors and unicystic ameloblastoma.

It is clear, based on the current body of evidence, that enucleation and curettage alone is inadequate for



Fig. 32.13 Massive ameloblastoma of the mandible. (Courtesy of Dr J.T. Arotiba.)

definitive treatment of solid ameloblastoma. Resection is the gold standard and currently offers the most predictable recurrence-free treatment for solid ameloblastoma (Fig. 32.14). For many tumors, however, resection with 1 cm tumor-free margins will commit the surgeon and patient to a segmental mandibulectomy or maxillectomy, often with sacrifice of the inferior alveolar nerve or creation of an oral–antral communication, and will necessitate primary or secondary reconstruction of continuity defects of the mandible or maxillectomy defects. Techniques have been described for segmental mandibular resection with inferior alveolar nerve preservation,¹¹² however this technique runs the theoretical risk of inviting perineural recurrence. Since ameloblastoma is a benign disease, many surgeons are reluctant to perform radical surgery on these patients. This understandable reserve has led to a number of other techniques including enucleation with peripheral ostectomy^{114,115} and/or adjuvant treatment with liquid nitrogen cryotherapy.^{53,116–118}

Cryosurgery has been used most commonly by orthopedic surgeons for the treatment of giant cell tumors of the long bones. Its use in the jaws for treatment of ameloblastoma has been limited. Curi *et al.*,¹¹⁹ treated 36 patients with ameloblastomas of the jaw with curettage and cryosurgery. They achieved a local recurrence rate of 30.6% in a follow-up of

Table 32.6 Outcomes of various treatment modalities for ameloblastomas.

Treatment method	Author	Year	Number of cases	Recurrence rate
Enucleation and curettage	Sehdev <i>et al.</i> ¹⁰²	1974	81	93%
	Shatkin and Hoffmeister ¹⁰³	1965	22	86%
	Mehlich <i>et al.</i> ¹⁰⁵	1972	126	36%
	Muller and Slootweg ¹⁰⁷	1985	56	58%
	Olaitan <i>et al.</i> ¹⁰⁸	1993	93	40%
	Ueno <i>et al.</i> ¹⁰⁹	1989	91	45%
	Ghandhi <i>et al.</i> ¹¹¹	2006	51	80%
	Hong <i>et al.</i> ¹¹³	2007	206	29%
Excision with bony margin (peripheral ostectomy) +/- cryotherapy	Sachs ¹¹⁵	2006	12	0%
	Sampson and Pogrel ¹¹⁸	1999	9	0%
	Curi <i>et al.</i> ¹¹⁹	1997	33	31%
	Hong <i>et al.</i> ¹¹³	2007	63	12%
Radical resection	Sehdev <i>et al.</i> ¹⁰²	1974	81	13%
	Shatkin and Hoffmeister ¹⁰³	1965	22	14%
	Mehlich <i>et al.</i> ¹⁰⁵	1972	126	21%
	Muller and Slootweg ¹⁰⁷	1985	56	15%
	Olaitan <i>et al.</i> ¹⁰⁸	1993	93	8%
	Ueno <i>et al.</i> ¹⁰⁹	1989	91	9%
	Ghandhi <i>et al.</i> ¹¹¹	2006	51	80%
	Hong <i>et al.</i> ¹¹³	2007	36	5%



Fig. 32.14 Transcervical segmental resection for solid ameloblastoma of the mandible and delayed reconstruction with autogenous corticocancellous bone harvested from the posterior iliac crest. (a) Multilocular, expansile radiolucency of the left mandible. (b) Presurgical marking for segmental resection. (c) Transcervical approach to the mandible with application of the reconstruction plate prior to segmental resection. (d) Specimen. (e) Radiograph of the specimen demonstrating negative resection margins. (f) Reconstruction plate *in situ*. (g) Postoperative panoramic radiograph. (h) Secondary (delayed) reconstruction of the same patient 6 months later using autogenous corticocancellous bone harvested from the posterior ilium. (i) Postreconstruction panoramic radiograph. (Courtesy of David L. Hirsch, DDS, MD.)

1–10 years. Sampson and Pogrel¹¹⁸ reported no recurrence in a smaller number of patients. Early experience with these less radical approaches is promising but deserves a note of caution. The surgeon should balance the clinical findings with the patient's social situation and ability to comply with close follow-up prior to embarking on less predictable forms of therapy. Clearly a non-compliant patient, one with massive tumor growth or pathologic fracture, or a patient with no ability to return for treatment (e.g. third world country), should be managed using the technique that offers the most predictable cure with the fewest number of operations. For many patients, this will generally involve segmental resection and immediate reconstruction using free tissue transfer, or staged bony reconstruction in the form of bone grafting.

It is important to distinguish technical differences when evaluating the literature regarding recurrence rates for the treatment of ameloblastoma. The term "conservative treatment" is often used to describe a heterogeneous group of therapeutic modalities excluding segmental resection, including enucleation and curettage, excision with peripheral ostectomy, excision with margin and adjuvant treatment such as cryotherapy. Hong *et al.*¹¹³ recently performed an evaluation of 305 cases of ameloblastoma in which they considered the various clinical and histopathological factors associated with recurrence, and distinguished between "conservative treatment" (enucleation and curettage), "resection with bone margin" (no continuity defect), and "radical resection" (segmental resection or maxillectomy). The dif-

ferences between the conservative and resection with bone margin and between the conservative and segmental resection or maxillectomy was highly significant in terms of disease-free survival. The difference between the resection with bone margin and segmental resection or maxillectomy groups was not significant, however. These data suggest clearly that enucleation and curettage are associated with an unacceptably high recurrence rate; however, many patients can be spared segmental mandibulectomy or maxillectomy without sacrificing tumor control.

The author's approach for all but the most expansile tumors is generally one of transoral excision with peripheral ostectomy, with or without cryotherapy, and immediate reconstruction. In the mandible, this can usually be accomplished via a transoral and/or a transbuccal approach using various rotary instruments and/or a reciprocating saw. Endoscopic techniques and intraoperative navigation may also provide an added measure of precision when establishing adequate resection/excision margins (Fig. 32.15). If necessary, a reconstruction plate can be applied to the inferior border of the mandible to stabilize the weakened segments. Patients with larger tumors or pathologic fracture undergo segmental resection and reconstruction with a microvascular free flap (Fig. 32.16).

Maxillary ameloblastomas are rarer and often more problematic than mandibular tumors (Fig. 32.17). Although histologically identical to their mandibular counterpart, surrounding anatomy is more complex. Involvement of the maxillary sinus and nasal cavity commonly occurs and infiltration

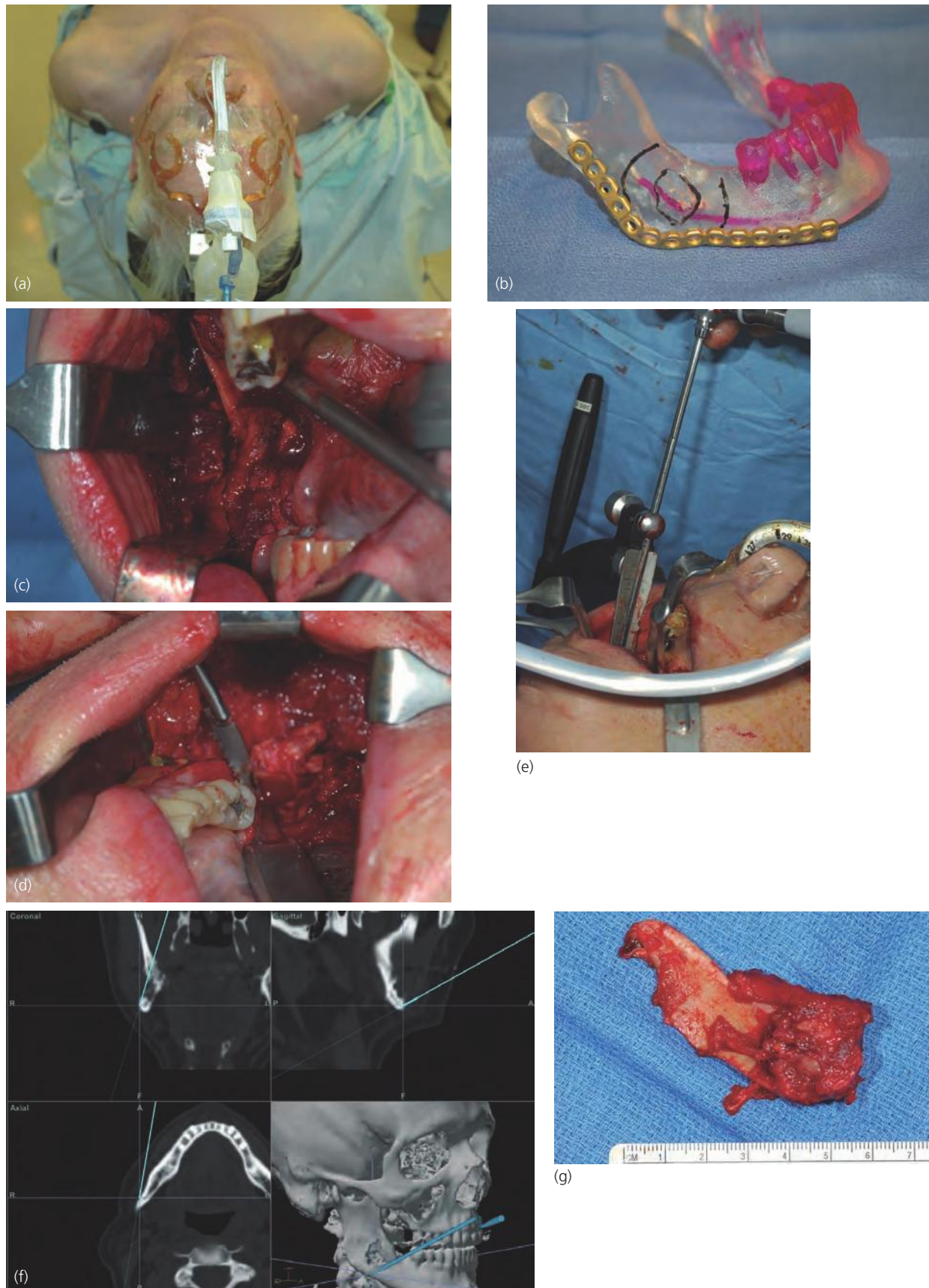


Fig. 32.15 Navigation and endoscope-assisted, transoral marginal resection for solid, recurrent ameloblastoma of the mandible and immediate reconstruction with autogenous corticocancellous bone harvested from the anterior iliac crest. (a) Preoperative set-up for intraoperative navigation. (b) Stereolithographic model for precise prebending of a load-bearing reconstruction plate, visualization of the tumor, and osteotomy design. (c) Soft tissue resection is initiated with 1 cm margins. (d) Prebent reconstruction plate is placed transorally under endoscopic visualization and navigation guidance. (e) Osteotomy is completed via a transbuccal approach under navigation guidance. (f) Intraoperative navigation confirming accurate osteotomy design with 1 cm tumor-free resection margins. (g) Resection specimen.

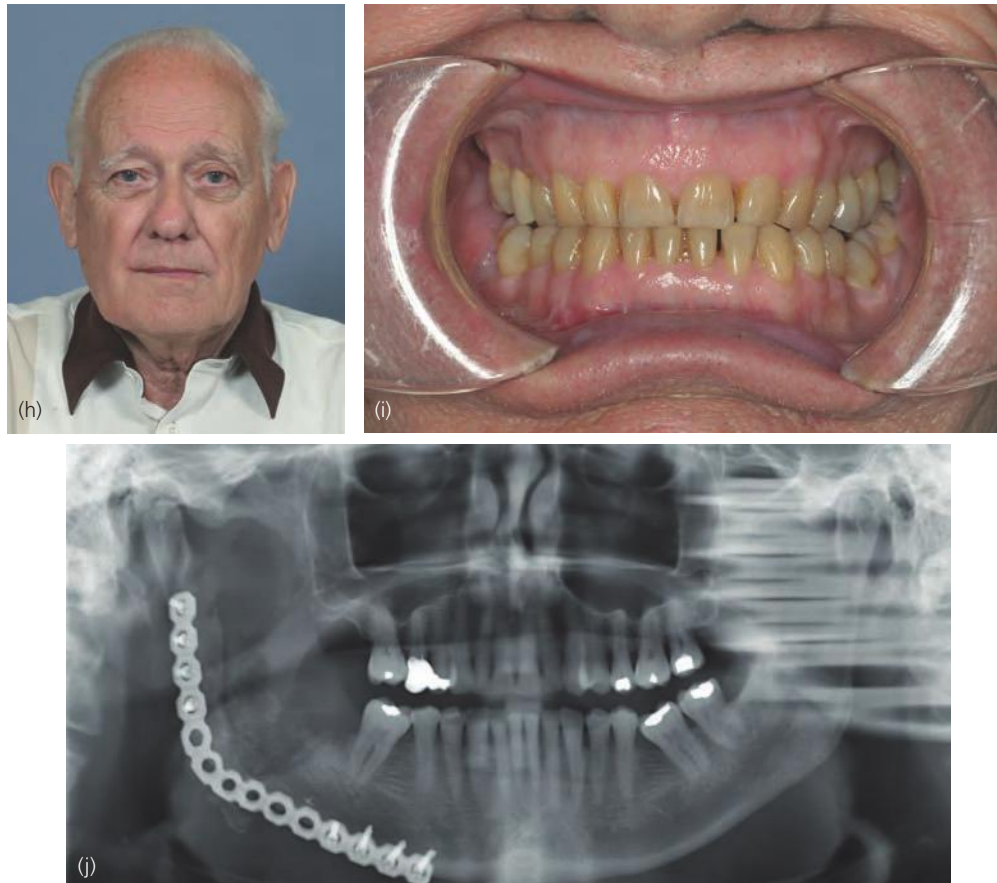


Fig. 32.15 (cont'd) (h) Postoperative facial appearance. (i) Postoperative occlusion. (j) Postoperative panoramic radiograph.

into the orbit, pterygomaxillary space, and skull base is distinctly possible in some patients with recurrent or uncontrolled disease. Even simple enucleation and peripheral ostectomy may result in oral–antral communication and thus require reconstruction. If it is possible to perform excision with peripheral ostectomy and treatment with liquid nitrogen therapy without creating an oral–antral/oral–nasal defect, then this is the preferred treatment for most small to medium-sized lesions. Because of the reconstructive issues, however, the author's general approach for larger tumors is to perform resection and immediate reconstruction using either local or regional rotational flaps, or alternatively microvascular free flaps.

Unicystic ameloblastoma

Unicystic growth patterns are seen in approximately 6% of ameloblastomas and were first described in 1977 by Robinson and Martinez.¹²⁰ They tend to occur in a younger population (mean age 22.1 years) and a high percentage of these lesions are associated with an impacted tooth.^{121–123} They will often present as a well circumscribed, unilocular periapical radiolucency (Fig. 32.18). Histologically, the unicystic ameloblastoma shows a cystic architecture with the typical ameloblastic changes confined to the cyst-lining epithelium (Fig. 32.12b). While most pathologists accept the concept of unicystic ameloblastoma as a separate entity rather than as a precursor to solid ameloblasto-

ma, much controversy exists over the terminology and how it should be classified.

One of the contentious issues regarding unicystic ameloblastoma is related to proliferation of the ameloblastic epithelium into the lumen of the cystic cavity or into the adjacent bone.¹²⁴ Extension of the ameloblastic epithelium into the lumen of the cystic cavity is termed intraluminal, or mural proliferation. There seems to be general agreement that as long as the ameloblastic characteristics are confined to the lining epithelial layers or if it remains intraluminal, conservative therapy such as enucleation and curettage is sufficient treatment. On the other hand, transmural involvement of the epithelium and bony invasion is defined by some pathologists as solid or “invasive” ameloblastoma, a term more descriptive of its aggressive biologic behavior.¹²⁵

It is clear that some unicystic ameloblastomas have a better prognosis and less chance for recurrence than do multicystic or solid variants when treated with enucleation and curettage (10.7–60%).^{121,124,126} It is also clear that unicystic ameloblastoma is considerably more aggressive than simple dentigerous cyst,¹²⁶ with which it is included in a differential diagnosis. Ueno *et al.*¹²⁷ reported on 91 patients treated surgically between 1968 and 1985 and found that of those treated with enucleation and curettage, with or without cryotherapy, 65% of the patients with unicystic lesions were locally controlled, compared to 39% of those

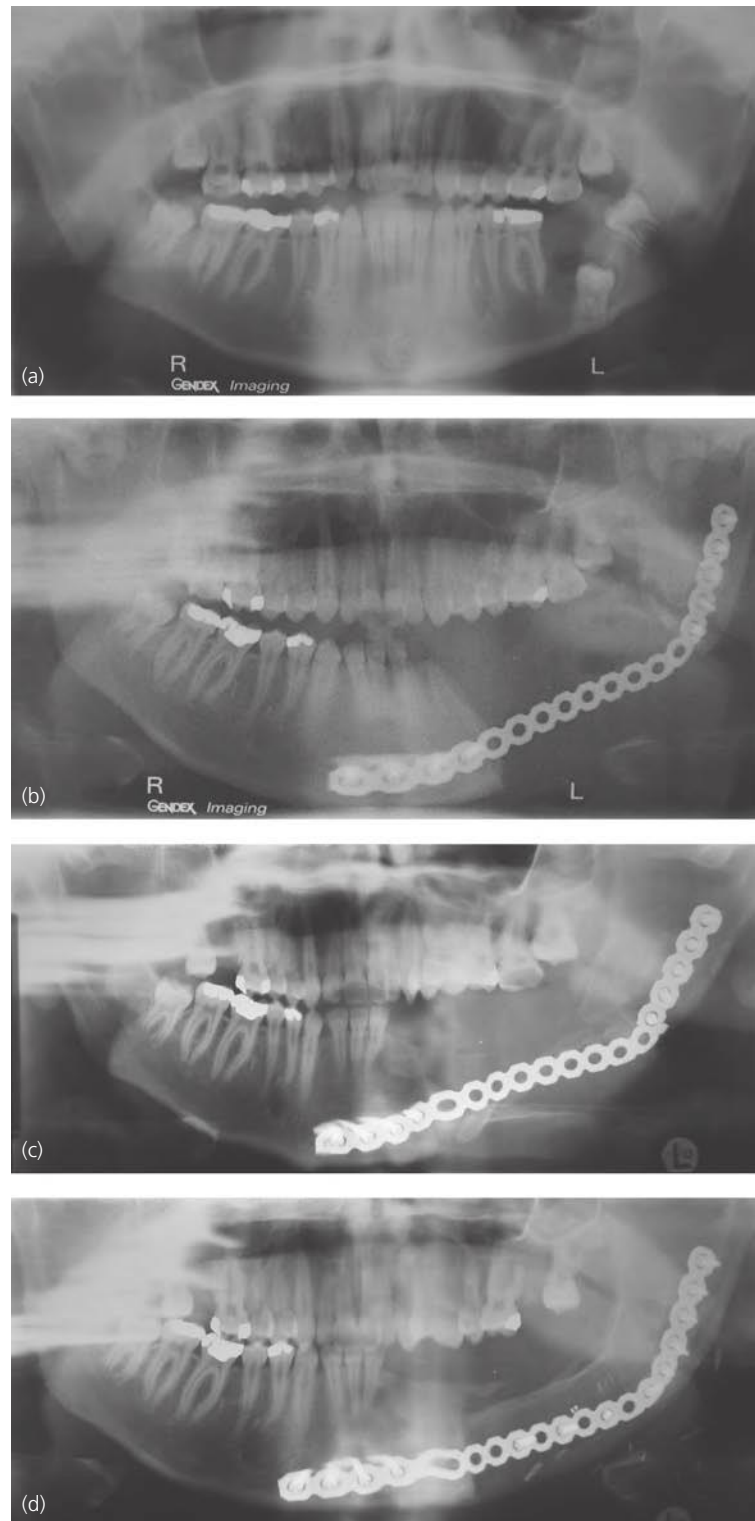


Fig. 32.16 A 21-year-old with failed reconstruction following segmental resection for ameloblastoma. Patient treated elsewhere and referred for microvascular reconstruction following fracture of the reconstruction plate. (a) Preoperative panoramic radiograph. (b) Segmental resection with placement of reconstruction plate. (c) Plate fracture. (d) Reconstruction with vascularized iliac crest flap based on the deep circumflex iliac artery.

with multicystic tumors ($P < 0.05$). Furthermore, Nakamura *et al.*,¹²⁸ reporting on 78 patients treated between 1960 and 1995, found that patients with unicystic lesions tended to have a high rate of local control regardless of extent of resection, whereas those with multicystic or solid tumors had better control with segmental resection. Lau and Samman¹²⁹ recently performed a systematic review that aimed to

identify all studies pertinent to the clinical question, “Which treatment for unicystic ameloblastoma results in the lowest recurrence rate?” A structured systematic search of the literature, with predefined inclusion and exclusion criteria, using computer and manual searches as well as personal communication, was performed. The recurrence rates were 3.6% for resection, 30.5% for enucleation alone, 16% for enucleation



Fig. 32.17 Computed tomography (CT) scan of large maxillary ameloblastoma (solid).

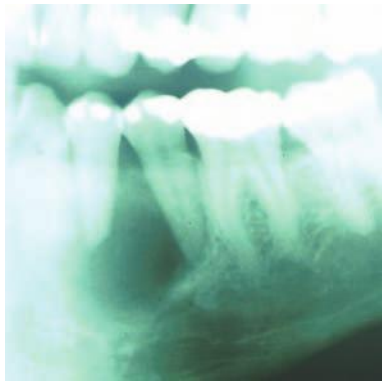


Fig. 32.18 Panoramic radiograph of unicystic ameloblastoma.

followed by application of Carnoy's solution, and 18% for marsupialization with/without other treatment in a second phase. It was concluded that there is only weak evidence showing that jaw resection resulted in the lowest recurrence rate, followed by enucleation with application of Carnoy's solution. Enucleation alone resulted in the highest recurrence rate and treatment by marsupialization cannot be sufficiently evaluated since most cases were followed by a second stage surgery of some kind.

The clinical problem rests in the fact that the diagnosis is often made retrospectively once the patient has undergone definitive treatment for a presumptive odontogenic cyst. The issue is complicated by the fact that to definitively establish a lesion as a unicystic ameloblastoma, complete microscopic evaluation of the bony margin surrounding the lesion must be performed, therefore necessitating *en bloc* resection of the osteolytic lesion. Clearly this is impractical and would represent gross overtreatment of the vast majority of lesions. Since incisional biopsy can never provide a definitive diagnosis, it is doubtful that the

issue will be settled in the near future. The surgeon should consider all of the clinical, radiographic, and patient-related factors, provide an appropriately tailored therapy warranted by the benign nature of these lesions, and probably accept a small rate of recurrence.

Extraosseous/peripheral ameloblastoma

The peripheral or extraosseous ameloblastoma was first described in 1949¹³⁰ and was more thoroughly documented by Stanley and Krogh¹³¹ in 1959. Peripheral ameloblastoma is defined as an odontogenic tumor with the histological characteristics of its intraosseous counterpart, but occurs exclusively in the gingiva without bony involvement (Fig. 32.12c). It is thought to represent between 2% and 10% of all diagnosed ameloblastomas.¹³² Similar in appearance to basal cell carcinoma, it has been suggested that some lesions previously reported as basal cell carcinoma of the gingiva are in fact peripheral ameloblastomas.¹³³ They present as a painless, firm, exophytic growth without gender predilection and in all age groups, with a mean age of 52.¹³⁴ Seventy percent of peripheral ameloblastomas occur in the mandible, more commonly anterior to the mental foramen. Simple excision with conservative margins is all that is required for treatment and these lesions rarely recur.¹³⁵

Desmoplastic ameloblastoma

The desmoplastic variant of ameloblastoma was first described by Eversole *et al.*¹³⁶ in 1984 and probably represents between 0.9% and 12.1% of all ameloblastomas.¹³⁷ The histological pattern is characterized by extensive stromal collagenization, or desmoplasia. Immunohistochemical studies suggest that the desmoplasia originates from *de novo* synthesis of extracellular matrix proteins. The mean age at initial presentation is 41.2 years (range 18–70 years) and there appears to be equal distribution between the maxilla and mandible.¹³⁸

Desmoplastic ameloblastomas often present as an asymptomatic, slowly enlarging bony or soft tissue mass in the maxilla or mandible. Unlike other ameloblastomas, the radiographic appearance is suggestive of fibro-osseous lesions, having been described as poorly defined radiolucent/radiopaque lesions, with or without loculations.

Treatment outcomes seem to be similar to those of other ameloblastoma variants: simple enucleation results in unacceptably high rates of local recurrence and radical resection is curative.¹³⁸ Intermediate forms of therapy using a combination of peripheral ostectomy with or without cryotherapy appear to offer a practical alternative.

The role of radiation therapy on unresectable or locally advanced ameloblastoma

There are few data regarding the efficacy of radiation therapy for benign, locally advanced or unresectable

ameloblastoma. A number of authors have attempted to use external beam radiotherapy in an attempt to achieve local control with variable success. Shatkin and Hoffmeister¹³⁹ reported two patients treated with radiotherapy for benign ameloblastoma, both of which had progressive disease and one of which died of tumor-related causes. Sehdev *et al.*¹⁴⁰ reported nine patients treated with radiotherapy for gross disease between 1921 and 1951, none of whom were locally controlled: three patients had persistent disease after treatment and six patients initially responded and then progressed. More recently, however, the use of radiotherapy has been re-examined. Several authors have reported excellent local control, either as definitive treatment or as postoperative adjuvant therapy for microscopic residual disease.^{141–143} A review of these reports showed that local control was achieved in 10 of 12 patients and has led to the recommendation that radiotherapy be considered for the occasional patient with positive margins who is not amenable to resection, those with unresectable tumors, or recurrent tumors involving the skull base or vital neurological structures.¹⁴⁴

Keratinizing odontogenic tumor

Formerly known as odontogenic keratocyst (OKC), the keratinizing odontogenic tumor (KOT) is a cystic lesion of odontogenic origin that demonstrates the behavioral characteristics of a benign neoplasm and has a propensity to recur following surgical treatment. A greater understanding of the clinical behavior and molecular biology of the KOT has prompted the terminology change from OKC and led to its recent classification as an odontogenic tumor.¹⁷ The microscopic characteristics of KOTs have been well defined.^{145,146} The salient histologic features include: (1) a thin, uniform layer of epithelium with little or no evidence of rete ridges; (2) a well defined basal cell layer with palisading cuboidal or columnar cells; and (3) a corrugated, keratinizing luminal surface that is primarily parakeratinized but may be orthokeratinized or a mixture of both (Fig. 32.19). The presence of “satellite” or “daughter” cysts has been recognized in a significant minority of KOTs and inflammation is an inconsistent finding. The radiographic appearance of KOTs is that of a unilocular or multilocular radiolucency, often with cortical expansion or erosion (Fig. 32.20).

Recurrence rates following initial treatment range as high as 62% and have prompted as yet unanswered questions about the pathophysiology of recurrence associated with this unique lesion.¹⁴⁷ Various theories have been popularized that correlate the potential for recurrence with the presence of “daughter” or “satellite” cysts remaining in the epithelium following enucleation, the presence of a thin cystic lining, incomplete removal of the cyst, or remnants of dental lamina not associated with the original cyst.^{148–151} Recent advances within the fields of

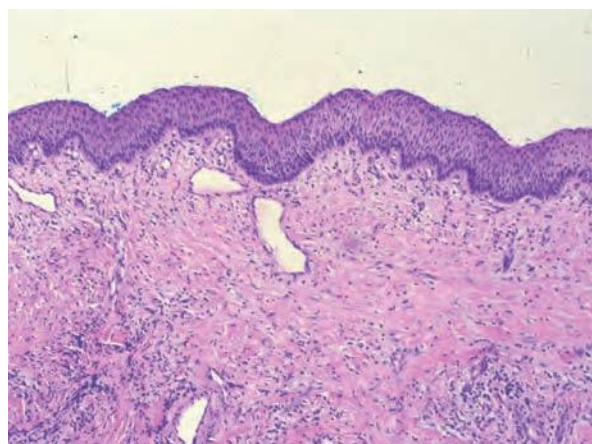


Fig. 32.19 Histological appearance of keratinizing odontogenic tumor (H&E). (Courtesy of Jeffery C. Stewart, DDS, MS.)



Fig. 32.20 Panoramic radiograph of keratinizing odontogenic tumor. Note multilocular appearance, cortical expansion, and erosion.

molecular genetics and immunocytochemistry may revolutionize our understanding of KOTs and, in time, render these time-honored explanations rudimentary in nature.^{152–154}

There is a growing body of evidence that a distinction should be made between parakeratinized KOTs, which have a relatively high risk of recurrence, and the orthokeratinized variant, which rarely recurs.^{145,146,155,156} Crowley and colleagues¹⁵⁷ reviewed 449 cases of KOT and separated them into parakeratinized, orthokeratinized, and mixed groups. The large majority of cysts were parakeratinized (86.2%) and there was no difference between the three groups with respect to age, race, sex, presenting symptoms, or clinical impression. Significantly, 42.6% of the parakeratinized group developed a recurrence, compared to only 2.2% of the orthokeratinized group. The subsequent identification of a histochemical marker (38-kD cell surface glycoprotein-gp38) found in basal cell carcinomas that was expressed within the basal and suprabasal epithelial cell membranes of parakeratinized KOTs, but not orthokeratinized KOTs, has led some authors to conclude that a distinction should be made between the two variants.^{158–160}

The aggressive nature of KOTs has been documented in numerous case reports and is manifested by cortical erosion,¹⁶¹ soft tissue involvement,¹⁶² and extension into the skull base,^{163–164} orbit,¹⁶⁵ and infratemporal fossa.¹⁶⁶ Studies published over the last three decades have reported recurrence rates following initial treatment with a variety of procedures that ranged from 2.5% to 62.5%.^{41–47,167–181} Myriad surgical strategies have been proposed to facilitate complete removal of the lesion and to minimize recurrent or residual disease, including more meticulous surgery with or without peripheral ostectomy, tissue fixation methods, soft tissue excision, radical treatment when necessary, and more conscientious follow-up. The optimum treatment modality has yet to be realized.

As previously stated, recurrence rates as high as 62.5% have been reported for KOTs that were treated with simple enucleation and curettage. Zhao *et al.*¹⁸² recently reported on the long-term outcome of 255 patients with KOTs treated by enucleation and curettage ($n = 163$), enucleation plus Carnoy's solution ($n = 29$), marsupialization plus enucleation ($n = 11$), and resection ($n = 52$). With follow-up of 3–29 years, the overall recurrence rate was 12.16%. Statistical comparison of the demographics of each group showed no difference with regard to site, age, or sex. However, there was a significant difference between groups with respect to method of treatment: KOTs treated by simple enucleation and curettage demonstrated a 17.79% recurrence rate compared to those treated with Carnoy's solution (6.70%, $P < 0.05$), marsupialization and resection (0.0%, $P < 0.01$). Some authors have proposed conservative management of recurrent KOTs based on close follow-up and repeated curettage, performed as recurrences develop.⁴² Given this documented propensity to recur after treatment, however, it seems that management of KOTs with simple enucleation should be avoided.

Stoelting⁴⁷ published the results of a prospective clinical trial conducted on 82 OKCs diagnosed in 80 patients over a 25-year period. Thirty-three patients underwent simple enucleation and curettage while 38 patients underwent excision of the overlying mucosa attached to the cyst where it had perforated through the bone, followed by treatment of the lesion with Carnoy's solution, enucleation of the cyst, and curettage of the bony cavity (Fig. 32.21). Five patients in the Carnoy's group presented with a recurrent tumor. With follow-up that ranged from 2 to 25 years there was an overall recurrence rate of 11% (nine patients). Most of the recurrences (six) occurred in the patients who were treated with enucleation and curettage alone. None of the patients presenting with recurrent lesions rerecurred following treatment using the authors' protocol. Of those with recurrence, six patients presented without signs or symptoms and the cyst was noticed on routine radiograph. There did not appear to be a correlation between age, sex, or site and recurrence.

Schmidt and Pogrel⁵⁴ recently published the results of 26 patients with KOTs who were treated with enucleation and cryotherapy. The majority of these cystic lesions ($n = 22$) were recurrent lesions. With follow-up of 2–10 years, recurrence was noted in three patients (11.5%). Complications were limited to wound dehiscence and local infection. The authors concluded that enucleation and liquid nitrogen cryotherapy may offer patients improved therapy in the management of KOTs.

There is little published data documenting the long-term outcome of patients with KOTs treated with marsupialization.^{183,184} Brondum and Jensen¹⁸⁵ found no recurrences among 12 patients with OKCs that were treated with decompression of the cyst cavity followed by cystectomy. Zhao *et al.*¹⁸² found no recurrences among 11 patients treated with marsupialization plus enucleation, with follow-up of at least 3 years. Recently, Pogrel and Jordan¹⁸⁶ and Pogrel¹⁸⁷ have published on the concept of carrying out marsupialization and decompression as definitive treatment for KOTs. These studies reported complete resolution and cure of these lesions based on clinical, histological, and radiographic evidence. Since that time, however, the investigators have published a partial retraction.¹⁸⁸ It has become apparent with long follow-up that recurrences may occur in as much as 12% of the cases.¹⁸⁷ Recurrences were detected at different time intervals, from 1.5 years to longer than 4 years. While no firm conclusions can be made from these small case series, this technique may be useful for the primary management of very large mandibular lesions in an effort to minimize surgical morbidity and/or the size of the reconstruction defect. Its role in the treatment of KOT remains ill defined but might be considered in two situations. Those lesions that have greatly expanded the shape of the mandible or have replaced the maxillary sinus would benefit from initial shrinkage and such shrinkage may very well allow a much more thorough secondary procedure (Fig. 32.22). In the case of mandibular lesions, initial marsupialization may help avoid pathologic fracture. The second area of utility of marsupialization for either primary or recurrent lesions is in the elderly or otherwise poor surgical candidate. Proper marsupialization may provide excellent palliative care in this patient group.

Resection is the only predictably curative procedure for patients with KOTs that offers a recurrence rate approaching zero.¹⁸² The concern with this method, of course, is the functional and esthetic morbidity associated with continuity defects of the mandible and maxilla. Advances in head and neck reconstruction, microvascular surgery, and dental implants during the last two decades, however, have made this aggressive treatment modality less foreboding (Fig. 32.23). The need for resection might be answered by this question: is the reconstructive utility of the residual osseous structure less than what could be provided entirely from distant tissue? If so, the resection and composite reconstruction would be appropriate.

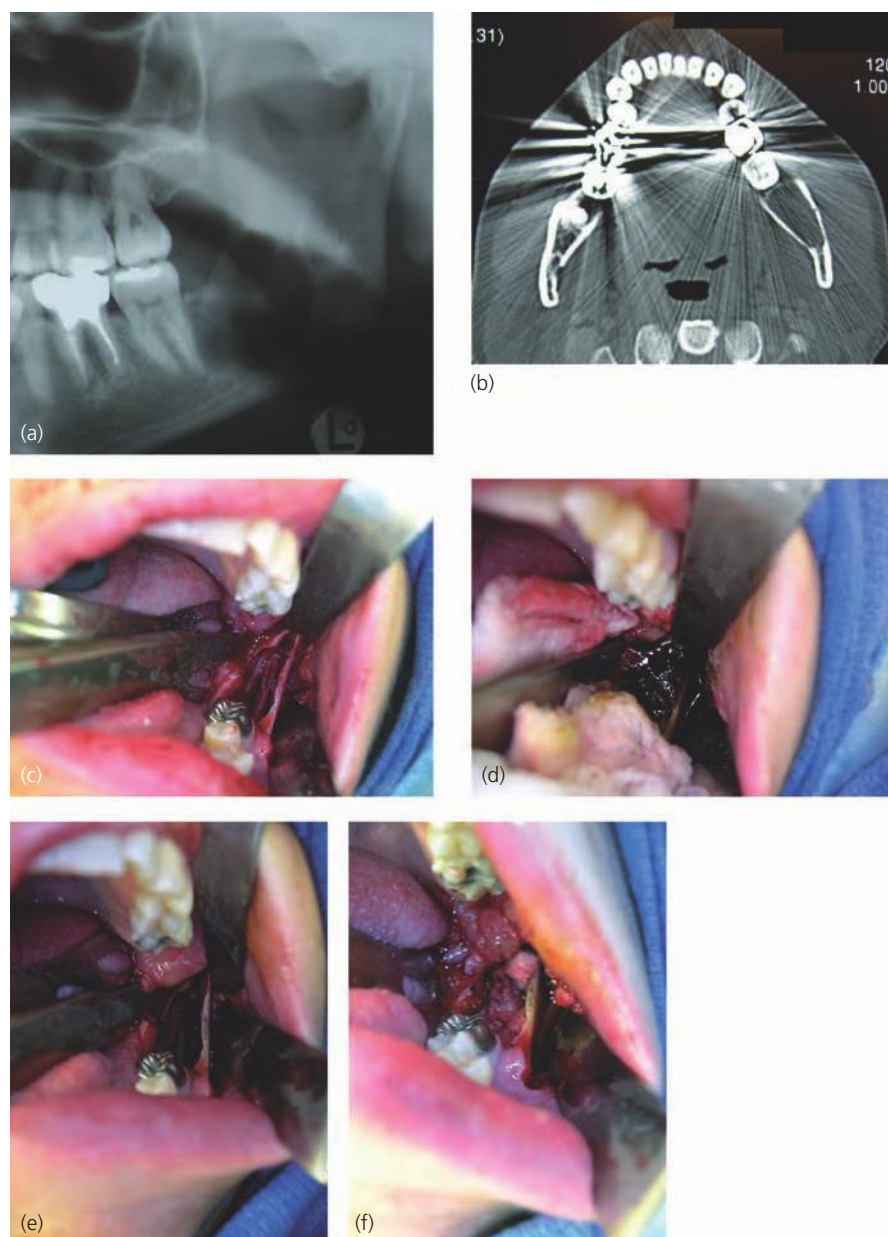


Fig. 32.21 A 34-year-old female with KOT involving the left mandible treated with enucleation and curettage with chemical fixation using Carnoy's solution. (a) Preoperative panoramic radiograph. (b) Preoperative CT scan. (c) Intraoperative photograph following enucleation and curettage (note inferior alveolar nerve). (d) Treatment with 5 minute application of Carnoy's solution. (e) Conservative peripheral ostectomy with rotary instrument. (f) Freeze-dried, demineralized bone graft reconstruction.

Based on the current level of evidence, treatment of most KOTs should involve enucleation and curettage with some sort of adjunctive procedure if resection is not a consideration (Table 32.7). The use of Carnoy's solution as a chemical fixative or cryotherapy as a thermal fixative appears to be a safe, effective way to predictably eradicate lesions with minimal morbidity. Alternatively, for larger tumors, decompression and marsupialization followed by cystectomy appears to offer a reasonably effective treatment.

Squamous odontogenic tumor

The squamous odontogenic tumor (SOT) was first described by Pullon *et al.*¹⁸⁹ in 1975. It is rare, with fewer than 50 cases having been reported.¹⁹⁰ Other synonymous terms were previously used to describe this tumor, including benign epithelial odontogenic tumor, acanthomatous ameloblastoma, acanthomatous ameloblastic fibroma, hyperplasia and squamous metaplasia of residual odontogenic epithelium, and benign odontogenic tumor unclassified.¹⁹¹ SOT is defined as a locally infiltrative neoplasm consisting of islands of well differentiated squamous epithelium

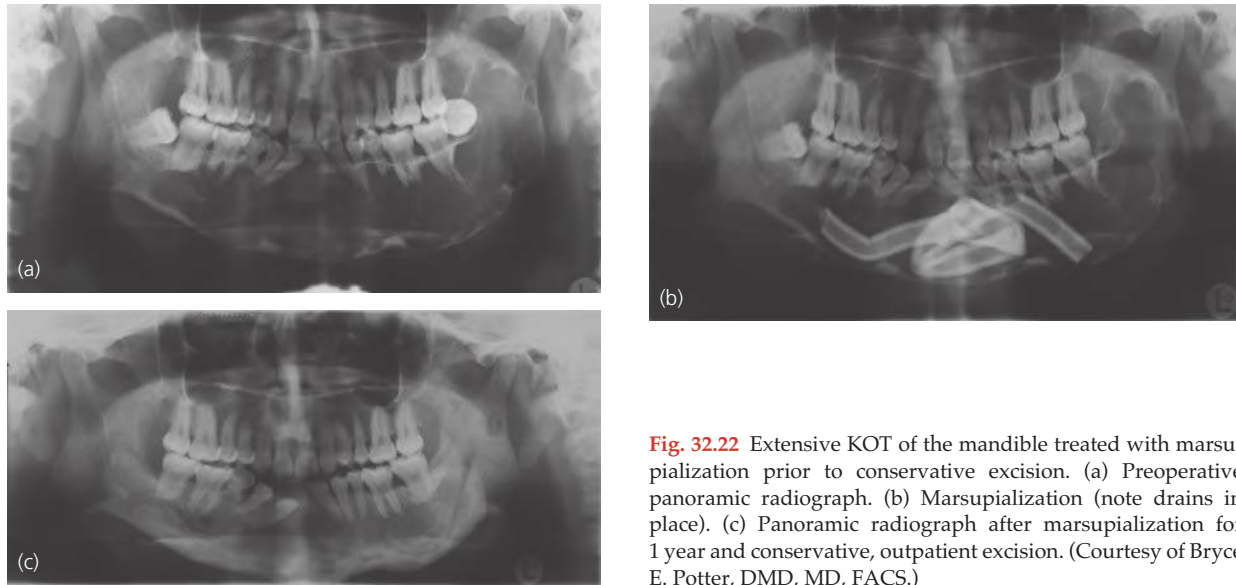


Fig. 32.22 Extensive KOT of the mandible treated with marsupialization prior to conservative excision. (a) Preoperative panoramic radiograph. (b) Marsupialization (note drains in place). (c) Panoramic radiograph after marsupialization for 1 year and conservative, outpatient excision. (Courtesy of Bryce E. Potter, DMD, MD, FACS.)



Fig. 32.23 Recurrent KOT of the left mandibular ramus with extension to the infratemporal fossa. The patient had been treated with three previous operations during a 20-year period, two of them aggressively through a transcutaneous approach. The full extent of the recurrent lesion involves not only the mandible but also the infratemporal fossa, parotid gland, and temporalis muscle, extending to the skull base. (a) Panoramic radiograph of the patient demonstrating multilocular radiolucency, however the extensive involvement cannot be appreciated on plain film, demonstrating the need for additional imaging. (b) Axial CT scan of the same patient demonstrating extensive involvement by KOT of the mandible, infratemporal fossa, parotid gland, and skull base. Note also the proximity of the lesion to skin. (c) CT of the same patient demonstrating cranial extension of the lesion with involvement of the temporalis muscle and skull base. (d) Panoramic radiograph demonstrates long-term result of the same patient 7 years after undergoing composite resection of the mandible and infratemporal fossa and superficial parotidectomy, with immediate reconstruction utilizing a fibular free flap. Note evidence of osseous healing, restoration of mandibular continuity, and no evidence of recurrent disease. (e) Clinical photograph of the patient 7 years after surgery. Note well healed parotidectomy scar and excellent restoration of form and function. (Courtesy of Eric J. Dierks, DMD, MD, FACS.)

Table 32.7 Treatment outcome for keratinizing odontogenic tumors.

Authors	Year	Number of patients	Treatment method	Rate of recurrence
Pindborg and Hansen ¹⁴⁷	1963	16	Enucleation or marsupialization	62.5%
Browne ¹⁴⁸	1971	85	Marsupialization or enucleation	24.7%
Brannon ¹⁴⁹	1976	312	Enucleation	12%
Vedtofte and Praetorius ¹⁷²	1979	72	Enucleation or marsupialization	51%
Voorsmit <i>et al.</i> ¹⁷³	1981	40	Enucleation, excision of mucosa; Carnoy's solution	2.5%
Brondum and Jensen ¹⁸⁵	1991	12	Decompression followed by cystectomy	0%
Schmidt and Pogrel ⁵⁴	2001	22	Enucleation and cryotherapy	11.5%
Zhao <i>et al.</i> ¹⁸²	2002	163	Enucleation and curettage	17.8%
Zhao <i>et al.</i> ¹⁸²	2002	29	Enucleation and Carnoy's solution	6.7%
Zhao <i>et al.</i> ¹⁸²	2002	11	Marsupialization and enucleation	0%
Zhao <i>et al.</i> ¹⁸²	2002	52	Resection	0%
Pogrel ¹⁸⁸	2007	42	Marsupialization	12%

in a fibrous stroma.^{192,193} The age range is broad (8–74 years, mean = 38.7) and there is no gender predilection. The etiology is unknown. The tumor typically presents as a localized gingival swelling or extraosseous mass in the maxilla or mandible with possible tooth mobility, pain, and gingival erythema.

Histologically, SOTs are composed of rounded or oval islands of well differentiated squamous epithelium within a fibrous stroma (Fig. 32.24). Individual islands may contain calcified material and may undergo central microcystic degeneration. Rests of Malassez may be the source of the epithelial proliferation for lesions that are associated with the alveolar process adjacent to the root surface,^{189,190} whereas the dental lamina has been implicated in the development of tumors associated with the crowns of unerupted or impacted teeth.^{194,195}

SOTs do not generally behave aggressively, however they can present as multiple lesions,¹⁹⁶ erode into cortical bone,¹⁹¹ and cause cortical expansion.¹⁹⁴ One case of malignant variant has been reported.¹⁹⁷ Treatment is by conservative surgical excision and recurrences are rare.

Calcifying epithelial odontogenic tumor

The calcifying epithelial odontogenic tumor (CEOT) is defined as a locally invasive epithelial odontogenic neoplasm, characterized by the presence of amyloid material that may become calcified.¹⁹⁸ First described by Pindborg in 1955,¹⁹⁹ the tumor is often referred to as a Pindborg tumor. Fewer than 200 cases have been reported in the literature and it is thought to represent approximately 1% of all odontogenic tumors.²⁰⁰ The tumor has been reported to occur in patients between 13 and 80 years of age and has no gender predilection.²⁰¹ The majority of cases occur in the mandible (3:1 ratio) and the premolar area is a common site.

The CEOT usually presents as an asymptomatic, slow-growing, expansile mass in the mandible or maxilla. More than 90% of the cases are intraosseous, but extraosseous variants have been reported. Radiographically, they have been described as a well defined, unilocular or multilocular, mixed radiolucent/radiopaque lesion, however these findings are variable.²⁰²

Histologically, the tumor is characterized by islands and sheets of polyhedral epithelial cells with abundant eosinophilic cytoplasm, sharply defined cell borders and well developed intercellular bridges, within a fibrous stroma (Fig. 32.25).¹⁹⁸ The nuclei are often pleomorphic and mitotic figures are rarely encountered. Homogeneous, eosinophilic hyalin material is present and confirmed by positive staining with Congo red and fluorescence with thioflavine T. Liesegang rings are calcifications in concentric

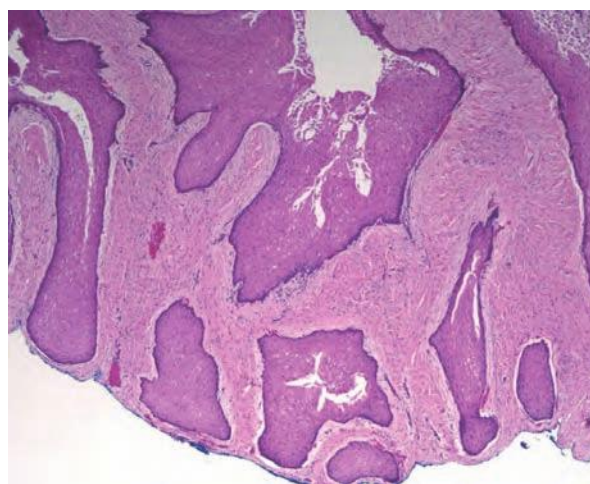


Fig. 32.24 Histological appearance of squamous odontogenic tumor. Note rounded or oval islands of well differentiated squamous epithelium within a fibrous stroma. (Courtesy of Jeffrey C. Stewart, DDS, MS.)

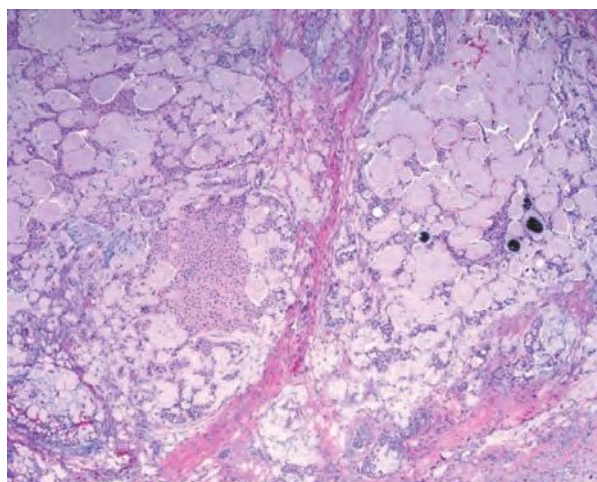


Fig. 32.25 Histological appearance of calcifying epithelial odontogenic tumor (CEOT). Note islands and sheets of polyhedral epithelial cells with abundant eosinophilic cytoplasm within a fibrous stroma. (Courtesy of Jeffery C. Stewart, DDS, MS.)

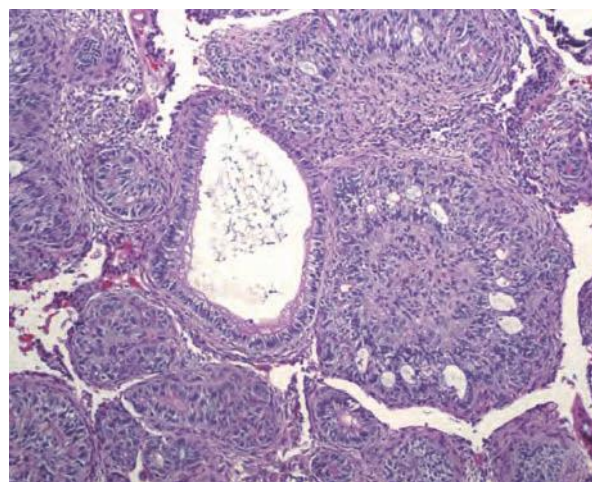


Fig. 32.26 Histological appearance of adenomatoid odontogenic tumor (AOT). Note proliferation of spindle, cuboidal, and columnar cells, scattered duct-like structures, eosinophilic material, and calcifications within a fibrous capsule (capsule not visualized). (Courtesy of Jeffery C. Stewart, DDS, MS.)

shapes that can occur within the amyloid and account for the radiopacities. Clear cells make up a significant proportion of the tumor, which is thought to arise from the stratum intermedium tissue of the developing enamel organ. Generally there is no nuclear pleomorphism, however there are reports of CEOT demonstrating malignant features.^{203,204}

CEOT is a locally invasive tumor that is associated with recurrence rates of 14–20% following enucleation and curettage.^{201,205,206} Slightly higher rates of recurrence have been associated with the clear cell variant. The occurrence of malignant transformation to odontogenic carcinoma ex Pindborg tumor is extremely rare, but has been reported.²⁰⁷ Although thought to be less aggressive than the solid ameloblastoma, excision with 0.5–1.0 cm margins is generally recommended. The same controversies that surround radical resection of ameloblastomas are applicable to CEOT and the clinician is cautioned to weigh all pertinent clinical, pathological, and social variables prior to recommending definitive surgical therapy.

Adenomatoid odontogenic tumor

The adenomatoid odontogenic tumor (AOT) is a unique lesion involving the jaws (centrally located), or in the soft tissue (gingival) overlying tooth-bearing areas or alveolar mucosa in edentulous regions. It is defined by the WHO as a tumor of odontogenic epithelium in a “variety of histoarchitectural patterns, embedded in a mature connective tissue stroma and characterized by slow but progressive growth”.²⁰⁸ Typically the tumor is made up of a cellular multinodular proliferation of spindle, cuboidal, and columnar cells in a variety of patterns; usually scattered duct-like structures, eosinophilic material, and calcifications in several forms; and a fibrous capsule of variable thickness (Fig. 32.26). It is regarded by

some authors as a benign neoplasm and by others as a developmental hamartoma. The first reported case was probably that of Harbitz in 1915,²⁰⁹ termed an “adamantoma”. Prior to and since that time, the literature has provided a confusing array of terms and descriptions.²¹⁰ In 1909 James and Forbes²¹¹ described the tumor as an “epithelial odontome”. Several years later, Wohl²¹² referred to it as a “tooth germ cyst of the jaw”. Stafne²¹³ reported the first series of AOT in 1948 under the title “epithelial tumors associated with developmental cysts of the maxilla”. Additional terms that have been used in the past to describe this tumor but have now been discarded include: adenoamantoblastoma,²¹⁴ adeno-ameloblastoma,²¹⁵ cystic complex composite odontome,²¹⁶ unusual pleomorphic adenoma-like tumor in the wall of a dentigerous cyst,²¹⁷ tumor of enamel organ epithelium,²¹⁸ adenomatoid ameloblastoma,²¹⁹ adenomatoid odontoma,²²⁰ ameloblastic adenomatoid tumor,²²¹ and odontogenic adenomatoid tumor.²²² Finally, Philipsen and Birn²²³ proposed the name adenomatoid odontogenic tumor in 1969, a term that was adopted by the WHO in 1971.¹¹

AOTs account for approximately 2–7% of all odontogenic tumors.^{223,224} They occur in almost any age group (3–82 years), however more than 90% are diagnosed prior to age 30 and half of the cases occur in adolescence. The male:female ratio is approximately 1:1.9 and AOT may be more prevalent in Asians.²²⁵ The most common site is in the anterior maxilla, with a 2:1 (maxilla:mandible) ratio.

AOTs are often detected on routine radiographic evaluation as an asymptomatic intraosseous lesion associated with an unerupted permanent tooth, commonly a maxillary canine. When intraosseous growth causes cortical expansion, it may present as a slow-growing, painless, bony mass, but peripheral variants are also described. The characteristic radiographic appearance is that of a well defined, unicystic radio-

lucency associated with the crown of an impacted tooth. The radiolucency shows focal areas of radiopacity in two thirds of the cases of intraosseous tumor.

Three histologically identical variants have been described: intraosseous follicular (70%); extrafollicular (26%); and peripheral (4%).²²⁶ Variably sized solid nodules of cuboidal or columnar cells of odontogenic epithelium that form nests or rosette-like structures with minimal stromal connective tissue are noted microscopically. Eosinophilic amorphous material ("tumor droplets") is present between the epithelial cells and in the center of the rosette-like configurations.

The biological behavior of this tumor is more hamartomatous than neoplastic, although aggressive variants are occasionally encountered. Treatment is with conservative local excision and recurrences are unusual.

Odontogenic epithelium with odontogenic ectomesenchyme with or without hard tissue formation

Ameloblastic fibroma/fibrodentinitoma

Along with odontoameloblastoma and dentinogenic ghost cell tumor, the ameloblastic fibroma (AF) is one of the benign "mixed" odontogenic tumors that is a true neoplasm characterized by expansile growth and potentially destructive behavior. It lies in contradistinction to the other "mixed" odontogenic tumors, ameloblastic fibro-odontoma and odontoma, which represent hamartomas that rarely result in significant morbidity. The AF consists of "odontogenic ectomesenchyme resembling the dental papilla and epithelial strands and nests resembling dental lamina and enamel organ", which distinguishes them from tumors of purely ectomesenchymal or pure epithelial origin.²²⁷ Generally, no dental hard tissues are present; however, if dentin is observed, the lesion is referred to as an ameloblastic fibrodentinitoma.²²⁸ It has been suggested that some cases in childhood may represent the primitive stage of a developing odontoma, however there are distinct differences between these entities.²³² AF tends to occur in the first two decades of life, but patients older than 22 years are not uncommon. The posterior mandible is affected in more than 70% of the cases.^{1,229} The tumor presents as a unilocular or multilocular radiolucency that may demonstrate cortical expansion, erosion or both.²³⁰ Chen *et al.*²³² recently published an extensive review of the English language literature since the first description of AF in 1891. A total number of 123 cases with well documented follow-up data were identified and an overall recurrence rate of 33.3% was reported in cases that were treated by conservative (91.5%) and/or radical (8.5%) means. Despite these findings, most authors recommend conservative excision, particularly in young patients, and find that the lesions

rarely recur.^{227,233} The author's approach is that of excision with peripheral ostectomy and adjuvant cryotherapy (Fig. 32.27). The patients are followed closely and if a recurrence occurs it is typically small and readily managed on an outpatient basis. Malignant transformation has been widely reported, however, so these patients must be followed for a significant period of time following definitive surgical extirpation.²³⁴

Ameloblastic fibro-odontoma

One of the "mixed" odontogenic tumors, the ameloblastic fibro-odontoma most likely represents a hamartoma, rather than a true neoplasm. It has been suggested that the ameloblastic fibro-odontoma is simply an immature complex odontoma. Histologically, it shares the same features of ameloblastic fibroma, except that it contains dentin and enamel.²³⁵ The tumor typically presents as an asymptomatic, well circumscribed, mixed radiopaque/radiolucent mass involving the posterior mandible.²³⁵ Rarely exhibiting aggressive behavior, the tumor is treated effectively with enucleation and curettage.²²⁹⁻²³⁵ Recurrence after conservative treatment is rare. Malignant transformation has been reported.

Odontoma, complex and compound type

Odontomas are the most common of the odontogenic tumors, represent a benign hamartoma rather than a true neoplasm, and generally have a benign clinical course. Odontomas are described in two types: complex and compound. The difference between the two forms is that compound odontomas contain recognizable enamel, dentin, and sometimes cementum, shaped in toothlike structures; whereas complex odontomas are composed of irregular masses of dentin and enamel and have no anatomic resemblance to a tooth.²³⁶ Complex odontomas are typically found in the posterior maxilla or mandible and compound odontomas are predominately in the anterior maxilla. The compound odontoma is twice as common as the complex odontoma.²³⁷ The lesions appear as well circumscribed, radiopaque masses, often surrounded by what appears to be a periodontal ligament (Fig. 32.28). The masses are treated with simple enucleation and curettage, which is virtually always curative.

Odontoameloblastoma

According to the WHO, odontoameloblastoma (OA) "combines the clinical and histological features of ameloblastoma with those of an odontoma".²³⁸ This is a rare neoplasm (fewer than 50 cases have been reported) that occurs in the maxilla and mandible with equal prevalence, usually in the first 3 decades of life.²³⁹ Clinical signs and symptoms, as well as biological behavior, may be identical to those of a solid

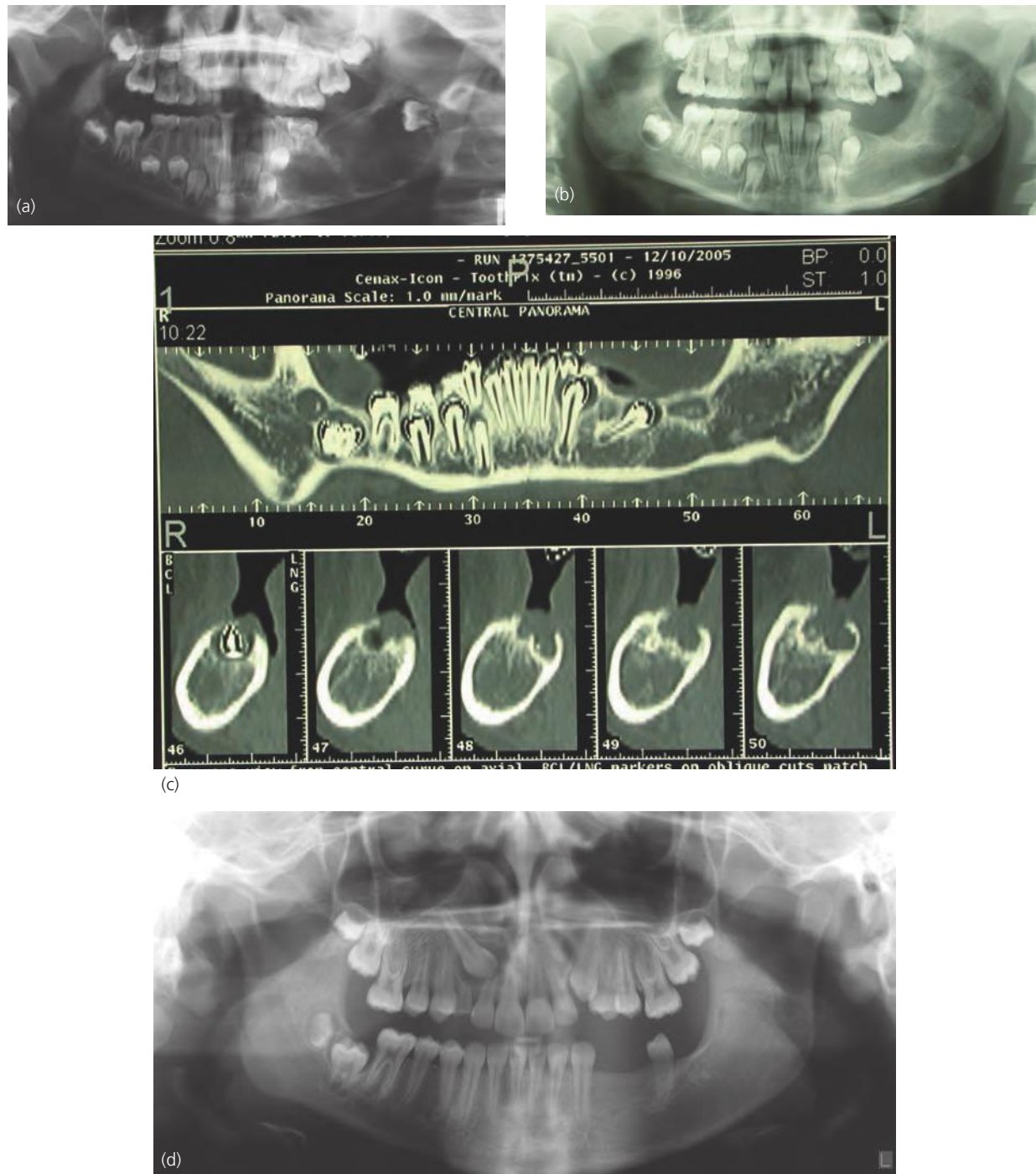


Fig. 32.27 Ameloblastic fibroma of the mandible treated by excision with peripheral ostectomy and cryotherapy. (a) Preoperative panoramic radiograph. (b) 1 year postoperative panoramic radiograph. (c) Small recurrence noted on cone-beam CT scan 2 years following treatment. (d) Postoperative panoramic radiograph 2 years following conservative, outpatient excision of recurrence. (Courtesy of Eric J. Dierks, DMD, MD, FACS.)

ameloblastoma.²⁴⁰ The radiographic appearance of this tumor differs from that of ameloblastoma by varying amounts of radiopaque material, which generally represents displaced or unerupted teeth. The histological appearance consists of islands and cords of odontogenic epithelium demonstrating follicular and plexiform patterns, in addition to a variable amount of cellular myxoid tissue that contains mineralized dental tissues in the form of odontomas.²³⁸ Similar to conventional ameloblastoma in its biological behavior, OA is locally aggressive and requires

surgical excision with 0.5–1.0 cm tumor-free margins.^{239–243} The same treatment-related controversies that surround optimal treatment of ameloblastoma are presumably shared by this rare neoplasm.

Calcifying cystic odontogenic tumor

Calcifying cystic odontogenic tumor (CCOT) is a benign odontogenic neoplasm that resembles ameloblastoma with ghost cells that may calcify.²⁴⁴ First described by Gorlin *et al.*²⁴⁵ in 1962, it is also termed



Fig. 32.28 Complex odontoma of the mandible.

Gorlin cyst and calcifying odontogenic cyst. The CCOT may present as an expansile mass involving the maxilla or mandible with equal frequency, and both intraosseous and extraosseous forms occur.²⁴⁶ It can occur at any age and there is no gender predilection.^{247,248} It typically presents as an asymptomatic, unilocular, well defined radiolucency, however it can exhibit a radiopaque component as well. Histologically, the CCOT is characterized by a cyst wall that is lined by thin ameloblastomatous epithelium with the formation of ghost cells (Fig. 32.YY).²⁴⁴ Ledesma-Montes *et al.*²⁴⁹ recently attempted to clarify and group the clinicopathological features of a number of CCOT cases from around the world and proposed a clinically useful classification under the 2005 WHO guidelines. CCOT was divided into four subtypes: (1) simple cystic; (2) odontoma associated; (3) ameloblastomatous proliferating; and (4) CCOT associated with benign odontogenic tumors other than odontomas. Aggressive clinical behavior is not typical of this benign entity, and simple enucleation and curettage is generally curative.^{247,248,250}

Dentinogenic ghost cell tumor

Formerly considered a solid variant of the calcifying odontogenic cyst (Gorlin cyst), the dentinogenic ghost cell tumor is a locally invasive neoplasm characterized by ameloblastoma-like islands of epithelial cells in a mature connective tissue stroma, with varying amounts of dysplastic dentin and ghost cells.²⁵¹ The tumor typically presents as an asymptomatic hard tissue or soft tissue mass (intraosseous or extraosseous variants), showing varying amounts of bone resorption or tooth displacement. Radiographically, it may present as a radiolucent or mixed radiolucent/radiopaque lesion with unilocular, well circumscribed borders. It is histologically distinguished from ameloblastoma and calcifying odontogenic tumor by the presence of ghost cells with dysplastic dentin. Although malignant transformation has been described, recurrences after appropriate excision with 0.5–1.0 cm margins are rare.²⁵² Similar challenges to

ameloblastoma are present with regards to the most appropriate surgical treatment and the extent of resection.

Mesenchyme and/or odontogenic ectomesenchyme with or without odontogenic epithelium

Odontogenic fibroma

The odontogenic fibroma (OF) is a very rare neoplasm that is defined by the WHO as containing “varying amounts of inactive-looking odontogenic epithelium embedded in a mature fibrous stroma”.²⁵³ Two histological subtypes have been described: epithelial poor (formerly termed simple type) and epithelial rich (formerly termed complex or WHO type). The OF occurs as a radiolucent lesion involving the jaws without respect to gender, age, or site (maxilla or mandible).²⁵⁴ The radiographic appearance is that of a radiolucent lesion that may be unilocular or multilocular and cause cortical expansion.²⁵⁵ Although aggressive variants have been described, neither subtype appears to behave in an aggressive fashion and both respond well to enucleation and curettage.^{256,257} Brannon recently reviewed 73 well documented cases of odontogenic fibroma (epithelial rich) as reported in the English literature. Most patients underwent some variant of enucleation and curettage. Follow-up information greater than 1 year was available in 27 cases, for which a recurrence rate of 26% was reported.²⁵⁴

Odontogenic myxoma/myxofibroma

The odontogenic myxoma (OM) is relatively common amongst odontogenic tumors and may comprise between 3 and 20% of all such lesions.²⁵⁷ It is the second or third most common odontogenic tumor with a reported incidence of 0.07 new cases/million people/year.²⁵⁴ The WHO characterizes this intraosseous neoplasm as containing “stellate and spindle-shaped cells embedded in an abundant myxoid or mucoid

extracellular matrix" (Fig. 32.29).²⁵⁸ The term myxofibroma is used when a relatively greater amount of collagen is evident.

Two thirds of OMs are located in the mandible, commonly in the molar regions, and typically present in the second to fourth decade of life (age range 1–70 years) without gender predilection.²⁵⁹ It is commonly found as an incidental finding on routine panoramic radiograph. The radiographic appearance is that of a well defined, unilocular or multilocular radiolucency, generally associated with an erupted tooth. The tumor typically appears as a hyperintense signal in contrast-enhanced magnetic resonance imaging (MRI); these lesions are identified due to their high proton density associated with water bound to the myxoid stroma.

The lesion has a rather bland histological appearance that is characterized by a loose mesenchymal fibrous tissue that lacks atypia.²⁵⁸ Randomly oriented stellate, spindle-shaped and round cells with eosinophilic cytoplasmic processes sit in a mucoid or myxoid stroma. OM has a similar histological appearance to that of hyperplastic dental follicles and the dental papilla of a developing tooth. Clinical correlation is essential to avoid misdiagnosis.

The OM is a benign but locally aggressive tumor that is probably less aggressive than ameloblastoma, but can result in progressive growth with skull base involvement if left untreated. Malignant variants have been described.²⁶⁰ Barker's review of the literature suggested an overall recurrence rate of between 10% and 33%.²⁶¹ Several authors have advocated conservative therapy and have claimed success;^{262–264} however, others have found poor results with enucleation alone.^{265–267} Resection with 1–1.5 cm bony margins and one uninvolved anatomic barrier margin has been advocated as the best chance for a curative treatment due to its potentially aggressive behavior.^{268,269} The author's approach to smaller lesions is typically enucleation with peripheral ostectomy. Larger lesions that exhibit aggressive biological behavior, such as cortical erosion or recurrence following treatment, should be excised with 0.5–1.0 cm tumor-free margins, and/or consideration should be

given to adjunctive treatment in the form of Carnoy's solution or cryotherapy.

Cementoblastoma

Cementoblastoma is a rare lesion that comprises less than 1% of all odontogenic tumors.²⁷⁰ It is characterized by the formation of a radiopaque cementum-like mass intimately associated with the root of a tooth (usually a lower second or third molar). The tooth typically remains vital and symptoms include varying degrees of cortical expansion and pain. Classically, the tumorous mass is surrounded by a radiolucent ring that represents the periodontal ligament (Fig. 32.30). Histologically identical to an osteoblastoma, the cementoblastoma is differentiated from its bony counterpart by virtue of its intimate association with teeth.²⁷¹ Biopsy will distinguish it from an odontoma, focal sclerosing osteomyelitis, and hypercementosis, all of which may have a similar clinical and radiographic appearance. Gigantiform variants have been encountered, requiring radical resection and reconstruction with free tissue transfer.²⁷² Treatment is otherwise by excision with peripheral ostectomy for all but the largest tumors. Recurrence is possible if incompletely excised.



Fig. 32.29 Odontogenic myxoma. Coronal CT scan demonstrating well defined expansile mass with displacement of teeth.



(a)



(b)

Fig. 32.30 Cementoblastoma of the maxilla. (a) Preoperative CT scan. (b) Resection specimen.

Non-odontogenic benign tumors

Ossifying fibroma

Ossifying fibroma (OF) is one of the so-called fibro-osseous lesions that include ossifying fibroma, fibrous dysplasia, and cemento-osseous dysplasia. Formerly known as cemento-ossifying fibroma, and thought to be derived from the periodontal ligament, this benign neoplasm is characterized by the replacement of normal bone by fibrous tissue and varying amounts of newly formed bone or cementum-like material. The WHO recognizes two distinct histological variants, neither of which differ from each other significantly in their biological behavior or treatment: juvenile trabecular ossifying fibroma (JTOF) and juvenile psammomatoid ossifying fibroma (JPOF).²⁷³ The juvenile variants occur in a younger age group than the conventional type of ossifying fibroma. The mean age for JPOF is 20 years, compared to that of 35 years in cases of adult ossifying fibroma, whereas the mean age range for JTOF is 8.5–12 years.²⁷⁴

OF is primarily found in the posterior mandible, JPOF occurs in the bony walls of the paranasal sinuses, and JTOF commonly presents in the maxilla.²⁷⁵ All variants of OF generally present as painless, slow-growing, expansile osseous lesions. The radiographic appearance is typically a well defined expansile mass with a variable degree of internal calcification (Fig. 32.31). Root displacement and root resorption may be seen.

Histologically, OF is composed of fibrous tissue with variable cellularity and a mineralized compo-

nent that consists of woven bone, lamellar bone, and possibly cementum-like material. The most important distinguishing histological feature of OF from that of fibrous dysplasia is the presence of demarcation and/or encapsulation of OF, as opposed to the ill defined borders seen in fibrous dysplasia.²⁷³ JTOF consists of cell-rich fibrous tissue containing bands of cellular osteoid without osteoblastic rimming with immature bone lined by enlarged osteoblasts.²⁷³ JPOF, on the other hand, is characterized by a fibroblastic stroma containing small ossicles resembling psammoma bodies.²⁷³

A number of chromosomal abnormalities have been associated with OF.^{275–277} All three histological subtypes harbor neoplastic growth potential, and may exhibit aggressive, destructive behavior if left untreated. Most adult forms of OF, however, do not display the same infiltrative pattern seen in ameloblastoma, and seem to be less aggressive than their juvenile counterparts.²⁷⁸ Conventional therapy includes appropriate excision dependent on the extent and location of the tumor, with recurrence rates reported between 1 and 63%.^{278–283} Some authors advocate more extensive surgery for lesions that exhibit aggressive behavior,^{284,285} however this approach should be tempered based on various clinical and social factors. Large paranasal tumors, for example should be excised so as to prevent skull base and intracranial extension. Minimally invasive, endoscopic techniques are now available to treat many of the aggressive skull base tumors, obviating the need for more conventional craniofacial resection.

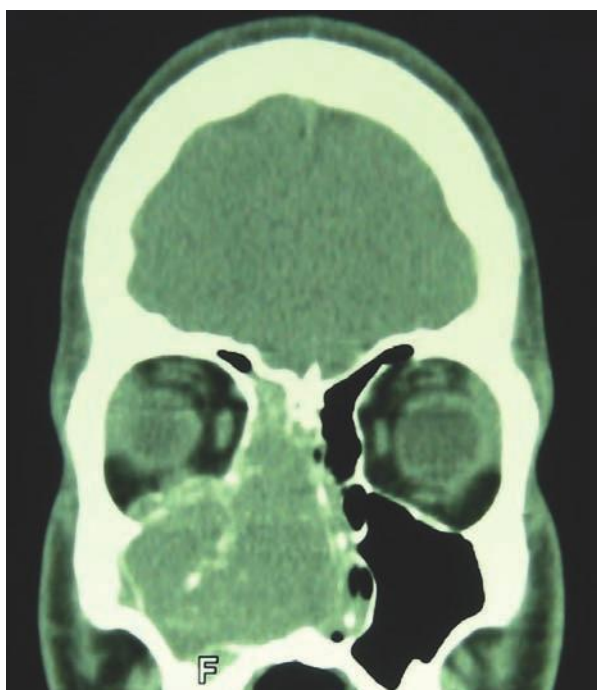


Fig. 32.31 Coronal CT scan of an ossifying fibroma of the maxilla with extension into the paranasal sinuses and anterior skull base.

Fibrous dysplasia

Fibrous dysplasia (FD) is a genetically based sporadic, non-odontogenic disease of bone that is characterized by the replacement of bone with benign fibrous tissue and that may affect single (monostotic) or multiple (polyostotic) bones throughout the human skeleton. Fibrous dysplasia involving only multiple adjacent craniofacial bones (termed craniofacial fibrous dysplasia) is regarded as monostotic.

The monostotic form has no gender or racial predilection, but is six times more common than the polyostotic form.²⁸⁶ Polyostotic FD has a female to male ratio of 3:1. The majority of cases are due to a sporadic mutation in the GNAS I gene, which encodes for the alpha-subunit of a signal transducing G-protein (Gs-alpha).^{287,288} Modulation of G protein results in increased cyclic AMP production which affects proliferation and differentiation of preosteoblasts and the phenotypic expression of FD.

FD presents most commonly in the long bones, followed by the jaws (maxilla more than mandible), base of skull, and ribs.²⁸⁹ Patients with craniofacial FD typically complain of painless swelling, facial asymmetry, loosening of teeth, malocclusion, and root resorption. Lesions encroaching on the orbital apex may cause visual impairment. Temporal bone

involvement has the potential to result in hearing loss. Uncontrolled growth in the maxilla or sphenoid can produce profound cranial neuropathies. Children that exhibit café-au-lait spots should be evaluated to rule out McCune–Albright syndrome.

The radiographic appearance has classically been described as that of a “ground glass” appearance with ill defined borders and cortical expansion (Fig. 32.32).²⁹⁰ In fact, there is a spectrum of appearances that includes an early-phase radiolucent appearance, a midphase appearance characterized by sclerosis, and a later phase that demonstrates a mixed

radiolucent/radiopaque character. Computed tomography (CT) is the imaging modality of choice, and three-dimensional reconstructions and stereolithographic models are useful in determining the extent of involvement and in planning surgery.

The histological appearance of FD is that of cellular fibrous tissue with spindle-shaped cells and immature, trabeculated woven bone (Fig. 32.33).²⁸⁶ Bundles of collagen fibers can be identified that are characteristically oriented perpendicular to the bony surface. Osteoblastic rimming can be noted, which may represent long-standing, mature lesions.

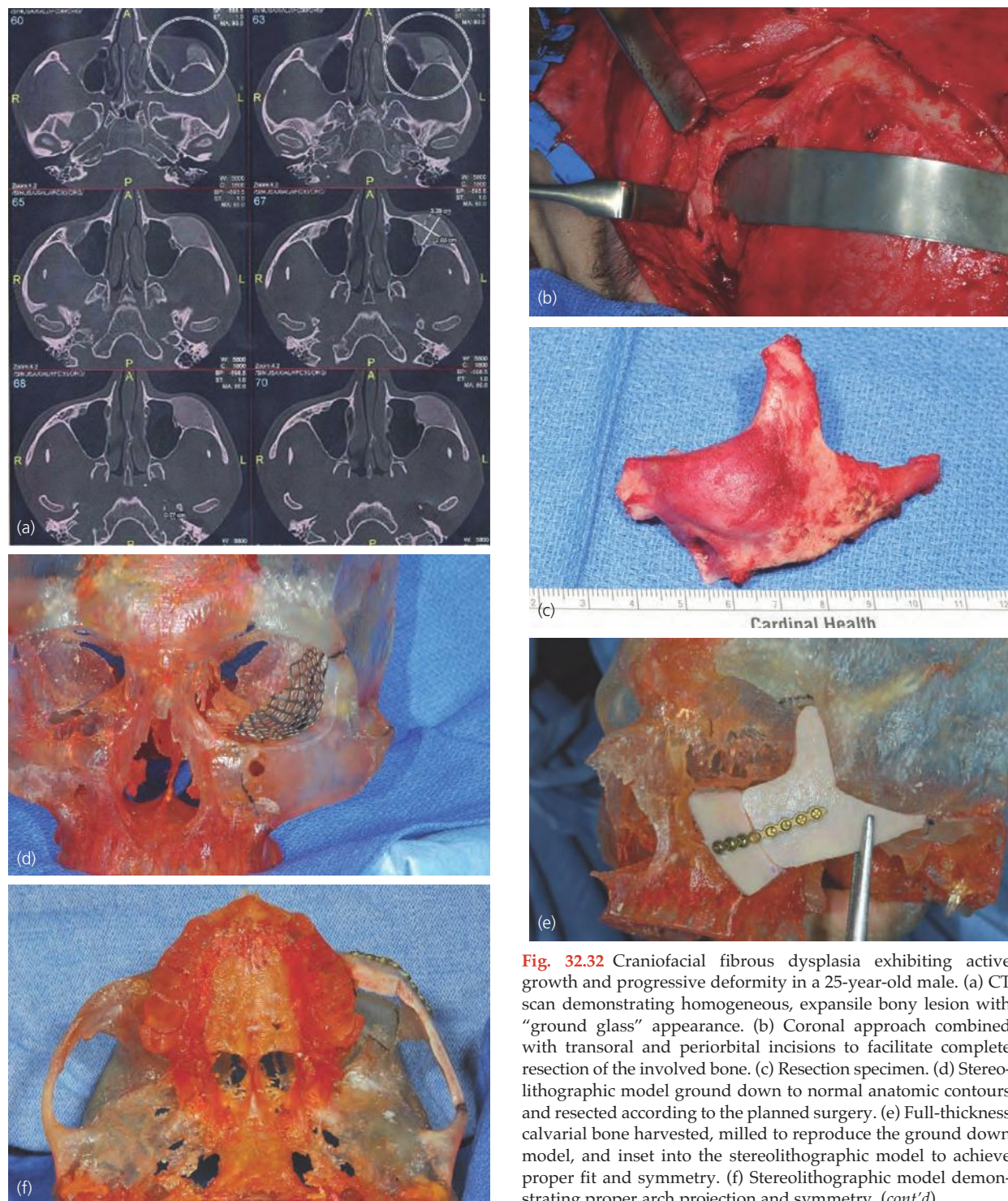


Fig. 32.32 Craniofacial fibrous dysplasia exhibiting active growth and progressive deformity in a 25-year-old male. (a) CT scan demonstrating homogeneous, expansile bony lesion with “ground glass” appearance. (b) Coronal approach combined with transoral and periorbital incisions to facilitate complete resection of the involved bone. (c) Resection specimen. (d) Stereolithographic model ground down to normal anatomic contours and resected according to the planned surgery. (e) Full-thickness calvarial bone harvested, milled to reproduce the ground down model, and inset into the stereolithographic model to achieve proper fit and symmetry. (f) Stereolithographic model demonstrating proper arch projection and symmetry. (*cont’d*)

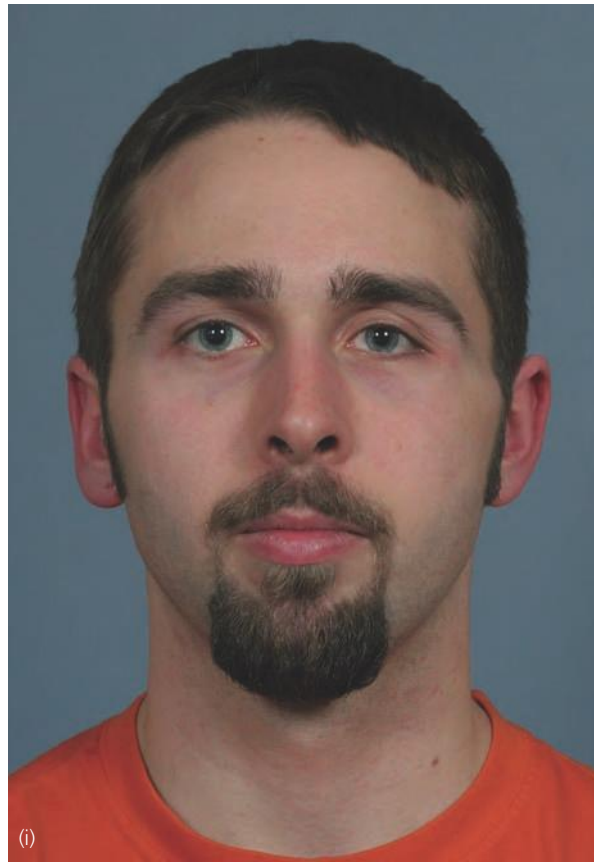
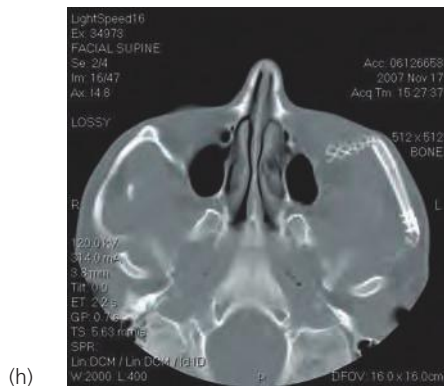
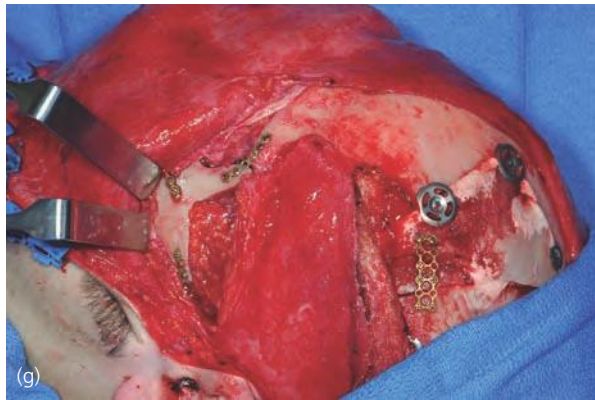


Fig. 32.32 (cont'd) (g) Inset of the neo-zygoma construct. (h) Postoperative axial CT scan of the patient demonstrating excellent symmetry. (i) Postoperative appearance of the patient.

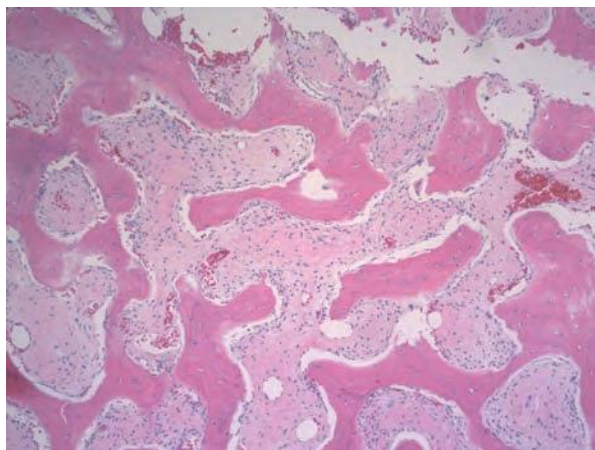


Fig. 32.33 Histological appearance of fibrous dysplasia. (Courtesy of Richard Slavin, MD.)

FD can often exhibit aggressive, destructive behavior prior to skeletal maturity; however, it often becomes indolent in adulthood. Encroachment on vital anatomic structures, such as the orbital apex or cranial foramina, mandates careful observation and/or surgical intervention if neurological deterioration is deemed eminent. Surgical recontouring or complete excision/resection can also be contemplated in cases of persistent deforming growth or in those patients that request it due to esthetic or functional concerns. Recent technological advances in computer-assisted surgery, presurgical planning using stereolithographic models, and intraoperative navigation have

armed the surgeon with a useful adjunct with which to restore these patients to form and function, with improved accuracy and predictability (Fig. 32.32).

Osseous dysplasias

Osseous dysplasias (ODs) are defined by the WHO as “idiopathic processes located in the periapical region of the tooth-bearing jaw areas, characterized by a replacement of normal bone by fibrous tissue and metaplastic bone”.²⁹¹ The terminology for this lesion is confusing due to the numerous synonyms that are used based on the site of occurrence within the jaws: in the anterior mandible the lesion has been referred to as periapical osseous dysplasia; and in the posterior mandible it is known as focal osseous dysplasia (formerly focal cemento-osseous dysplasia); periapical cemental dysplasia is a term used to describe this lesion in association with the roots of teeth; and florid osseous dysplasia describes widespread involvement of the mandible in multiple quadrants. The latter form may present with symptoms of pain or secondary infection, but the vast majority of these lesions are asymptomatic and are of no clinical significance. A rare variant that exhibits extensive and persistent expansion that can result in severe facial deformity has been reported and is referred to as a gigantiform cementoma.

The histological appearances of the various expressions of this lesion are identical: cellular fibrous tissue

surrounding sheets and strands of woven and lamellar bone interspersed between masses of cementum-like material. OD resembles ossifying fibroma and fibrous dysplasia and clinical correlation is often required to make an accurate diagnosis.²⁹²

OD does not require treatment unless complications occur (usually resulting from ill advised surgical exploration). When asymptomatic, the lesions should be observed for their clinical behavior. Biopsy should be performed in cases that demonstrate progression, cortical expansion or bony erosion. Aggressive surgical treatment is rarely needed except in the extremely unusual case of gigantiform cementoma.

Central giant cell lesion

The central giant cell lesion (CGCL) is a benign but potentially aggressive proliferation of fibroblasts and multinucleated giant cells that cause osteolysis and reactive bone formation. The CGCL was previously termed a central giant cell granuloma and central giant cell reparative granuloma in reference to the inaccurate belief that this tumor represented a reactive process. The proliferating cell in this lesion appears to be the fibroblast, which produces cytokines resulting in the recruitment of monocytes, which subsequently transform into multinucleated giant cells.^{293,294} Controversy exists with regards to whether the CGCL is unique to the jaws or whether it represents a continuum of the same disease process that affects the long bones (termed giant cell tumor).

CGCL of the jaws is most often diagnosed in children and young adults under the age of 30 years. One third of the patients are diagnosed under 20 years of age.²⁹⁵ The mandible is more often involved than the maxilla and the posterior mandible is affected more often than the anterior aspect of the jaws or the ascending ramus. The histological appearance is very similar to that of the giant cell tumor found in long bones, however some distinction has been made between the two entities by virtue of nuclei size, distribution of the giant cells, stromal cellularity, and necrosis.²⁹⁶

The lesion typically consists of spindle-shaped fibroblastic cells loosely arranged in a fibrous connective tissue stroma with areas of hemorrhage, hemosiderin deposits, macrophages, lymphocytes, granulocytes, and plasma cells (Fig. 32.34).²⁹⁷ Small clusters of osteoclast-like giant cell are found dispersed within the lesion. Mitoses are frequently observed. Brown tumor of hyperparathyroidism is histologically indistinguishable from CGCL, therefore a serum parathyroid hormone level is generally recommended to rule out primary or secondary forms of parathyroid dysfunction.

Clinical manifestations of the disease may include painless cortical expansion, facial asymmetry, cortical perforation, and/or root resorption (Fig. 32.35). Giant cell lesions have been classified as non-aggressive,

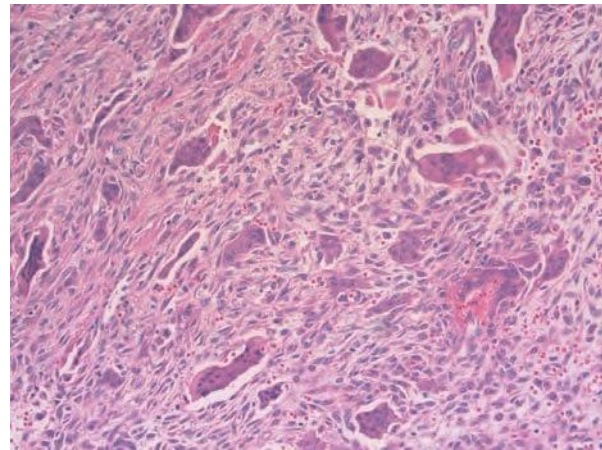


Fig. 32.34 Histological appearance of giant cell lesion. (Courtesy of Richard Slavin, MD.)

intermediate, or aggressive, based on clinical and radiographic criteria.^{298–301}

Complete excision of the tumor is required to prevent the sequela of persistent bony growth. Enucleation and curettage has been the primary mode of treatment, but is associated with an overall recurrence rate of between 15 and 50%.³⁰⁵ Non-aggressive lesions can be treated successfully by simple curettage, with a very low recurrence rate. Aggressive lesions, on the other hand, have recurrence rates as high as 70% after enucleation and curettage.^{298–305} There are no currently biological markers available to predict clinical behavior, and there is no association between histological appearance and biological behavior. Because of this high rate of recurrence and unpredictable behavior, a number of authors have described various therapeutic modalities to be used as an adjunct to conservative excision of these benign lesions.

The first non-surgical modality to be successfully applied was intralesional corticosteroid injections.³⁰⁶ Weekly injections of triamcinolone for 6 weeks have resulted in partial or complete resolution in some patients.^{306–309} Experience with this technique is varied, however, and response to therapy is unpredictable. Calcitonin has also been proposed as a definitive, non-surgical treatment for giant cell lesions, presumably based on its inhibition of osteoclastogenesis.^{310–313} Pogrel *et al.*³¹⁴ have popularized the use of subcutaneous calcitonin injections and demonstrated some success with daily subcutaneous injections carried out for approximately 18 months. To date, they have completed treatment of 27 patients with subcutaneous calcitonin and have had two failures.³¹⁴ de Lange *et al.*³¹⁵ published the only prospective, placebo-controlled randomized trial studying the efficacy of intranasally administered calcitonin. The researchers compared tumor reduction in 14 patients and found no differences between those patients treated with placebo and those treated with calcitonin. Complete remission was not observed in any patient. Finally, Kaban and colleagues³¹⁶ proposed the use of alpha-interferon given by subcutaneous injection as an

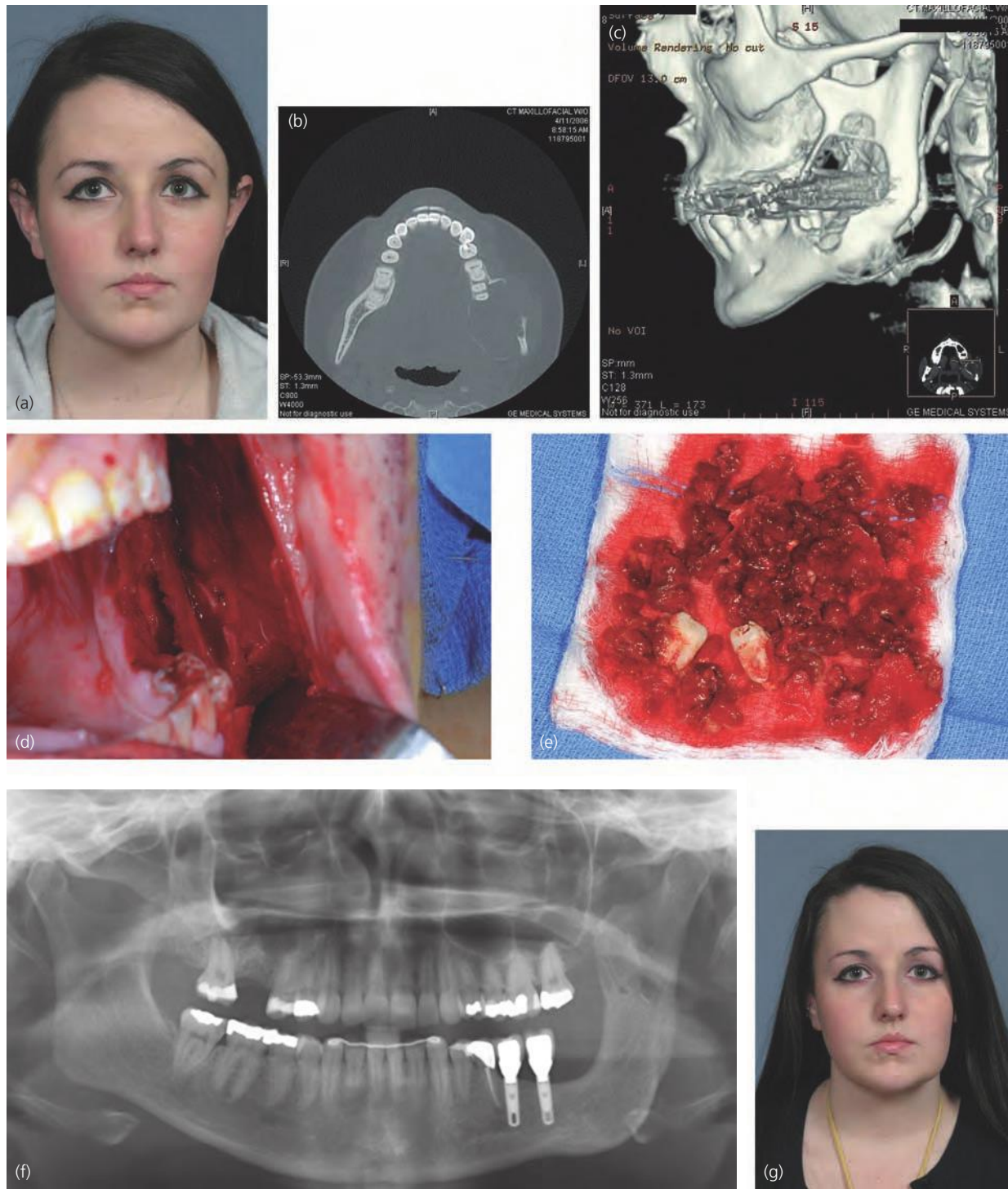


Fig. 32.35 Giant cell lesion of the mandible in a 17-year-old female. (a) Preoperative appearance with mild facial asymmetry. (b) Preoperative cone beam CT scan demonstrating multilocular, expansile lesion with cortical erosion. (c) Axial CT scan demonstrating cortical expansion and mandibular deformity. (d) Transoral excision with peripheral osteotomy and immediate anterior iliac crest bone graft reconstruction. (e) Specimen. (f) Postoperative panoramic image of the patient 2 years following surgery with maintenance of mandibular continuity and no evidence of tumor recurrence. (g) Postoperative appearance 2 years after surgery.

adjunct to surgical excision. The data thus far suggest that alpha-interferon treatment may result in less radical surgery and a reduced rate of recurrence, however this is not yet conclusive.^{298,317} Furthermore, treatment with alpha-interferon is associated with significant side-effects and potential morbidity and mortality. A recent report was published detailing the outcome of 26 patients treated at the Massachusetts

General Hospital according to a protocol of enucleation with preservation of nerves and teeth followed by treatment with interferon alpha-2a (Roferon A, Roche Laboratories, Nutley, NJ) at a dose of 3 million units/m² administered once per day by subcutaneous injection.³¹⁷ At the time of publication, all patients were either cured or in remission. Four patients enrolled in the study were unable to complete inter-

feron treatment due to significant side-effects (15%) and one of these patients required *en bloc* resection for control of residual/recurrent disease.

Brown tumor of hyperparathyroidism

Altered calcium regulation resulting from primary or secondary hyperparathyroidism or renal dysfunction can result in the formation of giant cell lesions of bone termed brown tumors due to the hemosiderin deposition within the lesion that causes a brown color (Fig. 32.36). Hyperparathyroidism is characterized by the overproduction of parathyroid hormone (PTH). Primary hyperparathyroidism is the uncontrolled production of PTH as the result of a parathyroid adenoma or adenocarcinoma. Secondary hyperparathyroidism occurs in response to hypocalcemia and is most often associated with chronic renal failure. These lesions are clinically and histologically identical to those of central giant cell lesions.

Patients with giant cell lesions should be evaluated for the presence of altered PTH levels, hypercalcemia (seen in primary hyperparathyroidism), hypocalcemia (secondary hyperparathyroidism), or impaired renal function. Brown tumors will typically resolve with treatment of the underlying metabolic abnormality.



Fig. 32.36 Brown tumor of hyperparathyroidism.

Persistent, locally destructive lesions that are recalcitrant to medical therapy should be surgically excised.^{318,319}

Cherubism

Cherubism is a rare hereditary condition that is characterized by giant cell lesions which cause symmetrical expansion of the jaws. First described in 1933, it follows an autosomal dominant pattern of inheritance with 100% penetrance in males, 50–75% penetrance in females, and variable expressivity.³²⁰ The genetic defect has been mapped to chromosome 4p16.3, which encodes the binding protein SH3BP2.^{321–324}

Cherubism is generally diagnosed in early childhood (14 months to 4 years). Mandibular involvement is most prominent, although both jaws may be affected and the process may extend into the anterior and inferior orbits. The mandibular condyles are spared. With involvement of the maxillary contribution to the orbital floor, the globes may be displaced upward, resulting in scleral show and the “looking toward Heaven” appearance that inspired the lesion’s name.

The radiographic appearance is that of bilateral multilocular radiolucencies exhibiting cortical expansion, premature exfoliation of teeth, and a “soap bubble” appearance (Fig. 32.37). The histological appearance is indistinguishable from the central giant cell lesion, but may be distinguished by the presence of hemosiderin deposits, stromal fibrosis, and perivascular cuff-like collagen deposits.

The clinical course is generally indolent. The lesions regress as the patient approaches skeletal maturity. Treatment is not recommended unless severe dysfunction is present.³²⁵

Aneurysmal bone cyst

The aneurysmal bone cyst (ABC) is defined by the WHO as an osteolytic lesion “with blood filled spaces separated by fibrous septa containing osteoclast-type giant cells and reactive bone”.³²⁶ ABCs often present



Fig. 32.37 Cherubism. Panoramic radiograph demonstrating bilateral multilocular radiolucencies exhibiting cortical expansion, premature exfoliation of teeth and “soap bubble” appearance.

as a secondary event within another bone lesion, a finding that has led some authors to question the existence of this lesion as a separate entity. Furthermore, as it often occurs within a central giant cell lesion and itself contains giant cells, other authors have suggested that the ABC is simply part of the spectrum of development in giant cell lesions. While many of these lesions are thought to be reactive, there are some cytogenetic data that suggest ABCs are indeed a distinct entity.

ABC is rare, with an incidence estimated to be 0.014 per 100 000.³²⁷ It occurs in young patients, generally below age 30 and with a peak incidence in the second decade. ABC may occur in virtually any bone in the body, and involvement of the jaws occurs in less than 3% of the cases.³²⁸ When present in the maxilla or mandible, it may present as an asymptomatic radiolucency discovered incidentally on routine dental radiography, or may present with marked facial swelling, similar to fibrous dysplasia (Fig. 32.38). Teeth remain vital and the same functional problems associated as regards encroachment on vital structures or foramina that exist in fibrous dysplasia are concerns with ABC.

The histological appearance of ABC is that of hemorrhagic, multilocular, well circumscribed blood-filled cavities that are lined by macrophages, not endothelial cells.³²⁹ Fibroblasts, osteoclast-like giant cells, and reactive bone are dispersed within osteoid, occasionally containing hemosiderin deposits and nuclear mitoses.

Genetic evidence supporting the classification of ABC as a distinct pathological entity is derived primarily from research in extracranial lesions: a 17p-rearrangement with balanced translocation with 16q has been reported by a number of investigators.^{330–333} These translocations have been regarded by some authors as resulting from acquired aberrations that provide evidence that some ABCs are clonal proliferations.

ABCs are typically treated with enucleation and curettage.³³⁴ The lesions can be highly vascular, therefore removal in an office setting is generally ill

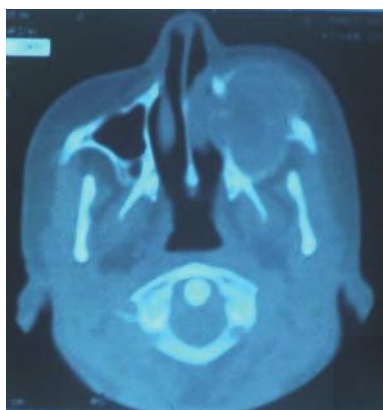


Fig. 32.38 Aneurysmal bone cyst. Axial CT scan of ABC demonstrating expansion and cortical erosion through the anterior and medial wall of the maxillary sinus.

advised for all but very small lesions. Appropriate hemostatic measures should be readied prior to surgery and even embolization has been applied successfully.^{335–337}

Simple bone cyst

The simple bone cyst (SBC) is an intraosseous pseudocyst that is devoid of an epithelial lining and may be empty or filled with serosanguinous fluid. Formerly known as a traumatic bone cyst or idiopathic bone cavity, the etiology is unknown. The most common age for presentation is in the second decade with no gender predilection.³³⁸ The mandible is the most often affected facial bone, generally, but not always, presenting in the anterior aspect.³³⁸

Most SBCs are discovered as an incidental finding of a unilocular radiolucency found on routine dental radiography. Most patients are asymptomatic, although rarely cortical expansion or teeth displacement has been reported. The histological appearance is that of a connective tissue lining covering bone with a membrane-like layer.³³⁹

Diagnosis and treatment generally occur at the same time and consist of enucleation and curettage.³⁴⁰ Occasionally, larger lesions or difficult to access sites require a more complex approach for definitive therapy.

Odontogenic carcinomas

Malignant odontogenic tumors are classified by the WHO as odontogenic carcinomas and odontogenic sarcomas.³⁴¹ All of these tumors are exceedingly rare and many are still reportable cases. They are classified as odontogenic if the tumor demonstrates epithelium that histologically resembles that seen in a developing tooth or recognizable odontogenic tumor, such as ameloblastoma or ameloblastic fibroma.

Ameloblastic carcinoma

Malignant ameloblastoma is subclassified into four distinct entities (Table 32.8),³⁴² three of them only of academic importance: (1) metastasizing ameloblastoma; (2) ameloblastic carcinoma – primary type; (3) ameloblastic carcinoma – secondary type (dedifferentiated), intraosseous; and (4) ameloblastic carcinoma – secondary type (dedifferentiated), peripheral. Metastasizing ameloblastoma is differentiated from ameloblastic carcinoma by its benign histologic appearance, despite its ability to metastasize. Ameloblastoma with metastatic deposits that exhibit cellular atypia is defined as ameloblastic carcinoma. Secondary ameloblastic carcinoma, either intraosseous or peripheral, arises in a pre-existing benign ameloblastoma.

All variants of ameloblastic carcinoma are histologically characterized by tall columnar cellular mor-

Table 32.8 World Health Organization classification of odontogenic carcinomas (2005).

Metastasizing (malignant) ameloblastoma
Ameloblastic carcinoma:
Primary type
Secondary type (dedifferentiated) intraosseous
Secondary type (dedifferentiated) peripheral
Primary intraosseous squamous cell carcinoma:
Solid type
Derived from keratocystic odontogenic tumor
Derived from odontogenic cysts
Clear cell odontogenic cysts
Ghost cell odontogenic carcinoma

phology and pleomorphism, mitotic activity, focal necrosis, perineural invasion and nuclear hyperchromatism may be present (Fig. 32.39).³⁴² Peripheral palisading and inverted nuclear polarity will be present along with a stellate reticulum.³⁴² Aneuploidy has been found to be more frequently present in the ameloblastic carcinoma and may be used as a strong predictor of malignant potential of questionable lesions.³⁴³ Recently 5q13 amplification was demonstrated by comparative genomic hybridization.³⁴⁴

The incidence of ameloblastic carcinoma is unknown, as fewer than 70 cases have been reported. Benlyazid *et al.*³⁴⁵ recently reviewed the existing literature and found 66 well documented cases. The median age of these patients was 44 years, however the tumor occurred in a wide age range of patients, with a sex ratio of 1.75 male to 1 female. The mandible was involved more frequently than the maxilla (ratio 2.14:1). Ameloblastic carcinoma seems to behave similarly to squamous cell carcinoma in terms of local invasion, regional lymph node metastasis, and distant metastasis. Of the 66 patients reviewed, 20 died of disease, and 15 developed distant metastasis.

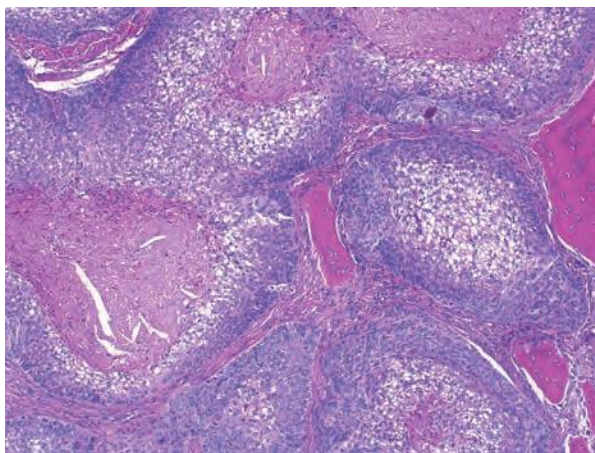


Fig. 32.39 Ameloblastic carcinoma. Histologic appearance characterized by tall columnar cellular morphology with pleomorphism, mitotic activity, focal necrosis, perineural invasion, and nuclear hyperchromatism. (Courtesy of Yi-Shing Lisa Cheng, DDS, MS, PhD.)

Excluding those patients with inadequate follow-up, the 5-year disease-specific survival rate was 67.8%.

Optimal treatment of ameloblastic carcinoma is unknown, but there is general agreement that it should be managed in similar fashion to squamous cell carcinoma of the oral cavity, consisting of composite resection with 1 cm tumor-free margins, neck dissection, and immediate reconstruction with microvascular free flaps.^{345,346} Adjuvant radiation therapy should be considered for all patients, especially those cases exhibiting unfavorable histologic characteristics, such as positive resection margins, perineural invasion, and lymph node metastasis.

Primary intraosseous squamous cell carcinoma

Primary intraosseous squamous cell carcinoma (PIOSCC) is a central jaw carcinoma derived from epithelium arising within benign odontogenic cysts or tumors, usually a dentigerous cyst or keratinizing odontogenic tumor (formerly odontogenic keratocyst).³⁴⁷ The incidence of malignant transformation of odontogenic cysts has been estimated to be approximately 0.12%, with keratinizing cysts demonstrating a higher incidence than non-keratinizing cysts.³⁴⁸ While originally thought to be extraordinarily rare, intraosseous squamous cell carcinoma of the jaws is becoming more frequently recognized. Gardner³⁴⁹ reviewed the literature from 1889 to 1967 and found only 25 examples of carcinoma arising from an odontogenic cyst. Yasuoka and colleagues³⁵⁰ identified an additional 50 cases from 1967 to 2000. The author's review of the literature revealed fewer than 60 cases of carcinoma arising in odontogenic cysts reported since 1967, and 13 of them were keratinizing odontogenic tumors.^{348–364} The average age of patients presenting with carcinoma arising within a cystic lining is 58.8 years with a range of 25–84 years.³⁵⁰ The most common sites are generally the same as for non-malignant cysts, most commonly the mandibular third molar, maxillary canine and third molar, and mandibular second premolar regions.

Diagnosis of so-called carcinoma ex-odontogenic cyst is challenging. It is often difficult to determine if the cancer arose from a cystic epithelium, oral mucosal epithelium, salivary gland tissue or *de novo*. Criteria for a diagnosis of carcinoma arising in a cyst have been defined by Gardner and are as follows: (1) a microscopic transition area from benign cystic epithelial lining to invasive malignant squamous cell carcinoma; (2) no carcinomatous change in the overlying epithelium; and (3) no source of carcinoma in the adjacent structures.^{349,365} Occasionally, the diagnosis of squamous cell carcinoma is made in the presence of a cystic lesion thought to be a recurrent keratocyst.

A high degree of suspicion must be maintained by the clinician if the cyst demonstrates abnormally aggressive behavior, ulceration, or neurosensory

changes, or if the patient develops cervical lymphadenopathy. Any suspicion should warrant a biopsy prior to definitive management. The role of tumor markers such as p53 is being defined. As the technology progresses, immunohistochemistry and other molecular genetic studies will aid in the diagnosis and potentially provide prognostic information on which to base treatment.

Treatment is similar to that of invasive squamous cell carcinoma with bony involvement: radical resection with ipsilateral neck dissection and immediate reconstruction using microvascular composite free flaps. Although data are lacking, adjuvant radiation therapy or chemoradiation therapy should be considered depending on the tumor's histopathological characteristics, margin status, and the status of the regional lymph nodes.

Clear cell odontogenic carcinoma

Clear cell odontogenic carcinoma (CCOC) is defined by the WHO as a malignant odontogenic tumor that is characterized by sheets and islands of vacuolated and clear cells. Previously known as clear cell ameloblastoma or clear cell odontogenic tumor, fewer than 40 cases have been reported since the early 1990s.^{367–369} There is a strong female predilection and it tends to occur in the mandible of older patients, with a mean age at diagnosis of 60 years.

The histological appearance is that of a fibrous stroma with islands of epithelial cells revealing clear to faintly eosinophilic cytoplasm, well demarcated cell membranes, and irregular dark-staining nuclei. The clear cells are positive for cytokeratin and negative for vimentin and mucicarmine, S-100 protein, desmin, smooth muscle actin, HMB 45, and alpha antichymotrypsin, which differentiates it from other clear cell tumors such as mucoepidermoid carcinoma, renal carcinoma, and CEOT.³⁶⁹ Regional and distant metastasis have been described and should be treated in similar fashion to squamous cell carcinoma. Adjuvant radiation therapy should be considered following composite resection, neck dissection, and microvascular free flap reconstruction.

Ghost cell odontogenic carcinoma

Ghost cell odontogenic carcinoma is a malignant odontogenic epithelial tumor that is defined by the WHO as having “features of calcifying cystic odontogenic tumor and/or dentinogenic ghost cell tumor”.³⁷⁰ This tumor is extremely rare, having been reported less than two dozen times in the English literature. It may be more common in Asia, appears to affect more men than women, and occurs in a wide age range. Twice as many tumors occur in the maxilla as in the mandible, similar to that of the calcifying odontogenic cyst.

Histological diagnosis is based on the identification of malignant epithelial cells within the histologi-

cal architecture of a calcifying cystic odontogenic tumor. Ghost cells are found in varying numbers. Immunohistochemical overexpression of p53 protein has been reported.³⁷¹ PCNA labeling may also be used to assist in diagnosis.

The clinical presentation is typical of the malignant osseous lesions. Imaging may demonstrate an osteolytic radiolucency with or without calcifications, which is poorly defined, may displace or destroy tooth roots, and extend into adjacent tissues. Treatment data are lacking; however, presumably the lesion should be managed with radical resection, neck dissection, and immediate reconstruction using composite free tissue transfer.^{370–374} Adjuvant radiation therapy or chemoradiation therapy should be considered, depending on the histopathologic features, margin status, and status of the cervical lymph nodes.

Odontogenic sarcomas

Odontogenic sarcomas are very rare tumors. The WHO defines two distinct types: ameloblastic fibrosarcoma and ameloblastic fibrodentinosa and fibro-odontosarcoma.³⁷⁵

Ameloblastic fibrosarcoma

The ameloblastic fibrosarcoma (AFS) is the malignant counterpart of the ameloblastic fibroma. An odontogenic tumor with a benign epithelial and a malignant ectomesenchymal component, the etiology is unknown. Many of these tumors appear to represent malignant transformation of pre-existing ameloblastic fibroma.³⁷⁵ They can occur at virtually any age (3–89 years), however most cases are diagnosed in the second decade of life.³⁷⁵ Patients with pre-existing ameloblastic fibroma appear to develop malignant tumors later than those patients whose tumor arises *de novo*.³⁷⁹ Two thirds of the patients are males and the mandible is the most common site of occurrence.

The clinical presentation of AFS is that of an expansile intraosseous lytic mass with ill defined borders, cortical erosion, and pain.³⁷⁹ The histological characteristics are those of ameloblastic fibroma in which the epithelium is benign, but the connective tissue component is malignant. The connective tissue stroma displays mitotically active cells surrounding the bland epithelial component.

The biological behavior of AFS appears to be somewhat less aggressive than the hard and soft tissue counterparts, such as osteosarcoma or rhabdomyosarcoma.^{375–380} Of more than 60 reported cases, only one had metastasis (mediastinal and liver).

Ameloblastic fibrodentinosa and fibro-odontosarcoma

The WHO defines these rare tumors as containing the histological features of ameloblastic fibrosarcoma,

together with dysplastic dentin (fibrodentinosarcoma) and/or enamel/enameloid and dentin/dentinoid (fibro-odontosarcoma).³⁷⁵ This tumor is extremely rare (14 cases in the literature). The histological appearance is typical of ameloblastic fibrosarcoma, except that the dental hard tissues are also dysplastic. The biological behavior seems to be less aggressive than that of other sarcomas and is generally regarded as low grade.^{379,380} Only one case has demonstrated an ability to metastasize. Treatment is radical resection and the prognosis appears to be good.

Non-odontogenic malignant jaw tumors

Osteosarcoma

Osteosarcoma (OS) is a malignant tumor of bone characterized by the formation of osteoid by neoplastic cells. OS is the most common primary sarcoma of bone and only plasma cell neoplasms outnumber this tumor in the category of all primary bone tumors. It has been classified as central type, which is more common and arises from the medullary portion of the bone, and peripheral or juxtacortical type, which is less common and originates on the surface of bone. OS may develop from pre-existing bone disorders such as Paget's disease, giant cell tumors, or fibrous dysplasia, or from prior radiation therapy, but most arise *de novo*. The molecular mechanisms associated with the pathogenesis of OS are related to a variety of genetic alterations involving tumor suppressor genes, oncogenes, and complex chromosomal abnormalities.³⁸¹

Central osteosarcomas most often involve the distal femur and proximal tibia of patients in their second decade of life. OS involving the jaws accounts for 5–7% of all OS, but the presentation appears to be older, more often presenting in the third or fourth decade of life. There is a slight male predilection and the mandible is more commonly affected than the maxilla.

The clinical presentation of OS is commonly that of jaw pain and swelling. Paresthesia is common in mandibular lesions, as is loosening of teeth. Nasal obstruction, epistaxis, proptosis, or diplopia may all be presenting signs or symptoms in advanced maxillary lesions. The radiographic appearance of central OS is classically that of an osteolytic lesion that is associated with symmetrical widening of the periodontal ligament and extracortical bone producing a "sunburst" appearance. In fact, the radiographic appearance is quite variable and can range from that of a dense radiopacity to a mixed radiopaque and radiolucent lesion to a radiolucent lesion (Fig. 32.40). The "sunburst" quality is a non-specific finding, as are cortical destruction and root resorption which are also commonly found.

The histological appearance of OS of the jaws is similar to that of other primary mesenchymal malignant neoplasms and characterized by pleomorphic spindle cells producing a variable amount of osteoid. The lesions are classified as osteoblastic, chondroblastic, and fibroblastic, depending on the relative amounts of osteoid, cartilage, or collagen that is produced by the malignant stroma. The osteoblastic type is the most common in the axial skeleton, but the chondroblastic type is most common in the jaws. A telangiectatic subtype of OS has been described which contains large blood-filled spaces and prominent multinucleated giant cells.³⁸² A small cell variant, similar to Ewing sarcoma has also been described in the jaws.³⁸³ The histologic subtypes do not predict prognosis. Indeed, biological behavior is best predicted by histologic grade, with high-grade lesions portending a poor prognosis.

Treatment for all subtypes includes radical resection (Fig. 32.40). The most important prognostic indicator of successful outcome is negative resection margins. Consequently, bone margins of up to 3 cm have been recommended. Well-designed, prospective outcome data for OS of the jaws are lacking. Radiation therapy, either as definitive treatment or in the adjuvant setting has not been shown to be beneficial.^{384,385} Current treatment strategies that employ a multimodal approach incorporating preoperative and postoperative chemotherapy for cases involving the long bones have resulted in dramatic survival improvement when compared to previous outcomes.³⁸⁵ Current recommendations for craniofacial OS include preoperative and postoperative chemo-

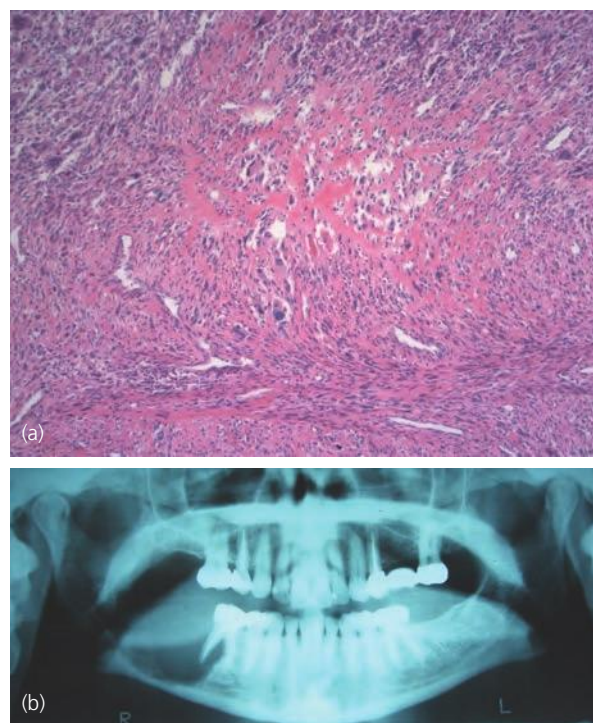


Fig. 32.40 Osteogenic sarcoma in 48-year-old female. (a) Histological appearance (courtesy of Richard Slavin, MD). (b) Preoperative panoramic radiograph demonstrating aggressive features of cortical destruction and root resorption. (cont'd)

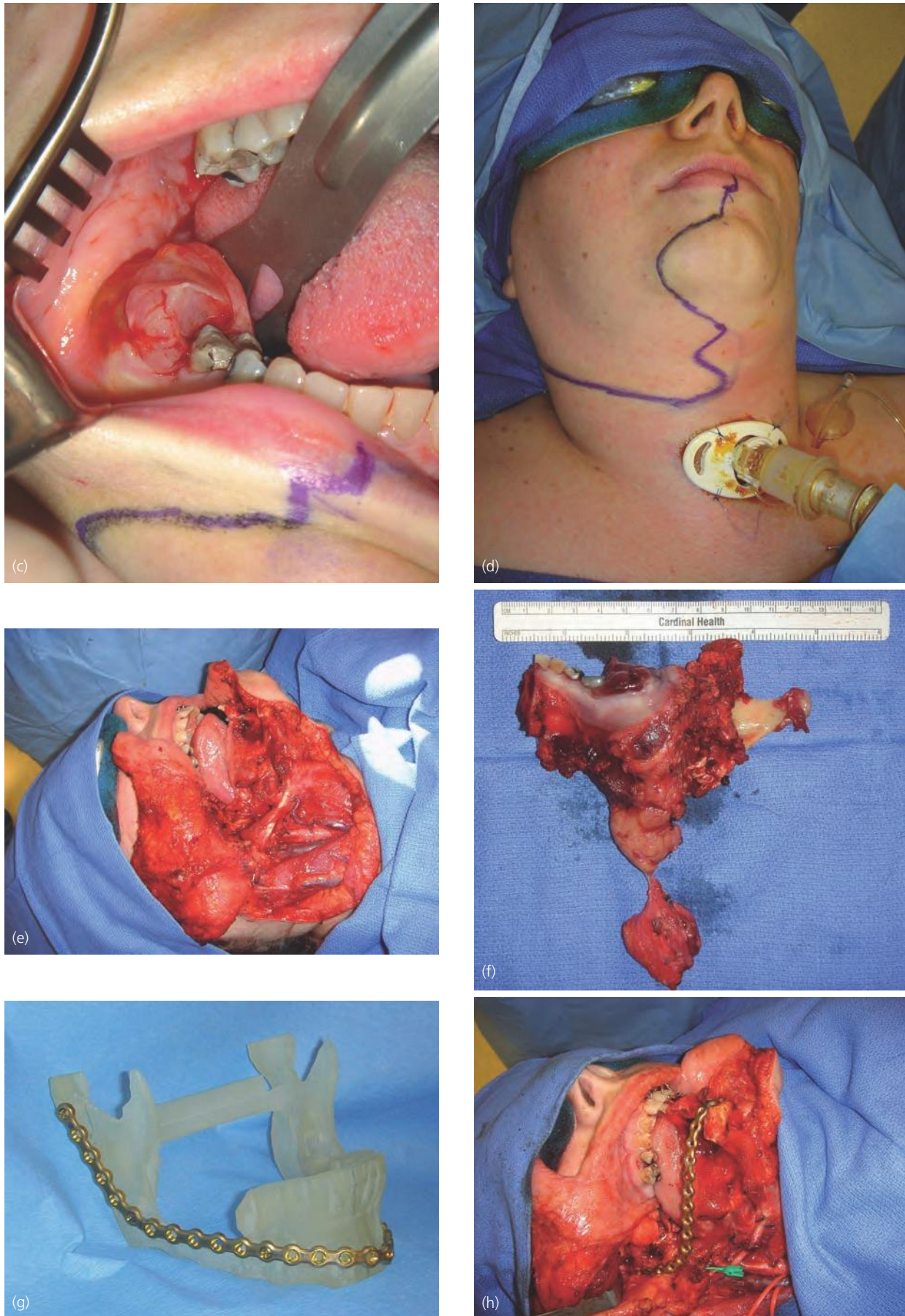


Fig. 32.40 (cont'd) (c) Clinical appearance of the tumor. (d) Outline of lip-splitting incision used to facilitate composite resection. (e) Composite resection of the floor of mouth and mandible with selective neck dissection, primarily for vascular access. (f) Resection specimen. (g) Stereolithographic model to prebend reconstruction plate prior to ablative surgery. (h) Inset of a fibular osteocutaneous free flap for reconstruction of the ablative defect.

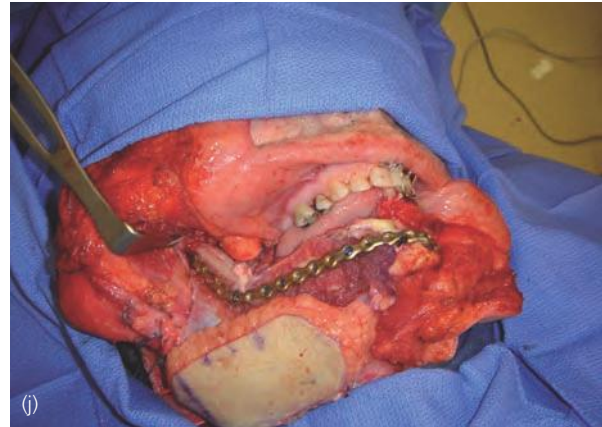


Fig. 32.40 (cont'd) (i) Fibular flap harvested. (j) Inset of the flap.

therapy in addition to radical resection and free flap reconstruction.³⁸⁶⁻³⁹⁹

In contrast to the central medullary OS, the peripheral OS may be less aggressive and is associated with a more favorable prognosis in long bones. There are two subtypes of peripheral OS: parosteal and periosteal. The parosteal variant appears as a radiodense mass on the cortical surface or the maxilla or mandible. Peripheral OS arises from a broad pedicle and enlarges along the cortical surface in a pedunculated fashion. A radiolucent line is noted between the tumor and the underlying cortex. Daughter masses may be seen in the region as well. The periosteal type lacks the pedunculated growth pattern and is usually not as radiodense. It is also thought to have a higher recurrence rate and greater metastatic potential in comparison with the parosteal subtype.

Most reports cite an overall 5-year survival rate for head and neck OS of 40–70%. Metastasis occurs in approximately 18–50% of cases, generally occurring in the lungs. The hematogenous route does not invite regional lymph node involvement and therefore cervical lymphadenectomy is not advocated.

Chondrosarcoma

A chondrosarcoma (CS) is a malignant tumor characterized by the formation of cartilage by malignant tumor cells. Only 1% or 2% of all chondrosarcomas are located in the head and neck. It is the second most common primary sarcoma of bone. Unlike osteosarcoma, chondrosarcoma is more frequent in the maxilla than the mandible. The maxillary lesions occur most often in the anterior region and the mandibular tumors occur most often in the posterior mandible. CS occurs over a wide age range without gender predilection, and generally occurs in the third decade of life.⁴⁰⁰

The most common clinical presentation is that of a slow-growing, painless mass in the involved anatomic region. The radiographic appearance is variable, although most lesions demonstrate an osteolytic process with ill defined margins, cortical erosion, and destructive behavior. Radiopacities are often present.

Similar to osteosarcoma, there may be a “sunburst” pattern with symmetrical widening of the periodontal ligament space around involved teeth.

The histological appearance of CS is characterized by variable amounts of cartilage formation, but not osteoid or bone, emanating from a sarcomatous stroma. If the malignant cellular elements are noted to produce osteoid or bone in any portion of the tumor, it is considered an osteosarcoma. Calcification of the chondroid matrix does occur in CS, however. The hypercellular tumor exhibits an increased number of cartilage cells with plump nuclei, binuclear or multinuclear cartilage cells, nuclear hyperchromatism, and cellular and nuclear pleomorphism. Mitotic figures may be scarce or absent. CS has been classified into grades I, II, and III on the basis of mitotic rate, cellularity, and nuclear size, all of which correlate with prognosis.⁴⁰²

The treatment of choice for CS is radical resection. Unlike osteosarcoma, chemotherapy has not been shown to be of significant therapeutic benefit.^{400,402} Radiation therapy may be considered in cases of unresectable, residual, or recurrent tumors. The prognosis for CS of the jaws is worse than that for tumors of the axial skeleton. The overall 5-year survival for CS of the jaws ranges from 32–81%.⁴⁰⁰⁻⁴⁰⁴ Factors influencing the prognosis include the site of origin, histological grade, treatment modality, and adequacy of the surgical resection. Mandibular tumors have a more favorable prognosis than maxillary tumors. Increasing grade correlates with worsening prognosis.⁴⁰⁵⁻⁴⁰⁷ Like osteosarcoma, the lung is the most common site of metastasis and cervical lymph nodes are generally not involved. Neck dissection is therefore not recommended.

Ewing sarcoma

Ewing sarcoma (ES) is one of the “small, round, blue cell” tumors and is in the family of tumors that includes the primitive neuroectodermal tumors. These tumors are characterized by a recurrent t(11;22)(q24;q12) chromosomal translocation, which is detectable in approximately 85% of cases (Fig. 32.41). ES accounts

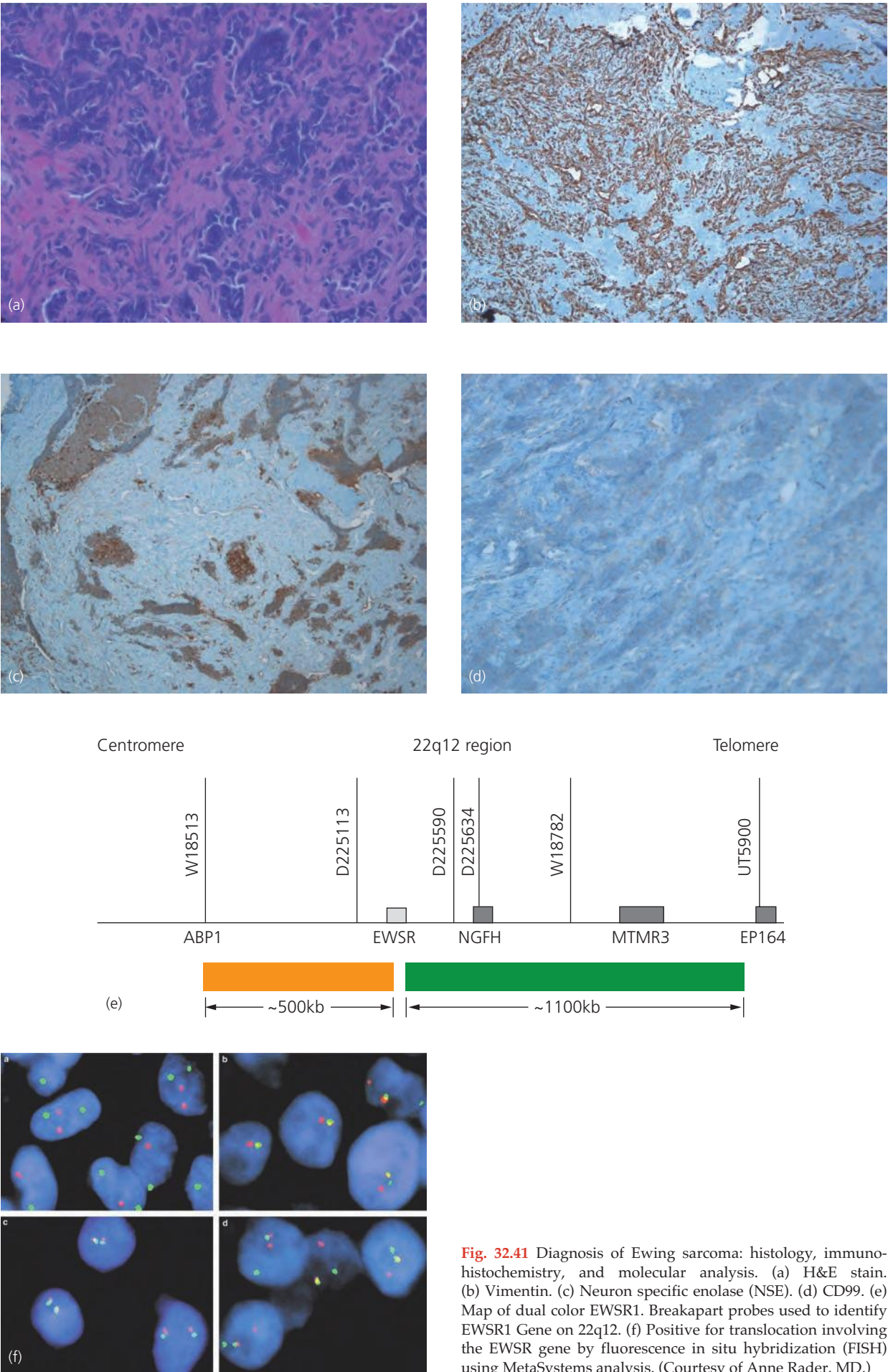


Fig. 32.41 Diagnosis of Ewing sarcoma: histology, immuno-histochemistry, and molecular analysis. (a) H&E stain. (b) Vimentin. (c) Neuron specific enolase (NSE). (d) CD99. (e) Map of dual color EWSR1. Breakapart probes used to identify EWSR1 Gene on 22q12. (f) Positive for translocation involving the EWSR gene by fluorescence in situ hybridization (FISH) using MetaSystems analysis. (Courtesy of Anne Rader, MD.)

for 6–8% of primary bone malignancies, although it is second to osteosarcoma as the most common sarcoma in bone and soft tissue in children. The pelvis and lower extremities are most commonly affected. Jaw tumors account for less than 3% of ES. ES primarily presents in children and young adults, usually under 20 years of age. A male predilection exists.

The clinical signs and symptoms usually include rapidly progressing pain and swelling (Fig. 32.42). Tumor expansion may also cause paresthesia or tooth mobility. The radiographic appearance is that of other malignant jaw tumors, including an osteolytic process with ill defined borders and displacement or resorption of teeth or tooth roots. Growth may be rapid and progressive. Metastatic disease is present in 15% of the patients with non-pelvic tumors at the time of diagnosis.

The histological appearance is that of closely packed cells that may be compartmentalized by fibrous bands, with round to oval nuclei, and finely dispersed chromatin. The cytoplasm of the tumor cell frequently stains with periodic acid-Schiff stain.

Patients should be evaluated by specialists from all disciplines (e.g. radiologist, medical oncologist, pathologist, surgeon, and radiation oncologist) as early as possible. Appropriate imaging studies of the site should be obtained prior to biopsy. The surgeon who will perform the definitive surgery should be involved prior to or during the biopsy so that the incision can be placed in an acceptable location. The radiation oncologist and pathologist should be consulted prior to biopsy/surgery in order to be sure that the incision will not compromise the radiation port and so that multiple types of tissue samples are obtained; including fresh tissue for cytogenetics and flow cytometry, frozen tissue, and formalin-fixed tissue.

The successful treatment of patients with Ewing family of tumors (EFT) requires systemic chemotherapy^{408–414} in conjunction with either surgery or radiation therapy or both modalities for local tumor control.^{415–419} In general, patients receive preoperative chemotherapy prior to instituting local control measures. In patients who undergo surgery, surgical margins and histologic response are considered in

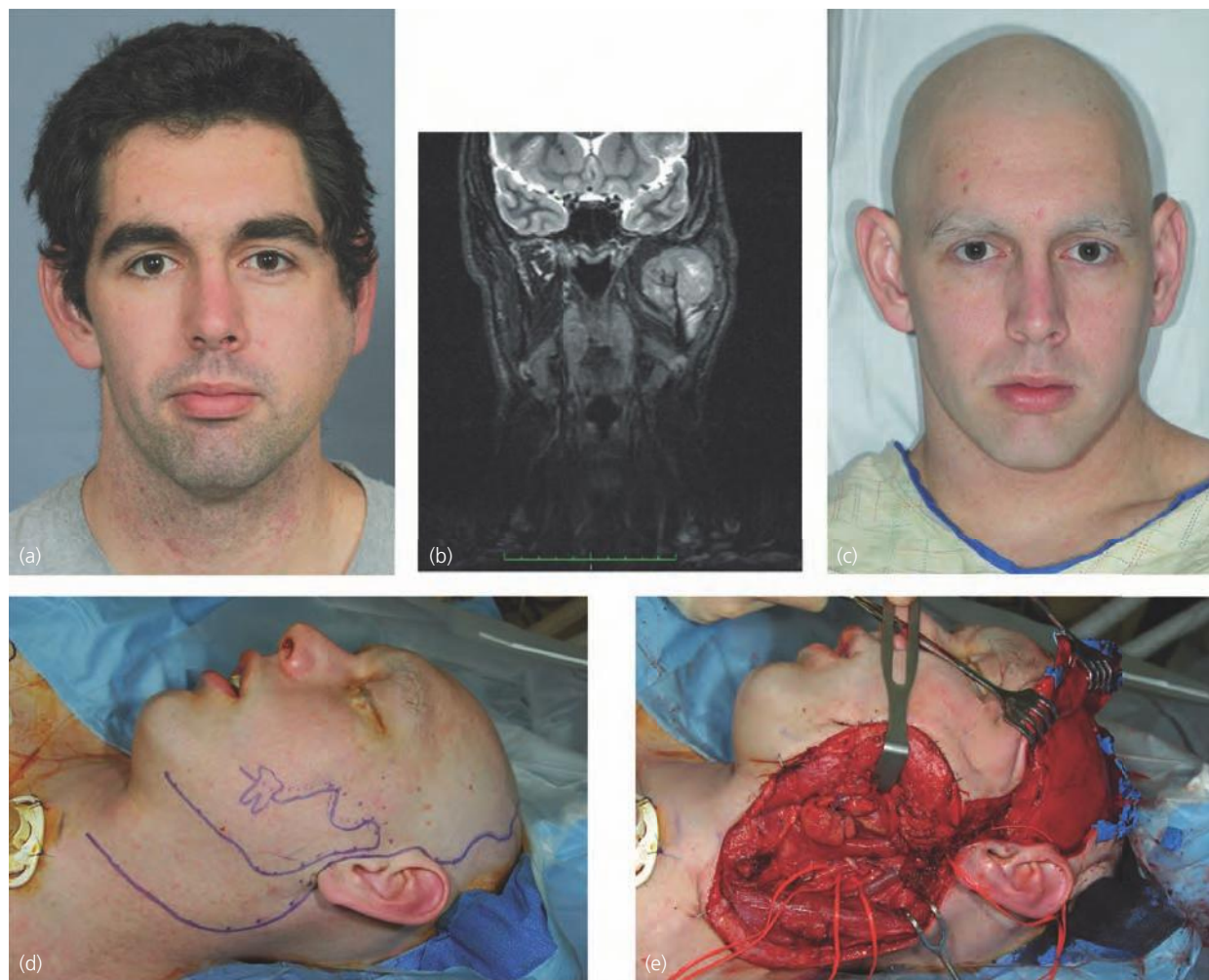


Fig. 32.42 A 25-year-old male with Ewing sarcoma involving the mandible, masticator space, and infratemporal fossa. (a) Preoperative photograph. (b) Pretreatment sagittal MRI demonstrating large tumor emanating from the mandibular condyle with involvement of the masticator space and infratemporal fossa. (c) Appearance following preoperative chemotherapy. (d) Outline of the planned surgical resection to include composite resection of the mandible, masticator space, and infratemporal fossa via combined transcervical and infratemporal approach using computer modeling and intraoperative navigation. (e) Intraoperative photograph demonstrating the surgical approach with neck dissection for vascular access and preservation of the facial nerve. (*cont'd*)

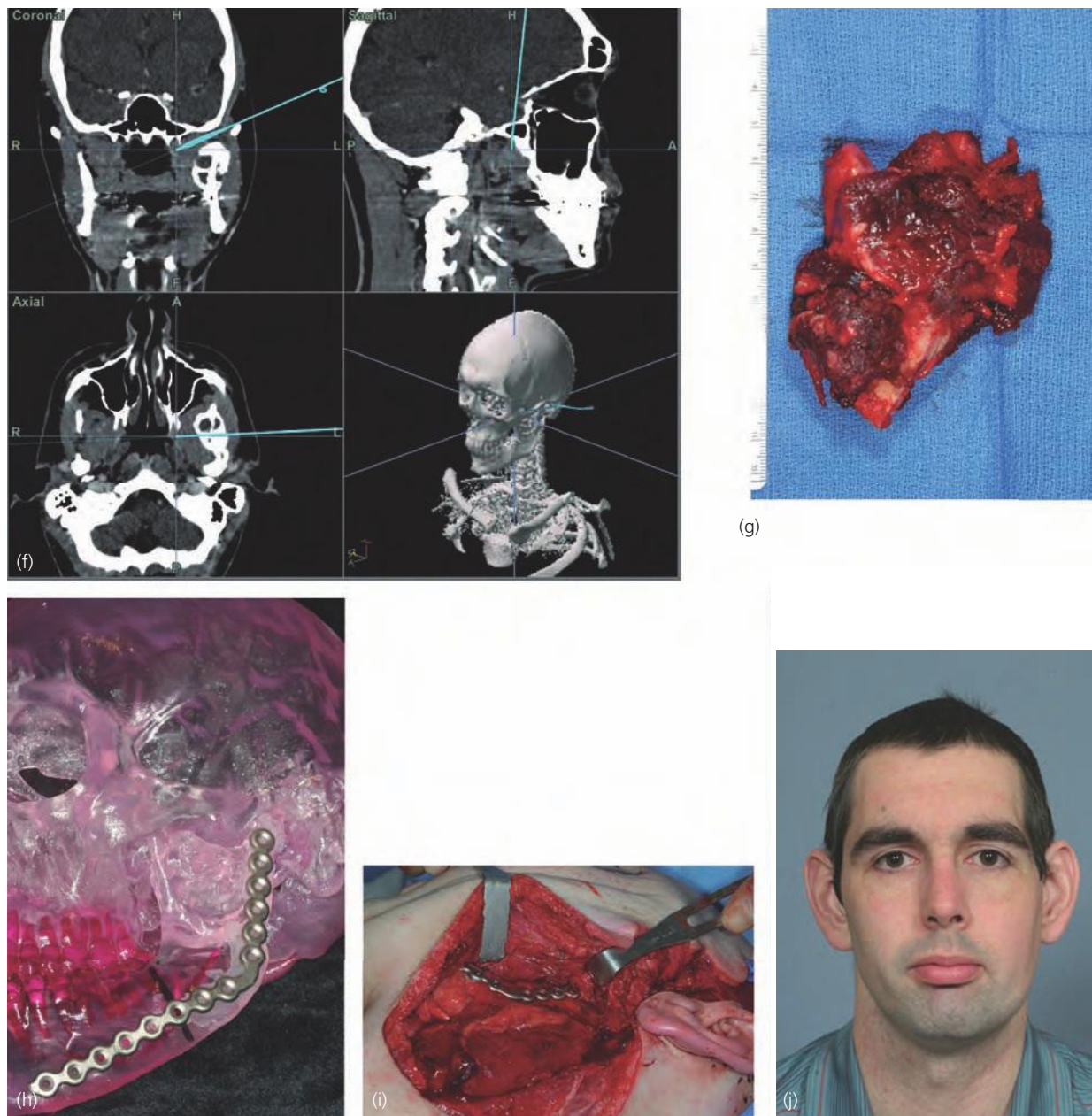


Fig. 32.42 (cont'd) (f) Intraoperative navigation used to assist in safe and accurate skull base resection. (g) Resection specimen. (h) Stereolithographic model used to plan resection and prebend reconstruction plate. (i) Inset of the fibular fascio-osseous free flap. (j) Postoperative photograph 3 months following surgery demonstrating resolving lymphedema, complete facial nerve function, and favorable esthetics.

planning postoperative therapy. In the Euro-Ewing study, patients who receive radiation alone for local control are stratified by pretreatment tumor volume for postradiation therapy.⁴²⁰ Most patients with metastatic disease have a good initial response to preoperative chemotherapy; however, in most cases, the disease is only partially controlled or recurs.^{420–423} Patients with lung as the sole metastatic site have a better prognosis than patients with metastases to bone and/or bone marrow.

Randomized clinical trials during the last 15 years have led to general acceptance of the combination of vincristine, doxorubicin, and cyclophosphamide as standard therapy for Ewing sarcoma.^{420–424} With these drugs and adequate treatment of the primary tumor, about 60% of patients with localized disease

can be cured. For patients with metastatic disease at diagnosis, the prognosis is much worse, with only 30–40% surviving long-term. Phase III clinical trials are underway using alternating cycles of vincristine–doxorubicin–cyclophosphamide and ifosfamide–etoposide, with granulocyte colony stimulating factor (filgrastim, G-CSF) for intensification of chemotherapy. Interdisciplinary cooperation is essential, particularly for complex head and neck tumors.^{425,426}

Malignant peripheral nerve sheath tumor

Malignant peripheral nerve sheath tumors (MPNSTs) comprise a rare group of tumors that arise from peripheral nerves or that display differentiation along

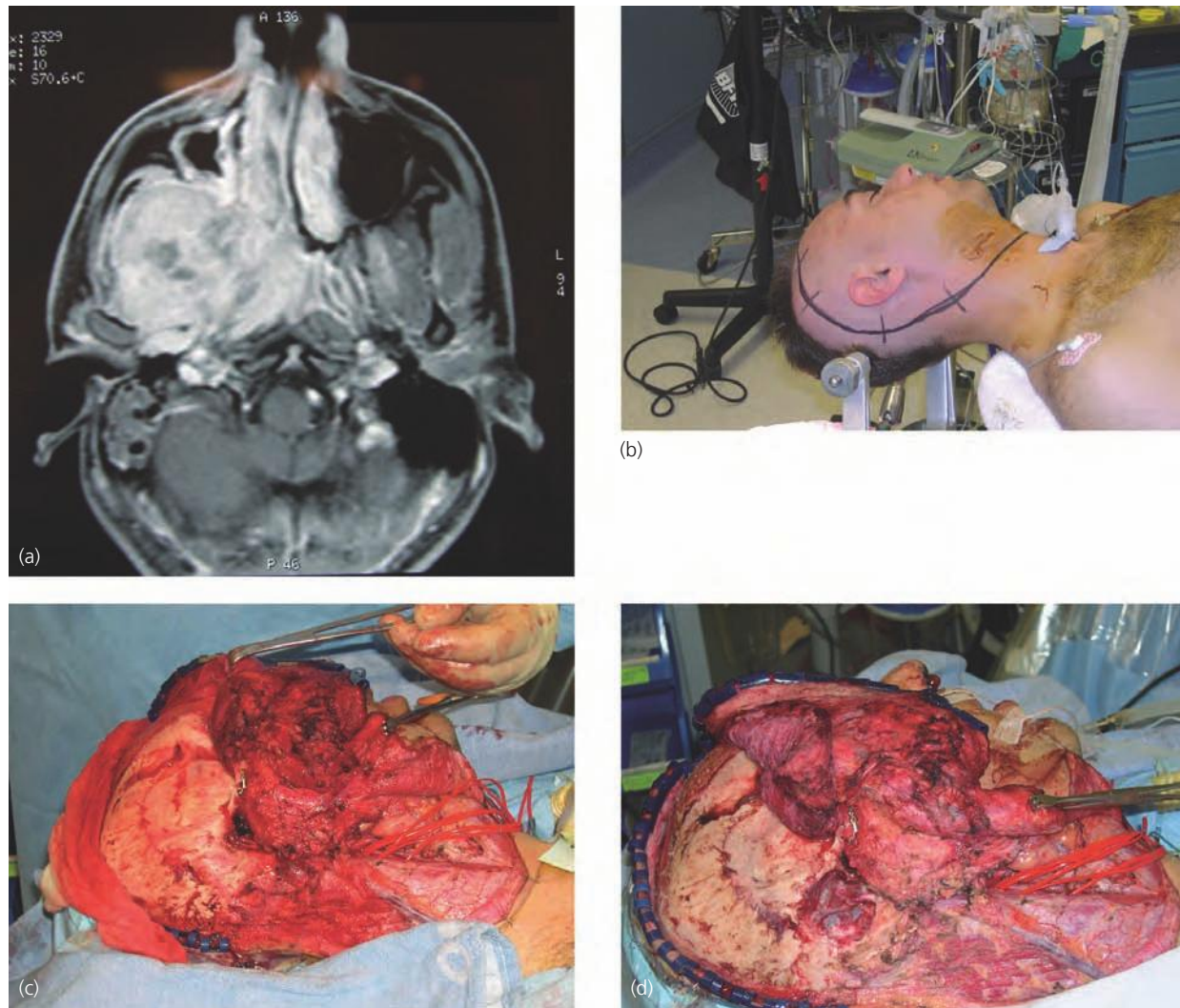


Fig. 32.43 A 21-year-old male with malignant peripheral nerve sheath tumor involving the anterior and lateral skull base. (a) Pretreatment axial MRI demonstrating massive tumor with extensive involvement of the anterior and lateral skull base. (b) Outline of the planned composite resection of the anterior and lateral skull base via a transcranial and infratemporal fossa approach to include resection of the maxilla, mandible, temporal bone with facial nerve sacrifice and interpositional nerve graft, orbital exenteration, and partial pharyngectomy with immediate reconstruction using a rectus muscle free flap. (c) Intraoperative photograph demonstrating the infratemporal fossa approach to the lateral skull base with disarticulation of the zygoma and neck dissection for vascular access. (d) Intraoperative photograph demonstrating lateral temporal bone resection with exposure of the sigmoid sinus, division of the facial nerve, and mobilization of the flap forward exposing the temporal bone. (*cont'd*)

the lines of the various elements of the nerve sheath, including Schwann cells, perineural fibroblasts, or fibroblasts. This group of tumors was previously referred to as malignant schwannoma, neurofibrosarcoma, or neurogenic sarcoma. MPNSTs account for about 5% of all soft tissue sarcomas. Although approximately half of the patients with this disorder will have a history of neurofibromatosis type 1, many cases will arise *de novo*, or as a postradiation sarcoma. Generally occurring in the extremities or trunk, they are seldom found in the head and neck area. The most common site in the head and neck for intraosseous tumors is the mandible, although skull base tumors originating various cranial nerves are relatively frequent. Patients generally present with a rapidly growing mass, pain, and/or neurosensory distur-

bance within the anatomic distribution of the involved region or nerve.

Radiographically, tumors of the mandible may produce widening of the inferior alveolar canal or the mental foramen. More commonly, they invade the mandible from adjacent structures or cranial nerves (Fig. 32.43). The microscopic appearance is that of fascicles of spindle cells that closely resemble the cells of fibrosarcoma. The nuclei may be wavy or comma shaped and nuclear pleomorphism may be present. MPNSTs are S-100 protein positive in 50% of cases.

MPNST is considered one of the most aggressive malignant lesions in the head and neck, with 5-year overall survival reported to be between 5–58%, depending on tumor size, location status of surgical

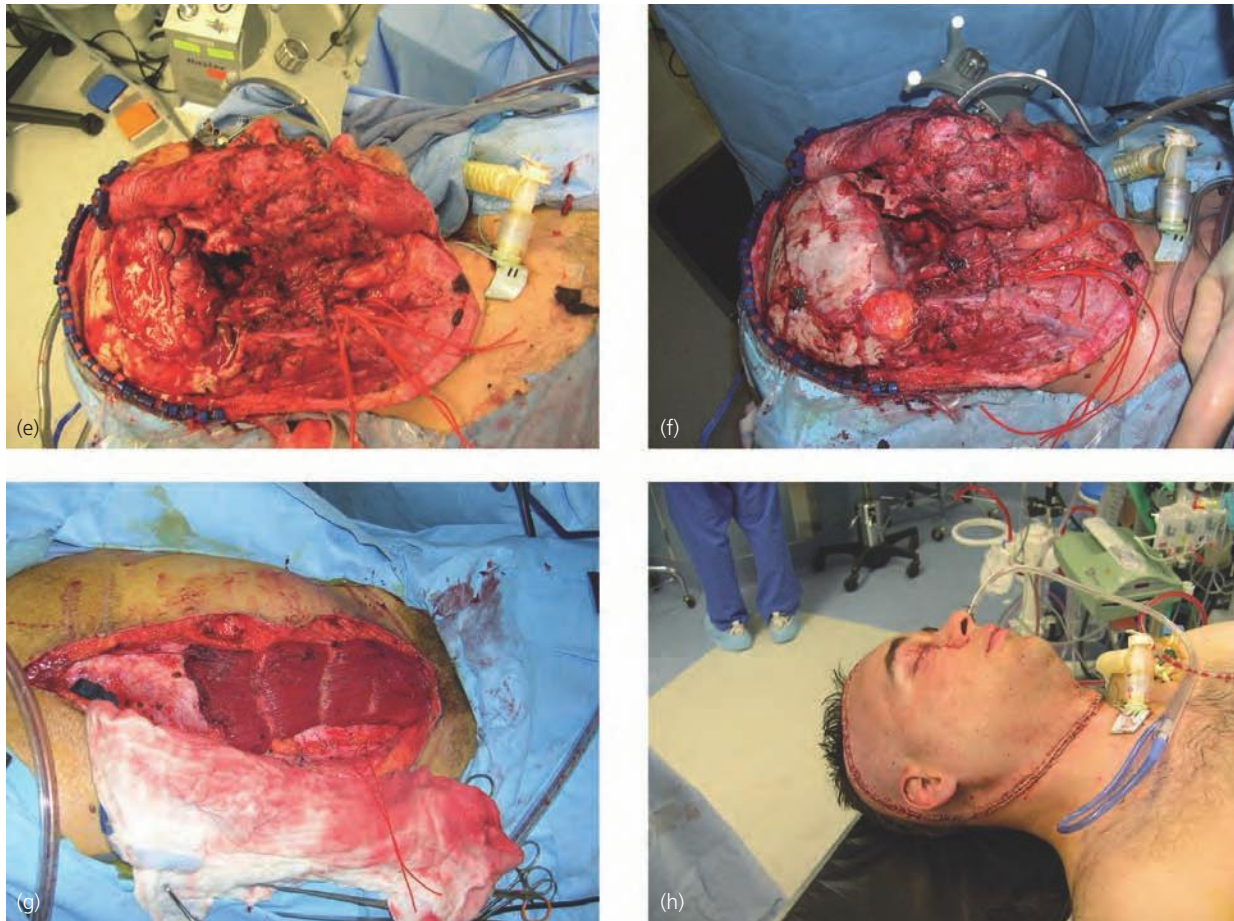


Fig. 32.43 (cont'd) (e) Intraoperative photograph of the ablative defect following craniectomy and resection of the tumor. (f) Replacement of the uninvolved temporal bone and fat graft to the mastoid defect. (g) Harvest of the rectus muscle free flap. (h) Wound closure.

margins, stage, grade, association with neurofibromatosis type 1 (NF1), and a history of previous radiation therapy.^{427–436} Complete surgical removal with negative resection margins is the most important prognostic indicator.^{427,430,434,435} Data are lacking regarding the role of adjuvant or neoadjuvant chemotherapy or radiation therapy, and these tumors have traditionally been thought to be relatively resistant to radiation and chemotherapy. Adjuvant radiation therapy has been used to assist in local control, despite the margin status at the time of surgery.^{430,434,437,438} The use of adjuvant chemotherapy in the form of ifosfamide/doxorubicin has been advocated for pediatric MPNSTs and its use in adults should be considered for patients with residual disease after initial surgery and in grade 3 tumors larger than 5 cm.^{430,434} Unresectable disease can be treated with chemotherapy in an attempt to shrink the tumor to resectable proportions, although data from this approach are lacking and prognosis remains dismal.⁴³⁴ Based on the current body of evidence, treatment recommendations are as follows: (1) complete surgical removal with as wide a margin of normal tissue as is feasible to achieve negative margins; (2) adjuvant radiation therapy and chemotherapy based on residual disease

after initial surgery, tumor size, and grade; (3) immediate reconstruction with free tissue transfer as appropriate.

Metastatic carcinoma

Metastatic carcinoma is the most common form of malignancy affecting bone. While the jaws are relatively uncommon sites for distant metastasis, bones with active marrow spaces such as vertebrae, ribs, pelvis, and skull are generally considered preferential sites for dissemination. Metastatic carcinoma has previously been thought to represent approximately 1% of all malignant jaw lesions. More recent data based on autopsied carcinoma cases, however, demonstrate that 16% of the mandibles have microscopic deposits of metastatic tumor cells, despite the lack of radiographic evidence of osteolysis. Metastatic spread of carcinoma to the jaws occurs by a hematogenous route, most commonly from the breast or lung, but also from the kidney, prostate, thyroid, colon, and rectum. Emboli of primary carcinomas may enter the venous circulation and bypass the lungs via the valveless prevertebral venous plexus of Batson to deposit in the jaws.

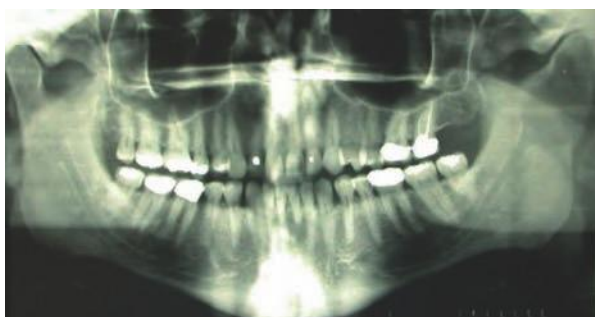


Fig. 32.44 Panoramic radiograph demonstrating bronchogenic carcinoma metastatic to the left mandibular condyle.

Metastasis to the jaws is much more common in the mandible than in the maxilla. Radiographically, a metastatic lesion appears as a poorly circumscribed radiolucency with cortical erosion, expansion and destruction of local anatomy, including tooth roots (Fig. 32.44). In the presence of known malignancy, the diagnosis is often made clinically or with confirmatory biopsy. If the primary lesion is occult (not yet identified) the histopathology and immunohistochemistry may be correlated with whole body CT and positive emission tomography (PET) to identify the site of origin.

Treatment is usually palliative and aimed at controlling symptoms and local extension into vital structures. This is commonly in the form of focused radiotherapy or systemic therapy based on the histopathologic diagnosis. Occasionally, surgery may be indicated to provide local control for radio-resistant tumors or in patients that have developed pathological fracture. The prognosis is generally poor, dependent on the tumor type, and a mean survival is generally measured in months, not years (mean 6–7 months).

Summary

Odontogenic and non-odontogenic tumors of the jaws are a relatively rare and heterogeneous group of benign and malignant neoplasms, hamartomas, and other bone-related lesions that demonstrate great variability in etiology, biologic behavior, and clinical significance. Ameloblastoma, keratinizing odontogenic tumor, giant cell lesions, fibrous dysplasia, and various sarcomas of bone are typically the most significant and commonly encountered for surgeons managing this diverse group of cellular proliferations. Treatment should be tailored for each individual patient based on tumor histology, size, and location, as well as a number of other patient-related conditions. In general, however, benign disease should be treated like benign disease and maxillomandibular continuity preserved whenever possible. Malignant tumors generally necessitate radical resection, often with adjuvant or neoadjuvant chemotherapy, radiation therapy, cervical lymphadenectomy, and immediate reconstruction with microvascular free tissue transfer.

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Chapter 33

Mucosal Lesions (Potentially Malignant Disorders of the Oral Mucosa)

Takashi Fujibayashi

The first part of this chapter will cover the concept and histology of potentially malignant disorders of the oral mucosa including the grading of mucosal dysplasia. The next section will cover potentially malignant diseases of the oral mucosa including leukoplakia, erythroplakia, keratosis of the palate, oral submucous fibrosis, and oral lichen planus. The final part will cover the early detection and management of potentially malignant disorders of the oral mucosa.

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Concept and histology of potentially malignant disorders of the oral mucosa

General concept of potentially malignant disorders of the oral mucosa

Many technical terms have been used in books or journals to describe oral premalignancies that may have a potential to transform into cancers of the oral

mucosa. These include “precancer”, “pre-malignant”, “precursor lesions”, “intraepithelial neoplasia”, and “potentially malignant”. The report from the World Health Organization (WHO) Collaborating Center¹ proposed that oral mucosal presentations recognized as precancerous can be classified in two major groups as precancerous lesions and precancerous conditions. A precancerous lesion was defined as “a morphologically altered tissue in which oral cancer is more likely to occur than in its apparently normal counterpart”. On the other hand, a precancerous condition was defined as “a generalized state associated with a significantly increased risk of cancer”. This classification was based on the concept that the origin of malignancy in the oral mucosa of a patient with a precancerous lesion would correspond with the specific local site of precancer; whereas a malignancy may arise in any part of the oral mucosa in a precancerous condition. This concept was generally accepted at the time when these terms were proposed.

Using this model, two precancerous lesions, leukoplakia and erythroplakia, were defined, and syphilis, sideropenic dysphagia, and oral submucous fibrosis were described as examples of precancerous conditions. Over a 20-year period this concept and classification had been used and quoted by many investigators and clinicians, and also discussed with regard to potential malignancy of the nominated and non-nominated oral lesions and conditions. As a result of this discussion, in 1997 the WHO modified the classification of precancerous lesions and precancerous conditions without altering the basic concept of two major groups of lesions and conditions.² Table 33.1 shows this clinical classification of precancerous lesions and precancerous conditions. Precancerous lesions include leukoplakia, erythroplakia, and palatal keratosis associated with reverse smoking, and precancerous conditions include sideropenic dysphagia, lichen planus, oral submucous fibrosis,

Table 33.1 Classification of precancerous lesions and conditions by WHO (1997).

Precancerous lesions	Precancerous conditions
Leukoplakia	Sideropenic dysphagia
Erythroplakia	Lichen planus
Palatal keratosis associated with reverse smoking	Oral submucous fibrosis
	Syphilis
	Discoid lupus erythematosus
	Xeroderma pigmentosum
	Epidermolysis bullosa

syphilis, discoid lupus erythematosus, xeroderma pigmentosum, and epidermolysis bullosa.

Many recent research reports of clinical studies on epidemiology, natural history, observation on malignant transformation rate, and investigations on morphological and cytological changes, chromosomal, genomic and molecular alterations suggest the need for some modification of the concept of precancerous lesions and conditions. It is now known that even clinically normal looking oral mucosa in patients with precancerous lesion on one side of the oral cavity may have dysplasia or molecular abnormality on the contralateral site, suggesting the possibility of malignant transformation, and that cancer could subsequently develop in the normal-appearing mucosa. A workshop coordinated by the WHO Collaborating Centre for Oral Cancer and Precancer in the UK was held in London in 2005, and the report of the workshop was subsequently published.³ The report recommended the term “potentially malignant disorders” to refer to precancer. Potentially malignant disorders of the oral mucosa are also indicators of risk of likely future malignancies elsewhere in clinically normal-appearing oral mucosa and not only site-specific predictors. Potentially malignant disorders of the oral mucosa include leukoplakia, erythroplakia, palatal lesions in reverse smokers, oral submucous fibrosis, actinic keratosis, lichen planus, discoid lupus erythematosus, and hereditary disorders with increased risk (Table 33.2).

Histology (grading of dysplasia)

Histological changes that may be observed in potentially malignant disorders of the oral mucosa can be

Table 33.2 Potentially malignant disorders of the oral mucosa agreed in an international working group coordinated by the WHO Collaborating Centre for Oral Cancer and Precancer in the UK (2005).

Potentially malignant disorders of the oral mucosa
Leukoplakia
Erythroplakia
Palatal lesions in reverse smokers
Oral submucous fibrosis
Actinic keratosis
Lichen planus
Discoid lupus erythematosus
Hereditary disorders with increased risk

expressed as oral epithelial dysplasia. Although the clinical appearance of oral epithelial dysplasia is variable, oral squamous malignancies of the lining mucosa are known generally to develop from dysplastic surface epithelium of the oral mucosa. Histological change that may contribute to a diagnosis of epithelial dysplasia was defined by the WHO as shown in Table 33.3.² The 13 items of histological change are a combination of features of both alterations of cell kinetics of the proliferative compartment and disturbed maturation toward keratinization of epithelial cells.

The grade of epithelial dysplasia can be expressed in several categories such as no dysplasia, mild dysplasia, moderate dysplasia, and severe dysplasia.^{1,2,4,5} The relationship of epithelial dysplasia in various grades to the subsequent development of cancer has not been fully clarified. However, it is generally believed that any degree of epithelial dysplasia, even a mild form, indicates an increased risk, and severe dysplasia suggests a very high risk of the subsequent development of cancer. Dysplasia is often seen coexisting with invasive carcinoma. It should be also noted that some cellular atypia, usually of a minor degree, is often present in inflammatory conditions. Severe grades of epithelial dysplasia may merge into the lesion customarily designated as carcinoma *in situ* (CIS), in which the whole, or almost the whole, thickness of squamous epithelium shows the cellular features of carcinoma without stromal invasion.²

Some attempts have been made to standardize the degree of dysplasia by providing weighted scores for the 13 microscopic features,⁴ but wide intra- and interobserver variability was encountered in grading oral epithelial dysplasia.⁵⁻⁷ Therefore, the thickness (height) of the cellular change was taken into account. In mild forms of dysplasia, recognizable changes are limited to the lower third of the epithelium; in moderate dysplasia they extend to the middle third; and in severe dysplasia, the changes extend to the upper layers.⁸ However, it was reported that the variation in the grading of dysplasia was still present even using

Table 33.3 Histological changes that may contribute to a diagnosis of epithelial dysplasia (WHO 1997).

Loss of polarity of the basal cells
The presence of more than one layer having a basaloid appearance
Increased nuclear–cytoplasmic ratio
Drop-shaped rete ridges
Irregular epithelial stratification
Increased number of mitotic figures
Mitotic figures that are abnormal in form
The presence of mitotic figures in the superficial half of the epithelium
Cellular and nuclear pleomorphism
Nuclear hyperchromatism
Enlarged nucleoli
Loss of intercellular adherence
Keratinization of single cells or cell groups in the prickle cell layer

the concept of depth of dysplasia.^{9,10} Knowledge of clinical information did not increase agreement rates between examiners.¹¹ Inflammation in the lesions further decreased agreement rates.¹²

In 2005 the WHO revised the criteria used for diagnosing oral epithelial dysplasia in epithelial precursor lesions from 13 to 16 items by classifying seven architectural and nine cytological criteria as shown in Table 33.4.¹³ They encouraged the use of a combination of architectural and cytological changes with more defined consideration of levels of change within the epithelium. When architectural disturbance is accompanied by cytological atypia, the term dysplasia applies.

Although dysplasia represents a spectrum and no criteria exist to divide this spectrum precisely into several grades, grading of oral epithelial dysplasia is still conventionally used.^{13,14} In this system, grade 1 (mild dysplasia) demonstrates general architectural disturbance limited to the lower third of the epithelium accompanied by cytological atypia (Fig. 33.1). Grade 2 (moderate dysplasia) demonstrates architec-

tural disturbance extending into the middle third of the epithelium (Fig. 33.2). Grade 3 (severe dysplasia) starts with greater than two thirds of the epithelium showing architectural disturbance with associated cytological atypia. However, architectural disturbance extending into the middle third of the epithelium with sufficient cytological atypia is upgraded from moderate to severe dysplasia (Fig. 33.3).

The term carcinoma *in situ* (CIS) denotes that malignant transformation has occurred but invasion is not present. It is not possible to recognize this morphologically. The following is recommended for the diagnosis of CIS: full-thickness or almost full-thickness architectural abnormalities in the viable cellular layers accompanied by pronounced cytological atypia (Fig. 33.4). Atypical mitotic figures, abnormal superficial mitoses, increased nuclear size, and anisonucleosis are often seen in CIS (Fig. 33.5).

Although the revised criteria of oral epithelial dysplasia of WHO (Table 33.4) describe accurate histological characters of the lesion, interobserver variability still occurs.¹⁵ Recently some reports

Table 33.4 Criteria used for diagnosing dysplasia (WHO 2005).

Architecture	Cytology
Irregular epithelial stratification	Abnormal variation in nuclear size (anisonucleosis)
Loss of polarity of basal cells	Abnormal variation in nuclear shape (nuclear pleomorphism)
Drop-shaped rete ridges	Abnormal variation in cell size (anisocytosis)
Increased number of mitotic figures	Abnormal variation in cell shape (cellular pleomorphism)
Abnormally superficial mitoses	Increased nuclear–cytoplasmic ratio
Premature keratinization in single cells (dyskeratosis)	Increased nuclear size
Keratin pearls within rete pegs	Atypical mitotic figures
	Increased number and size of nucleoli
	Hyperchromasia

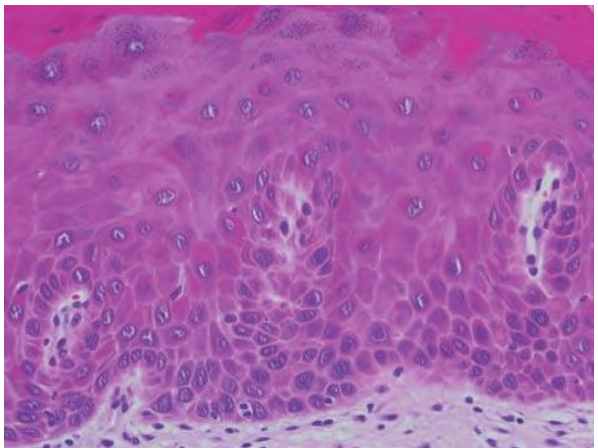


Fig. 33.1 Mild dysplasia. There is hyperplasia of basal and parabasal cells with weak loss of polarity showing slightly increased nuclear size and increased nuclear:cytoplasmic ratio. The architectural changes are limited to the lower third of the epithelium.

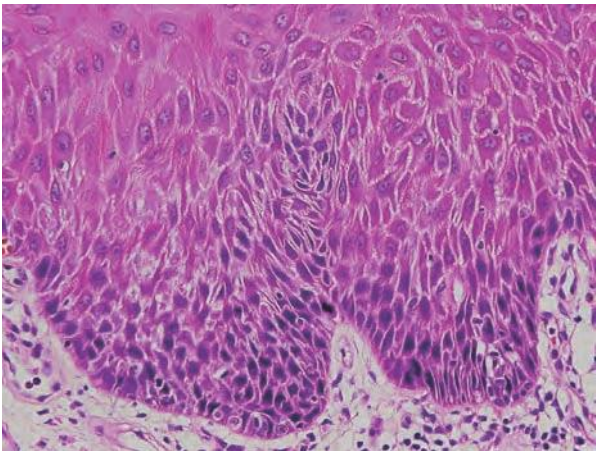


Fig. 33.2 Moderate dysplasia. The epithelial cells demonstrate variation in nuclear size and shape with increased number and size of nucleoli with some disturbance in epithelial stratification and polarity of basal and parabasal cells. The architectural changes extend into the middle third of the epithelium.

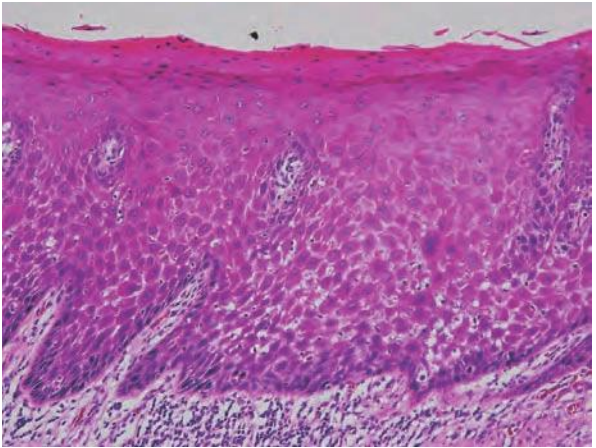


Fig. 33.3 Severe dysplasia. The architectural disturbance with cytological atypia including loss of intercellular adhesion extends to more than two thirds of the epithelium.

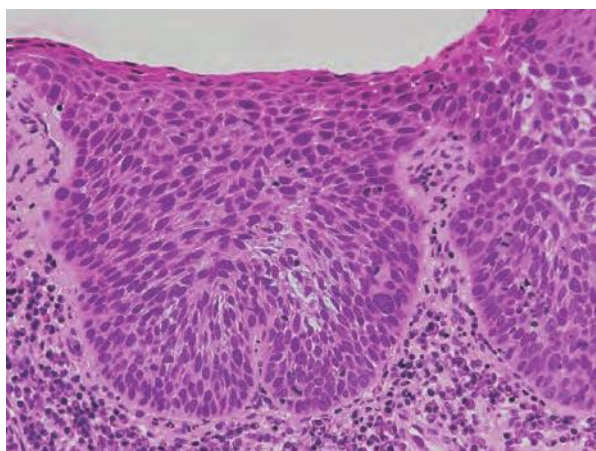


Fig. 33.4 Carcinoma *in situ* (CIS). The full thickness of the epithelium shows architectural disturbances such as irregular epithelial stratification, irregular polarity of basal cells, drop-shaped rete ridges with cytological atypia of anisonucleosis, nuclear and cellular pleomorphism, and atypical mitotic figures.

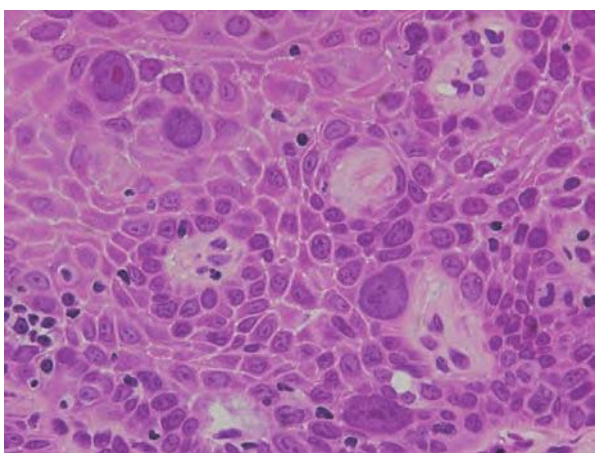


Fig. 33.5 Carcinoma *in situ* (CIS). The epithelial cells demonstrate increased nuclear size, nuclear pleomorphism, and increased size of nucleoli.

demonstrated superiority of a new binary system of grading for predicting malignant transformation,¹⁶ in which the grade of dysplasia was classified into two categories: “low risk” with no dysplasia or mild dysplasia and “high risk” with moderate dysplasia or severe dysplasia, and this concept was supported by the working group.¹⁷ Some pathologists suggested the use of the term squamous intraepithelial neoplasia (SIN) which was originally used for the uterine cervical premalignant lesions, or variations on this such as oral intraepithelial neoplasia (OIN).¹⁸ This terminology is based on the concept that many of the potentially malignant disorders of oral mucosa are committed on a path to malignancy. According to the SIN system, mild dysplasia is regarded as SIN 1, moderate dysplasia as SIN 2, and both severe dysplasia and CIS as SIN 3.¹³ However, the SIN concept that all levels of dysplasia are actually different levels of neoplasia even if prior to invasion, is disputed.^{14,17} The histological findings of dysplasia therefore indicate no more than a lesion has a statistically increased

risk of malignant change; they cannot be used for confident prediction of malignant change in any individual case.¹⁹

Potential molecular biological markers or molecular genetics of potentially malignant disorders of the oral mucosa which may have special association with progression of oral epithelial dysplasia to carcinoma, have been studied.^{19–22} Loss of heterozygosity (LOH) at 3p and 9p increased the risk of progression of dysplasia to squamous cell carcinoma (SCC). Mutation of p53 tumor suppressor gene is one of the most common genetic events in carcinogenesis. Although LOH at the p53 loci appears to be associated with progression, mutation of the p53 gene is rare in dysplasia and appears to be a late event. The p53 protein and other markers of apoptosis appear in basal and parabasal layers of dysplastic lesions that progressed to SCC. The measurement of nuclear DNA (DNA ploidy) may have a prognostic value. While some studies found an association of aneuploid pattern with progression to SCC, others have found no such relationship. Matrix metalloproteinases (MMP)-1 and -9 were more commonly expressed in dysplastic tissue that progressed to SCC. Cytokeratin-4 (K4), K13, transglutaminase 3 (TG-3) were suppressed and expression of K14 and K17 was elevated in SCC and severe dysplasia. Epidermal growth factor receptor shows a linear increase in intensity of histochemical staining, suggesting an increase in dysregulation of epithelial cell proliferation with increasing degree of dysplasia. However, to date, no single definitive molecular marker to predict the progression of potentially malignant disorders of the oral mucosa to SCC has been identified.

Potentially malignant disorders of the oral mucosa

Leukoplakia

The most common and important potentially malignant disorder of the oral mucosa is leukoplakia. WHO¹ first defined leukoplakia as “a white patch or plaque that cannot be characterized clinically or pathologically as any other disease” and as a main part of oral precancerous lesions, which means it has an increased risk of malignant potential. This concept has been adapted and refined worldwide through several international seminars.^{2,13,23,24} The latest report of the working group³ further refined the description as: “The term leukoplakia should be used to recognize white plaques of questionable risk having excluded (other) known diseases or disorders that carry no increased risk for cancer.”

Leukoplakia must be differentially diagnosed from other known predominantly white lesions or disorders such as white sponge nevus, frictional keratosis, moscicatio buccarum, chemical injury, acute pseudomembranous candidosis, leukoedema, lichen

planus (plaque type), lichenoid reaction, discoid lupus erythematosus, skin graft, hairy leukoplakia, and leukokeratosis nicotina palate. Leukoplakia is a clinical term. At clinical examination a provisional diagnosis of leukoplakia is made if a predominantly white lesion cannot be clearly diagnosed as any of these other diseases. A biopsy is mandatory to exclude any of the other named diseases. A definitive diagnosis of leukoplakia can be made when any etiological cause, such as trauma, biting, chemicals other than tobacco/areca nut use, has been excluded and histopathology has not confirmed any other specific disorder. However, leukoplakia itself has no specific histology, it may show atrophy or hyperplasia (acanthosis), and may or may not demonstrate epithelial dysplasia.

Oral leukoplakia occurs most frequently on the lip vermillion, buccal mucosa, lateral border of tongue, floor of mouth, and gingival mucosa. The clinical finding of leukoplakia has been reported using various terms, but classification into two types is generally accepted: homogeneous and non-homogeneous leukoplakia.² The classification is based on surface color and morphologic characteristics. Homogeneous leukoplakias look uniformly flat and thin, and may

exhibit shallow cracks of the surface keratin (Figs 33.6 and 33.7). Non-homogeneous leukoplakias include several subtypes:

- speckled type – mixed appearance of white and red, but still retaining predominantly white character (Figs 33.8 and 33.9);
- nodular type – small polypoid outgrowths with rounded red or white excrescences;
- verrucous type – wrinkled or corrugated surface appearance.

In general terms, non-homogeneous leukoplakias present a greater risk of malignant change than the homogeneous types. However, the latest working group³ reported that these two categories were imprecise and of limited value. However, they also reported that it should be recognized that non-homogeneous type may include the following two distinct groups which have a higher risk of malignancy.

- Erythroleukoplakia is one type of non-homogeneous leukoplakias. It is very similar to speckled type leukoplakia and appears white and red, with red areas of minimal keratin production inter-

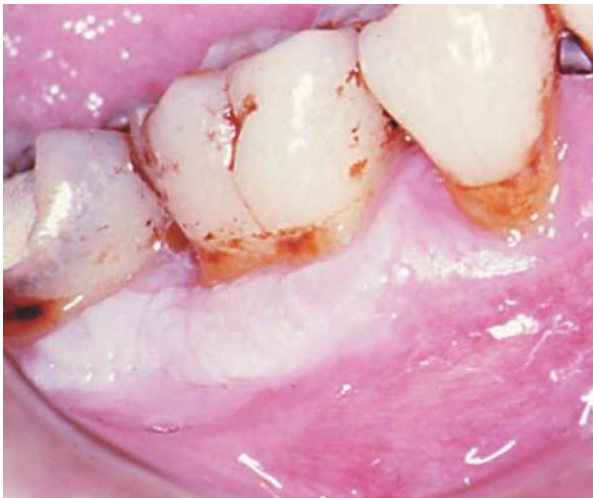


Fig. 33.6 Homogeneous leukoplakia in the lower gingival mucosa.



Fig. 33.8 Non-homogeneous leukoplakia (speckled type) on the lateral to ventral surface of the tongue.

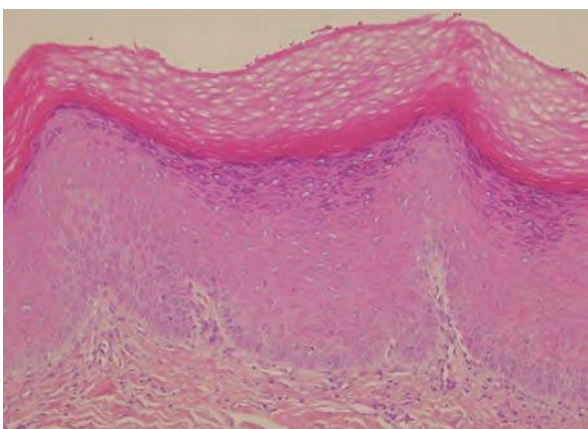


Fig. 33.7 Histopathologic feature of homogeneous leukoplakia showing hyperorthokeratosis and acanthosis with no dysplasia.

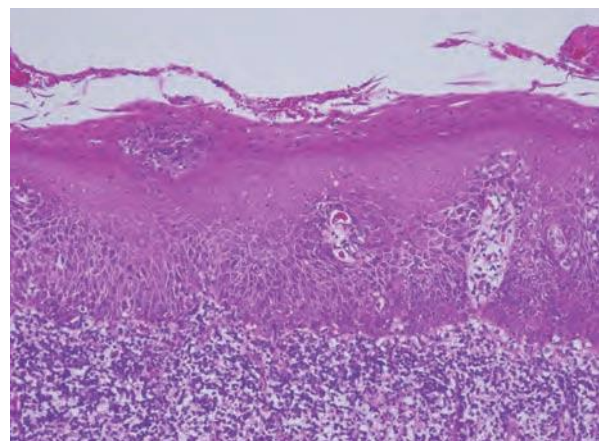


Fig. 33.9 Histopathology of speckled type leukoplakia showing moderate epithelial dysplasia and severe round cell infiltration in the subepithelial connective tissue.

persed among the background of white areas with thickened keratin. The red areas of erythro-leukoplakia are larger than in speckled type leukoplakia, and erythroleukoplakia shows a rather thin epithelial layer with some dysplasia (Figs 33.10 and 33.11). An alternative term for this is erosive leukoplakia.

- Proliferative verrucous leukoplakia (PVL) is another type of oral leukoplakia. PVL develops nodular, papillary or verrucous surface projections (Figs 33.12 and 33.13), which gradually spread laterally to encompass large areas of oral mucosa. Sometimes it presents at multifocal sites in the same oral cavity.^{25–27} PVL occurs predominantly (74%) in females, unlike other leukoplakia, and 63% of patients did not use tobacco products.²⁸ In histology almost half of all PVL patients demonstrate epithelial dysplasia and more than 70% of them will develop SCC later.^{27–30} PVL is an aggressive form of oral leukoplakia with considerable morbidity and strong prediction for malignant transformation. Conservative management of these lesions has been unsuccessful and wide surgical excision offers the best hope for control.³¹

Malignant transformation rates from oral leukoplakia reported in the literature vary greatly from study to study.^{20,32} Table 33.5 shows a summary of major reports on this topic from various countries.^{25,33–45} In the large-scale community-based surveys in India, estimates of the malignant transformation rate range from 0.13–0.9%.^{33–35} In contrast, reports from hospital-based sample studies derived from patients referred to selected special units by primary health care workers show much higher rates, ranging widely between 1.8 and 17.5%.^{25,36–45} A study of pooled estimates reports that the prevalence rate of oral leukoplakia is 2.60% in the world population and that a global malignant transformation rate of oral leukoplakia is 1.36% per year.⁴⁶ In a general review performed by the latest workshop group³² for predicting malignant transformation in oral leukoplakia, older patients, particularly females, are more at risk than younger patients; the duration of the disorder may also be important. Those who have never used tobacco seem at greater risk than smokers. Tongue, floor of mouth, and retromolar/soft palate complex are more at risk. The non-homogeneous type develops malignancy far more frequently than the homogeneous



Fig. 33.10 Erythroleukoplakia on the lateral surface of the tongue. The red area is larger than in speckled type leukoplakia.



Fig. 33.12 Proliferative verrucous leukoplakia (PVL) on the buccal mucosa with papillary and verrucous surface projection in female who does not use tobacco. This patient later developed squamous cell carcinoma.

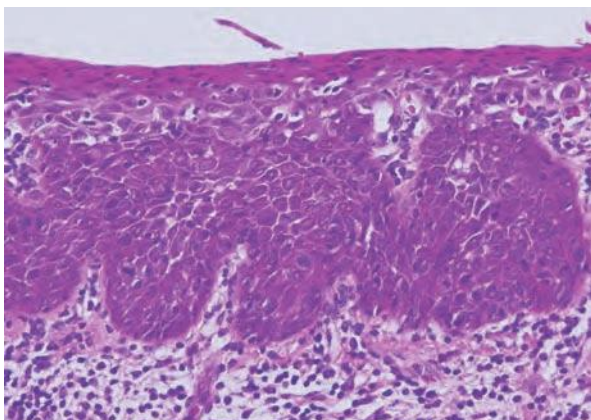


Fig. 33.11 Histopathologic findings of erythroleukoplakia showing decreased parakeratotic layer with severe dysplasia. The architectural disturbance with cytological atypia extends to more than two thirds of the epithelium.

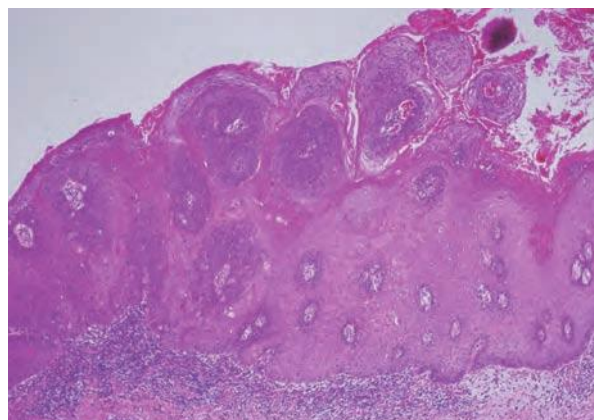


Fig. 33.13 Histopathologic findings of proliferative verrucous leukoplakia (PVL) showing papillary exophytic proliferation of squamous epithelium with little dysplasia but no stromal invasion.

Table 33.5 Selected studies on malignant transformation of oral leukoplakia.

Author(s)	Year reported	Country where investigation was performed	Patients with leukoplakia	Observation period (years)	Malignancy developed patients/leukoplakia patients	Malignant transformation rate (%)	Reference
Silverman <i>et al.</i> ³³	1976	India	4762	2	6/4762	0.13	33
Gupta <i>et al.</i> ³⁴	1980	India	5345	10	24/5345	0.45	34
Mehta <i>et al.</i> ³⁵	1972	India	117	10	1/117	0.9	35
Pindborg <i>et al.</i> ³⁶	1968	Denmark	214	1–5	4/214	1.8	36
Roed-Peterson ³⁷	1971	Denmark	331	4.3	12/331	3.6	37
Kramer <i>et al.</i> ³⁸	1970	UK	187	1–16	9/187	4.8	38
Bánóczy ³⁹	1977	Hungary	670	9.8 (1–30)	40/670	6.0	39
Silverman and Rosen ⁴⁰	1968	USA	105	1–11	7/105	6.7	40
Holmstrup <i>et al.</i> ⁴¹	2006	Denmark	236	6 (1–20)	17/254*	6.7	41
Amagasa <i>et al.</i> ⁴²	2006	Japan	444	1–29	35/444	7.9	42
Lind ⁴³	1987	Norway	157	6–16	14/157	8.9	43
Gangadharan and Paymaster ⁴⁴	1971	India	626	0.25–23	63/626	10.1	44
Schepman <i>et al.</i> ⁴⁵	1998	Netherlands	166	2.6 (0.5–16.8)	20/166	12.0	45
Silverman <i>et al.</i> ²⁵	1984	USA	257	7.2 (0.5–39)	45/257	17.5	25

* Shown by number of lesions (carcinoma developed lesions/lesions of leukoplakia).

type. Large lesions covering several intraoral sites also appear more at risk.

Erythroplakia

Oral erythroplakia is defined as “a fiery red patch that cannot be characterized clinically or pathologically as any other definable lesion”.² The workshop group of 2005³ did not make any significant change to this definition. Erythroplakia is often flat with a smooth or granular surface, and numerous other red patches and/or macules that could arise on the oral mucosa should be excluded before considering erythroplakia. The differential diagnosis must include inflammatory and/or immune disorders such as desquamative gingivitis, erythematous lichen planus, discoid lupus erythematosus, pemphigoids, hypersensitivity reactions, Reiter’s disease, infections such as erythematous candidiasis and histoplasmosis, and hamartoma/neoplasms such as hemangiomas and Kaposi’s sarcoma.⁴⁷

Oral erythroplakia is a rather rare disease and the prevalence rate in the general population is in the range 0.02–0.83%.⁴⁷ The soft palate, the floor of the mouth, ventral surface of the tongue (Fig. 33.14), and the buccal mucosa in the middle-aged and the elderly are most commonly affected. The etiology and pathogenesis of oral erythroplakia are poorly understood, but it was suggested that tobacco and alcohol use are probably involved in most cases.

Erythroplakia is a clinical term, just like in leukoplakia, and it does not have any specific histopatho-

logical connotation. It may show superficially invasive carcinoma, CIS, severe dysplasia, or moderate to mild dysplasia (Fig. 33.15). Oral erythroplakia has the highest risk of malignant transformation compared to all other oral potentially malignant disorders – these red lesions often show invasive carcinoma and CIS or severe dysplasia.⁴⁸ Because of the limited number of clinical cases of oral erythroplakia and the fact that it is usually treated soon after diagnosis, the true malignant transformation rate of pure erythroplakia is unknown. Transformation rates of oral lesions histologically diagnosed as CIS or severe dysplasia, including cases of erythroplakia but also leukoplakia and erythroplakia, vary from 14–50%.⁴⁷



Fig. 33.14 Erythroplakia on the ventral surface of the tongue showing red, smooth, flat patch.

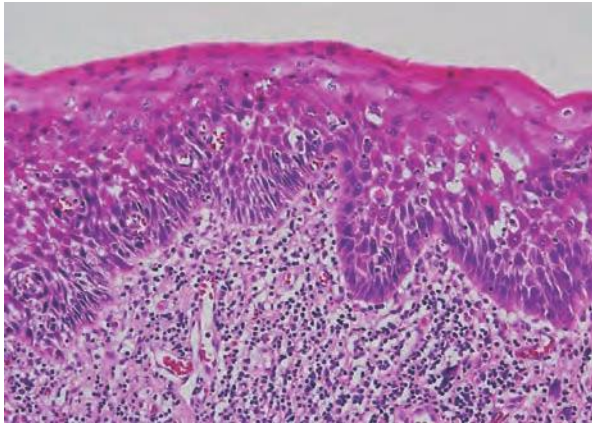


Fig. 33.15 Histopathologic findings of erythroplakia with severe dysplasia. The architectural disturbances include decreased epithelial layer, irregular epithelial stratification, irregular polarity of basal and parabasal cells with loss of intercellular adhesion and some cellular atypia.

Palatal lesions in reverse smokers

This disorder is observed in certain rural populations in the Indian subcontinent, New Guinea, and Amazon who smoke with the lighted end of homemade cigarettes and cigars inside the mouth. This habit leads to red, white or mixed lesions with very thick keratin build-up on the surface of the hard palate. This reverse smoking is linked to a very high rate of malignant transformation of the affected palatal mucosa where carcinoma seldom otherwise develops.³⁴

Oral submucous fibrosis

Oral submucous fibrosis (OSF) is a chronic disorder of the upper digestive tract including the oral cavity, oropharynx, and often the upper third of the esophagus, characterized by fibrosis of the mucosa. Clinical presentations in early forms include a burning sensation with exacerbation by spicy food, vesiculation or ulceration of oral mucosa, blanching of mucosa, and “leathery” mucosa which shows thickened, firm tissue with a wrinkled surface. Late forms present fibrous bands within the mucosa, limitation of mouth opening, difficulties with mastication and phonation, narrowing of the oropharyngeal orifice with distortion of the uvula, and “woody” changes to the mucosa and tongue.³ In histopathology, OSF shows epithelial atrophy, with metaplasia of non-keratinized areas to para- or ortho-keratinization, with various degrees of dysplasia. Thickening of the basement membrane is common, and there is marked reduction in vascularity of the connective tissues in inverse proportion to increased density of collagen, which appears hyalinized.⁴⁹

OSF has been reported mostly among subjects living in the Indian subcontinent, and neighboring Asian countries, who have a habit of chewing areca nut.⁵⁰ A clear dose-dependent relationship was observed for both frequency and duration of chewing areca nut in the development of OSF. The commer-

cially available freeze-dried products such as pan masala, guthka, and mawa (areca and lime) have high concentrates of areca nut per chew and appear to cause OSF more rapidly than self-prepared conventional betel quid.⁵¹ Areca alkaloids, such as arecoline and its active metabolite arecaidine, cause fibroblast proliferation and increase collagen synthesis. In addition, tannin present in areca nut reduces degradation of collagen by inhibiting collagenases; and the copper content in areca nut is high and copper is known to stimulate fibroblast proliferation. The areca nut may also induce the development of OSF by increased levels of cytokines such as interleukin (IL)-1, IL-6, transforming growth factor (TGF)-beta, platelet derived growth factor (PDGF), and basic fibroblast growth factor (bFGF) in the lamina propria. Current evidence implicates collagen-related genes in susceptibility and pathogenesis of OSF.⁵¹

Epithelial dysplasia in OSF tissues appeared to vary from 7–26% depending on the population. Malignant transformation rate of OSF to oral cancer was reported as 7.6% by a long-term follow-up study of 17 years in India.⁵²

Oral lichen planus

Oral lichen planus (OLP) is one of the most prevalent diseases of the oral mucosa and can be found in every country of the world. Diagnosis of OLP should be based on both clinical features and histopathological findings that meet the diagnostic criteria of OLP.^{53,54} The most characteristic clinical feature of OLP is the presence of white striations and/or papules which often appear in bilateral areas of the oral mucosa (Fig. 33.16). These bilateral, often symmetrical reticular lesions are strong determining components of the clinical profile of OLP. In addition to the white lesions, erythematous lesions with plaque-type, atrophic, ulcerative or sometimes bullous appearance may also be observed during the course of the disease, but these red lesions are not OLP specific and



Fig. 33.16 Typical clinical feature of oral lichen planus (OLP) showing white striations with linear, reticular, and annular shapes in light erythematous or erosive background. This patient has bilateral symmetrical lesions.

can also be seen in other similar disorders. Essential histological criteria for diagnosing OLP are a band-like dense lymphoid cell infiltrate confined to subepithelial connective tissue and liquefaction degeneration of the epithelial basal layer. The epithelium may develop hyperkeratosis, atrophy, hyperplasia, acanthosis, and saw-toothed rete ridges (Fig. 33.17). There have been many discussions concerning atypical cytomorphology and dysplastic epithelial structure of the lesion clinically considered as OLP.^{55–60} The consensus has been that the histopathological diagnosis of OLP should be applied in the absence of epithelial dysplasia.

The criteria for diagnosing malignant transformation of OLP proposed by Krutchkoff *et al.*⁶¹ have also been generally agreed (Table 33.6). This has been confused by another discussion of differential diagnosis between true OLP (idiopathic OLP) and similar so-called oral lichenoid lesions (OLL)⁵⁷ which includes such lesions as oral lichenoid drug reactions (OLDR), oral lichenoid contact lesions (OLCL), oral lichenoid lesions in graft-versus-host disease (OLL-GVHD) (Figs 33.18, 33.19),⁶² and amalgam-associated oral lichenoid reactions (AAOLR).⁶³ Table 33.7 shows differential diagnostic criteria between OLP and OLL proposed by van der Meij and van der Waal.⁵⁷ In these criteria, absence of epithelial dysplasia is confirmed to diagnose OLP, and if either clinical or histopathological features are not obvious as OLP, the term OLL will be used.

Table 33.8 shows a summary of papers reporting malignant transformation rate in OLP from 1985 to the present, where the study involved more than 500 OLP patients.^{64–73} The malignant transformation rate varies from 0.4–6.4% in the observed period and 0.07–0.74% per year. Patients with OLL may have an increased risk of developing malignancies compared with patients with OLP.^{59,60} Some of the reports listed in Table 33.8 may include OLL patients among patients reported as OLP when the differential diagnosis of OLP and OLL had not been well discussed.

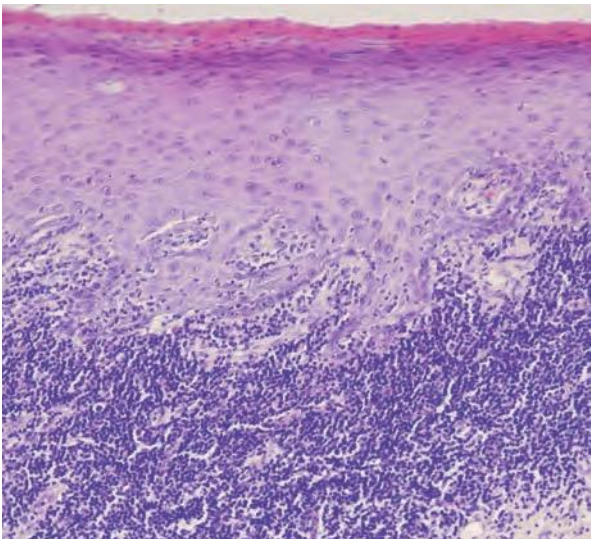


Fig. 33.17 Histopathologic findings of oral lichen planus (OLP). The dense band-like zonal cellular infiltration of lymphocytes with some macrophages is evident just under the epithelial tissue. The epithelium shows acanthosis with a little hyperparakeratosis, liquefaction degeneration in the basal cell layer, and some saw-tooth rete ridges, but no dysplasia.



Fig. 33.18 Oral lichenoid lesion in graft-versus-host disease (OLL-GVHD) in a bone marrow transplanted patient. Clinical features are quite similar to those of OLP.

Table 33.6 Criteria for diagnosing as malignant transformation of oral lichen planus (OLP) (from Krutchkoff *et al.* 1978).⁶¹

Original diagnosis
The diagnosis of OLP must have been properly verified with acceptable histopathologic evidence demonstrating at least two of the following four features: 1. Hyperkeratosis or parakeratosis (this is not necessary in the case of erosive OLP) 2. Saw-toothed rete pegs 3. Superficial infiltrate of lymphocytes 4. Basal cell liquefaction
History and follow-up
1. Clinical and historical features of alleged transformations must been adequately described. Information such as age, sex, precise location, and clinical description of the lesion are necessary 2. Reported transformations should have had proper follow-up (at least 2 years), with all changes in clinical features properly recorded
Tobacco exposure
Tobacco habits should have been properly documented to help distinguish between true malignant transformations and conventional carcinomas occurring in the mouths of patients who happen to have OLP

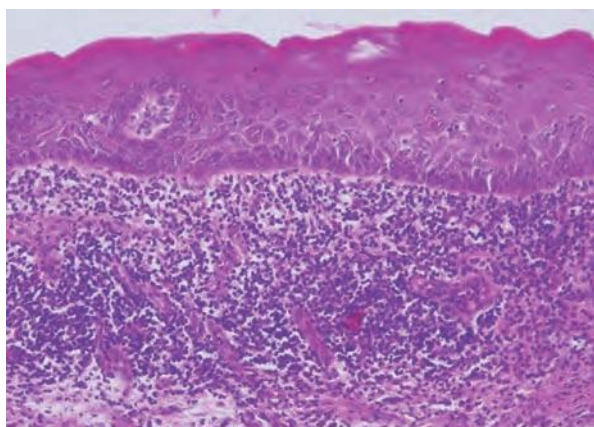


Fig. 33.19 Histopathologic findings of oral lichenoid lesion in graft-versus-host disease (OLL-GVHD). Dense lymphocyte infiltration with small number of granulocytes is observed from upper to lower connective tissues. The epithelium has flat rete ridges with slight dysplasia. These findings are histopathologically compatible with OLP. Histopathology alone cannot reliably distinguish between OLP and OLL.

Table 33.7 The differential diagnostic criteria between oral lichen planus (OLP) and oral lichenoid lesions (OLL) proposed by van der Meij and van der Waal (2003).⁵⁷

Clinical criteria

- Presence of bilateral, more or less symmetrical lesions
- Presence of a lace-like network of slightly raised gray-white lines (reticular pattern)
- Erosive, atrophic, bulbous and plaque-type lesions are only accepted as a subtype in the presence of reticular lesions elsewhere in the oral mucosa

In all other lesions that resemble OLP but do not complete the aforementioned criteria the term “clinically compatible with” should be used

Histopathological criteria

- Presence of a well-defined band-like zone of cellular infiltration that is confined to the superficial part of the connective tissue, consisting mainly of lymphocytes
- Signs of “liquefaction degeneration” in the basal cell layer
- Absence of epithelial dysplasia

When the histopathological features are less obvious, the term “histopathologically compatible with” should be used

Final diagnosis OLP or OLL

To achieve a final diagnosis clinical as well as histopathological criteria should be included

OLP: A diagnosis of OLP requires fulfillment of both clinical and histopathological criteria

OLL: The term OLL will be used under the following conditions:

1. Clinically typical of OLP but histopathologically only “compatible with” OLP,
2. Histopathologically typical of OLP but clinically only “compatible with” OLP,
3. Clinically “compatible with” OLP and histopathologically “compatible with” OLP

Table 33.8 Reported malignant transformation rate in oral lichen planus patients from 1985 to the present (studies involving more than 500 OLP patients).

Author(s)	Year reported	Country where investigation was performed	Patients with OLP	Mean follow-up period (years)	Malignancy developed patients/OLP patients	Malignant transformation rate (%)	Malignant transformation rate per year (%)	Reference
Silverman <i>et al.</i> ⁶⁴	1985	USA	570	5.6	7/570	1.2	0.22	64
Murti <i>et al.</i> ⁶⁵	1986	India	722	5.1	3/702	0.4	0.08	65
Holmstrup <i>et al.</i> ⁶⁶	1988	Denmark	611	7.5	9/611	1.5	0.20	66
Rajenthiran <i>et al.</i> ⁶⁷	1999	UK	832	11.0	7/832	0.8	0.07	67
Mignogna <i>et al.</i> ⁶⁸	2001	Italy	502	5.0	18/496	3.7	0.74	68
Eisen ⁶⁹	2002	USA	723	4.5	6/723	0.8	0.18	69
Rödström <i>et al.</i> ⁷⁰	2004	Sweden	1028	6.8	5/1028	0.5	0.07	70
Xue <i>et al.</i> ⁷¹	2005	China	674	Unknown	4/674	0.6	Unknown	71
Ingafou <i>et al.</i> ⁷²	2006	UK	690	7.0	13/690	1.9	0.27	72
Mignogna <i>et al.</i> ⁷³	2007	Italy	700	16.0	45/700	6.4	0.40	73

However, the report by Mignogna *et al.*⁷³ showing the highest rate of malignant transformation did not include OLL patients. Therefore, the malignant potential of OLP still remains controversial. WHO³¹ recommends that until distinct clinical and histopathological criteria have been developed on how to differentiate OLP from OLL, both lesions have to be considered as at risk for malignant transformation. The report of the 4th World Workshop on Oral Medicine (WWOM)⁶² also concluded that there is no consensus about the possible premalignant character of OLP at present; nevertheless, monitoring is recommended at least annually.

Others

Actinic keratosis of the lip is considered to be a potentially malignant condition. The squamous epithelium of the vermilion of the lip may be atrophic or hyperplastic with disordered maturation showing varying degrees of keratinization, atypia, and increased mitosis.

Discoid lupus erythematosus (DLE) sometimes shows very similar features to OLP. Clinical distinction of DLE from OLP and erythroplakia can be difficult. Malignant transformation has been reported when DLE affects the lip.

Hereditary disorders with increased risk are dyskeratosis congenita and epidermolysis bullosa. Dyskeratosis congenita (DC) is an inherited bone marrow failure syndrome that is characterized by lacey reticular hyperpigmentation of the skin, dystrophic nails, mucous membrane hyperkeratosis, and pancytopenia.⁷⁴ Oral white plaques in DC may be confused with leukoplakia. Development of SCC

from the lesion on the dorsum of the tongue at a very young age has been reported with a rather high incidence in DC patients.

Epidermolysis bullosa is a rare genetic disease characterized by cutaneous and mucosal blistering associated with subsequent scarring in response to minor trauma. Oral hyperkeratosis and SCC have been reported in association with epidermolysis bullosa.

Early detection and management of potentially malignant disorders of the oral mucosa

Early detection and diagnosis

The potentially malignant disorders (PMD) of the oral mucosa can arise not only from preceding lesions but also from any part of normal-looking mucosa^{3,75} and most PMD must be confirmed by histopathological diagnosis. The clinical diagnosis confirmed by histopathological diagnosis by scalpel biopsy is the gold standard for any PMD. Several diagnostic aids and adjunctive techniques for screening of patients for PMD and oral cancers have been developed recently.⁷⁶ Table 33.9 gives a summary of these examination methods and adjunctive techniques.

Conventional oral examination

A conventional oral examination under normal light by inspection and palpation is the standard method for screening PMD or oral cancers. The diagnostic ability of examination methods can be evaluated in

Table 33.9 Examination methods and adjunctives of diagnosing potentially malignant disorders or cancers of the oral mucosa.

Examination methods and adjunctive techniques	Material	Normal mucosa	Dysplasia (atypia)
Conventional oral examination	Inspection and palpation	Normal looking	Some abnormality
Vital staining:			
Toluidine blue	Tolonium chloride	No staining	Pale or dark royal blue
Iodine	Iodine glycerol or Lugol's solution	Dark (parakeratotic cell)	No staining
Tissue reflectance light visualization:			
ViziLite Plus®	Chemiluminescent light	Light bluish	Reflect and shine white
MicroLux DL	Battery-powered light	Light bluish	Reflect and shine white
Fluorescence imaging and spectroscopy:			
VELscope®	Handpiece-emitted blue light	Fluorescent glow	Loss of fluorescence
Brush cytology:			
OralCDx®	Cytology by brush biopsy	Negative (normal)	Atypical (abnormal), Positive (dysplasia/ca)
Scalpel biopsy:			
Incisional	Incisional biopsy for diagnosis	Normal tissue	Dysplasia or cancer
Excisional	Diagnosis and treatment		

terms of sensitivity, specificity, positive predictive value, negative predictive value, and accuracy of diagnostic examination as shown in Table 33.10. Conventional oral examination is performed without using any specific device, but the detection of PMD depends on the skill of each examiner which differs with experience and knowledge. A review of this subject showed an overall sensitivity of 85% and specificity of 97% in conventional oral examination by general dentists when compared with the result of examination by an oral medicine specialist considered as a “true” clinical diagnosis.⁷⁷ This indicates the ability of an oral examination as a screening test in detecting lesions of the oral mucosa but does not necessarily mean making the correct diagnosis of the lesion.

Vital staining

Toluidine blue is a vital staining dye, which is also known as tolonium chloride, and it may stain nucleic acids and some abnormal tissues. The nuclear uptake of the dye in malignant lesions is consistent with the high density of nuclear material in neoplastic cells which shows a dark royal blue color, whereas non-malignant lesions such as inflammatory granulation tissues may show pale blue.⁷⁸ Toluidine blue is commercially available as a ready-to-use kit (OraScan®) comprising 1% toluidine blue as the staining rinse and 1% acetic acid for use as both pre- and postrinses. The efficacy of using this product was reported as high (100% in sensitivity) in detecting oral cancers but rather low (62% in specificity) in discriminating benign lesions.⁷⁹ The overall sensitivity and specificity of the test for detecting PMD (dysplasia) was only 74% and 66%, respectively. Therefore, toluidine blue appears to be good at detecting cancers but is positive in only two thirds of lesions with dysplasia. In addition, it may stain common, benign conditions such as non-specific ulcers. TBlue^{630®} is another product containing 0.5% toluidine blue in the swab which is used in combination with ViziLite® Plus.

Iodine vital staining can be also used for detecting PMD or oral cancers by applying Lugol’s iodine solution or iodine glycerol solution. Iodine can bind cytoplasmic glycogen of the oral mucosal epithelium,

and normal oral mucosa with parakeratosis will stain dark brown, but dysplastic or cancer cells will be poorly stained or unstained by loss of glycogen in their cytoplasm. The sensitivity and specificity in detecting oral dysplastic or malignant lesions was reported as 87.5% and 84.2%, respectively.⁸⁰ Iodine vital staining is difficult to apply to keratinized oral mucosa such as hard palate and gingiva where glycogen content is much decreased. A combination of toluidine blue staining and iodine staining is also possible.

Tissue reflectance light visualization

Tissue reflectance light visualization has been advocated for detecting PMD or oral cancers. Light system products are commercially available. ViziLite Plus® uses a disposable chemiluminescent light packet, while the MicroLux unit offers a battery-powered light. Normal oral epithelium absorbs the illuminated light, while a dysplastic lesion with abnormal cells reflects the light and shines clear white because of increased nuclear:cytoplasmic ratio in the non-keratinized oral mucosa. This visualization may enhance brightness and sharpness of white lesions and lesions with both red and white, but it was reported not to improve visualization of red lesions.⁸¹ However, a recent study of screening oral examination using 100 consecutive patients showed that an acetic acid rinse may have some benefit in making mucosal changes more visible, but it also showed that the chemiluminescent light visualization was no better than conventional oral examination in detecting diagnosable lesions.⁸²

Fluorescence imaging

VELscope® is a portable device which has been developed to visualize the fluorescence alteration in PMD and oral cancers. This unit emits blue light of 400–460 nm through the handpiece into the oral cavity; the light penetrates the oral mucosa from the surface of the epithelium through to the basement membrane. Normal tissue produces fluorescence and appears as an apple-green glow, while dysplastic epithelium and underlying stromal disruption cause loss of fluo-

Table 33.10 Concept of diagnostic accuracy of the examination method in diagnosing the disease.

	Disease present	Disease absent
Diagnostic examination positive	True positive (TP)	False positive (FP)
Diagnostic examination negative	False negative (FN)	True negative (TN)
Sensitivity of the diagnostic examination (%) = $TP \times 100 / (TP + FN)$		
Specificity of the diagnostic examination (%) = $TN \times 100 / (FP + TN)$		
Positive predictive value (PPV) (%) = $TP \times 100 / (TP + FP)$		
Negative predictive value (NPV) (%) = $TN \times 100 / (TN + FN)$		
Accuracy of the diagnostic examination (%) = $(TP + TN) \times 100 / (TP + FP + FN + TN)$		

rescence and appear dark by comparison to the surrounding normal mucosa. The efficacy of the device in detecting dysplasia or carcinoma which was confirmed in histopathology by scalpel biopsy has been reported with a rather small number of patients.⁸³ The fluorescence is absorbed by hemoglobin so any vascularity will appear dark. Further extensive investigation will be required to confirm the usefulness of this device.

Brush cytology

Oral exfoliative cytology has been used for many years with some criticism as not presenting adequate and reliable results. A new technology of brush cytology named OralCDx[®] BrushTest[™], taking a transepithelial sample with a specially designed brush (Fig. 33.20) and diagnosis by computerized analysis in the laboratory (CDx Laboratories, Inc.) has been developed recently. The computer assists in search for and identification of abnormal cells, which are then visually assessed by the pathologist, and the result of the specimen is put into the following categories; “negative”: no epithelial abnormality; “atypical”: abnormal epithelial changes of uncertain diagnostic significance; “positive”: definitive cellular evidence of epithelial dysplasia or carcinoma.⁸⁴ This is a potential oral cancer case-finding device, and when an abnormal result is reported as atypical or positive then it must be followed by a scalpel biopsy to obtain a definitive diagnosis. The sensitivity and specificity of the OralCDx technique were reported as 92.3% and 94.3% respectively, in detecting dysplasia or carcinoma when both brush and scalpel biopsies were performed on all 96 lesions in 80 patients.⁸⁵

Scalpel biopsy

Histological diagnosis made by taking a sample tissue material from the lesion by means of a scalpel biopsy is still considered the gold standard for definitive diagnosis. However, the scalpel biopsy is a more invasive technology which can only be applied to a

limited number of sites at the same time. A method of “microbiopsy” by scraping the whole layer of epithelial tissue using a disposable dermatological curette to cause slight bleeding has been reported recently.⁸⁶ This microbiopsy can be applicable to PMD in the presence of multiple lesions such as in proliferative verrucous leukoplakia or oral lichen planus which may require repeated histological assessment to detect any dysplastic or malignant changes. Although a similar accuracy to scalpel biopsy in diagnosis by microbiopsy has been reported, the reduced dimensions of the microbiopsy samples and their lack of orientation suggest some disadvantages in recognizing the invasiveness of the lesion across the basement membrane.

A scalpel biopsy should be performed in cases of dysplasia to avoid false-negative results in cases of carcinoma. There are two methods for scalpel biopsy: incisional and excisional. An incisional biopsy is performed by taking a small piece of tissue sample with a scalpel for the purpose of making the definitive diagnosis. In the biopsy of PMD, the most malignancy-suspected part of the lesion, such as indurated, erosive, ulcerative, verrucous, or red areas, rather than a white area, is the preferred site to perform the biopsy. Vital staining by toluidine blue or iodine may help to define the most malignancy-suspected site of the PMD. If the lesion has a homogeneous appearance a marginal area of the lesion, including a normal-looking area, is a preferred site to make the incision. On the other hand, an excisional biopsy is performed to remove the whole area of the lesion for both treatment and definitive diagnosis (Figs 33.21–33.24). Therefore, it is indicated for smaller lesions, and the excision line must be placed outside the lesion, including peripheral normal mucosa.

Management

The main concept of the management of PMD is to avoid malignant transformation. As the most common PMD encountered in clinical practice is oral leukoplakia, the major focus of management of PMD



Fig. 33.20 A kit for brush biopsy.



Fig. 33.21 The lateral surface of the tongue showing white and red lesion. Clinical diagnosis was a non-homogeneous leukoplakia (speckled type).



Fig. 33.22 Excisional biopsy was performed with scalpel, including the whole area of the lesion.



Fig. 33.23 Excised tissue, 20 × 17 mm in dimensions.

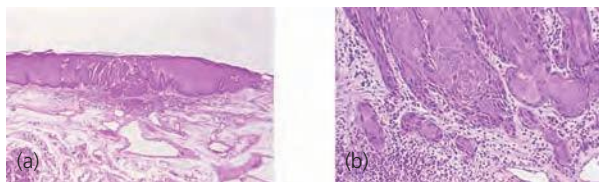


Fig. 33.24 Histopathological findings showed early invasive squamous cell carcinoma in the central part (b in high power field view) with acanthosis and hyperkeratosis in the periphery (a in low power field view).

can be considered to be that of leukoplakia. The management of other PMD can be modified according to the biological nature of each PMD and the histopathological characteristics of each PMD, including the degree of epithelial dysplasia.

The report by the latest workshop group of WHO (2005) on the consensus view on the management of PMD mainly focused on oral leukoplakia.⁸⁷ There are several protocols for the management of PMD: surgical treatment, such as excision by scalpel, electrosurgery (Figs 33.25–33.28), laser surgery; medical treatment with systemic or local chemopreventive agents such as vitamin A, retinoids, beta carotene, lycopene or some other topical ointments; cessation of risk activities such as tobacco, alcohol; “wait and see” policy under strict clinical and histological surveillance with frequent clinic visits and examinations including vital staining, tissue reflectance light visualization, fluorescence imaging, brush cytology, and scalpel biopsies if necessary.

Among these options, surgical removal is generally considered to be the recommended first choice and the most reliable way of management of PMD, especially for lesions with high malignant potential such as erythroplakia, proliferative verrucous leuko-



Fig. 33.25 A homogeneous leukoplakia on the lateral side of the tongue.



Fig. 33.26 Excisional biopsy was performed using an electro-surgical unit. (a) Excised tissue. (b) Surgical wound.

plakia, erythroleukoplakia, speckled type leukoplakia, palatal lesions in reverse smokers, and oral submucous fibrosis. The hypothesis that removing PMD by active surgical intervention (either scalpel or laser) can prevent the onset of oral cancer, remains unproved, however, and it is also considered reasonable that more conservative treatments or “wait and see” policy are options if the lesion is considered to have low malignant potential. The report of the 4th WWOM recommends the use of topical corticosteroids as the first-line treatment for oral lichen planus

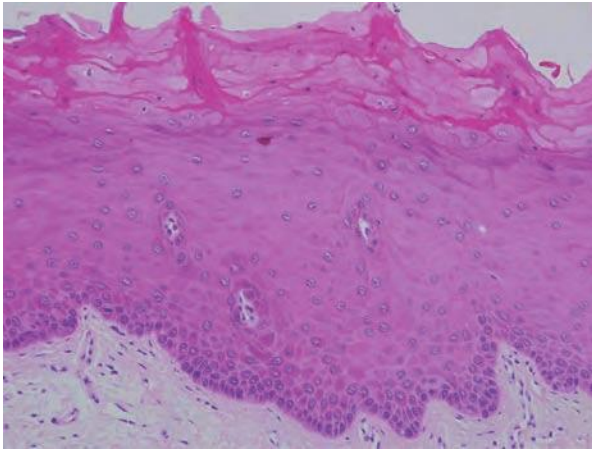


Fig. 33.27 Histopathological findings of tissue from Fig. 33.26(a) showing hyperparakeratosis and acanthosis without dysplasia.



Fig. 33.28 The surgical site from Fig. 33.26(b) has a normal appearance 1 year after the surgery.

(without dysplasia) and systemic corticosteroids for severe, widespread oral lichen planus.⁶² A report of long-term treatment outcome of PMD (leukoplakia and erythroplakia) in 236 patients in Denmark indicated that clinical types of leukoplakia (homogeneous or non-homogeneous) and the size of the lesion (exceeding 200 mm² or not) showed increased risk, but no other variables such as epithelial dysplasia, site and surgical intervention (surgical removal or not) showed statistically significant risk for malignant development by logistic regression analysis.⁴¹ However, most reported observational studies may have selection bias. It is possible that lesions considered at higher risk had a greater likelihood of being actively treated, whereas lesions with a less troubling clinical appearance may have been more likely to be selected for surveillance only.⁸⁷

Although removal of the PMD seems to be the predominant method of treatment, no definitive randomized controlled clinical trials have been undertaken. As for laser surgery, the CO₂ laser, NdYAG laser, and KTP laser have been employed for excision or vaporization for the treatment of PMD, especially oral leukoplakia. The main advantages of laser sur-

gery are the hemostatic effects, limited tissue contraction and scarring after therapy, reduced postoperative pain, swelling, and infection. These advantages may permit the treatment of large or multiple lesions. Vaporization does not provide a tissue specimen for histological diagnosis, which is a major disadvantage in laser vaporization procedures.⁸⁸ Cryosurgery does not seem to be of particular benefit in the management of leukoplakia: recurrence rates of 20–71.4% and malignant transformation rates of 7–25% are reported.

Surgical excision of PMD offers significant advantages over laser vaporization. The most important advantage is that excision generates a surgical specimen that provides the patient and surgeon with histological evidence of the entire lesion upon which to base future treatment planning.⁸⁹ However, the report of the 4th WWOM²¹ summarized from a systematic literature review that it is not possible to offer evidence-based recommendations for specific surgical (scalpel or lasers) or non-surgical (medical) interventions of dysplastic oral lesions due to the lack of randomized clinical trials at present. If further investigations can identify specific molecules associated with malignant progression, these could possibly serve as therapeutic targets for future management strategies of PMD.

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Chapter 34

Principles of Oral Cancer Management

Brian L. Schmidt

This chapter presents the reader with the principles of oral cancer management. The focus of the chapter will be the comprehensive evaluation and treatment of the oral cancer patient. Oral cancer management should be patient focused, evidence based and surgeon driven. Oral squamous cell carcinoma (SCC), which comprises 95% of oral cancers, is a surgical disease. Oral cavity carcinoma has peculiar biologic and capricious clinical behavior relative to other head and neck sites. The recommendations for treatment are based on the available literature. Few studies focus specifically on oral cavity carcinoma and most published information has come from studies which include other head and neck sites (i.e. oropharyngeal, hypopharyngeal, and laryngeal). A comprehensive understanding of the relevant literature, as well as its shortcomings, is requisite. Moreover, the surgical oncologist specializing in oral cavity carcinoma must have a working and intimate knowledge of the indications, toxicity, and efficacy of adjuvant therapy.

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Epidemiology and risk factors

The incidence of oral squamous cell carcinoma, particularly in younger patients, is increasing in North America and the UK.^{1,2} In certain regions of the world, such as India, oral cancer is one of the most common malignancies. In these regions of the world the use of betel nut and betel quid clearly contribute to the high incidence of oral cancer. However, the etiology, outside of tobacco and alcohol use, in the western portions of the world is less clear. Survival rates for oral cancer patients have not changed significantly. Survival for young patients with oral SCC has been equivocal with early studies showing poorer

survival³ and more recent studies showing improved survival.⁴ The roles of tobacco and alcohol are firmly established as risk factors for oral cancer. However, Schmidt *et al.* demonstrated that approximately 33% of oral squamous cell carcinoma patients were never smokers.⁵ Our understanding of the role of smokeless tobacco in oral carcinogenesis has gone through considerable evolution over the last three decades. The conclusion of a recent review of 32 relevant studies published between 1920 and 2005 concluded that smokeless tobacco, at most, plays only a minor role in the development of oral cancer.⁶ A facile, but erroneous, explanation for the increase in oral cancer rates with younger patients has been human papilloma

virus (HPV). There has been considerable confusion regarding the role of HPV in the development of oral cancer. When evaluating studies addressing the role of HPV in head and neck cancer it is important to consider the method of sample collection as well as the assay used to measure HPV. Based on the available studies there is a lack of reproducible molecular data supporting the role of HPV in the development of oral cancer.⁷ A multicenter case-control study of 1415 cases of oral cavity cancer detected HPV DNA in only 3.9% of biopsy specimens.^{7,8} This is in contrast to oropharyngeal carcinogenesis where HPV has a much stronger etiologic role.^{7,8}

Histologic grading, tumor staging, and clinical behavior

Ninety-five percent of oral cancers are squamous cell carcinoma (SCC). Different histologic variants of SCC include verrucous SCC, basaloid SCC, and papillary SCC (Fig. 34.1). Clinicians and investigators have attempted to use the combination of clinical information and histologic findings to predict the behavior of oral SCC. The histologic features that have been evaluated include tumor differentiation, tumor thickness, tumor size, and tumor invasive pattern.

Tumor differentiation

Broders' grading system categorizes carcinomas into well, moderately, and poorly differentiated; however, there is little evidence that tumor grading is of value in predicting metastasis or managing oral SCC.⁹ While Byers *et al.* demonstrated a significant association between tongue SCC tumor grade and metastasis, a significant limitation of this study is that the authors did not analyze the clinically negative nodes in the patients with T1 and T2 lesions.¹⁰ A more recent study of 71 patients with SCC of the oral tongue evaluated the Anneroth grading system, the Bryne grading system, and the Martinez-Gimeno grading system, as well as stage, perineural invasion, vascular invasion, lymphovascular invasion, shape, stage, growth pattern, and thickness.¹¹ After careful analy-

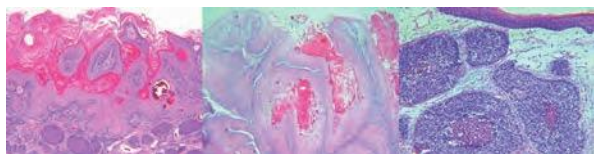


Fig. 34.1 Histologic variants of oral squamous cell carcinoma. Papillary SCC (left) shows mild degrees of atypia with invasive islands seen in the lamina propria. Verrucous carcinoma (center) is characterized by proliferation with broad, deeply pushing epithelial rete with parakeratin plugging and no atypia. Basaloid SCC (right) demonstrates islands of invasive, basaloid appearing malignant epithelial cells with palisading peripheral cells and comedo (central) necrosis.

sis, only tumor thickness was an independent risk factor for nodal metastasis. This study demonstrated no association between tumor grade and subclinical nodal metastasis.

Modified versions of the grading system have been published by Anneroth *et al.*¹²⁻¹⁴ This grading system includes several factors including degree of keratinization, nuclear polymorphism, number of mitoses per high power field, pattern of invasion, and lymphoplasmacytic infiltration. Other factors that have been studied include, but are not limited to, tumor shape (reductive or expansive), growth pattern (endophytic or exophytic), perineural invasion, vascular invasion, muscular invasion, and depth of invasion. Kademani *et al.*, using the Anneroth malignancy grading system with modifications by Woolgar *et al.*, found in a series of 215 patients that this grading system was predictive both of disease-free survival as well as neck metastasis in oral SCC.¹⁵ Overall, tumor grading has not been shown to be a reliable predictor of outcome or metastasis.

Tumor thickness

Similar to the significant predictive value of tumor thickness with malignant melanoma, investigators have studied tumor thickness in oral cancer to determine whether thickness can be used to predict cervical metastasis. In 1970, Breslow demonstrated that tumor thickness is a primary predictive variable with malignant melanoma.¹⁶ For oral SCC tumor thickness is the only factor that has consistently shown promise as an indicator of risk for occult nodal metastasis; however, the recommended thickness cutoff for prescribing elective neck treatment based on the rate of metastasis varies greatly in the literature and ranges from 1.5–8 mm.^{11,14,17-23} Mohit-Tabatabai *et al.* retrospectively reviewed 84 cases of patients with floor of the mouth SCC and found that when patients had tumor thickness less than 1.5 mm the incidence of cervical metastasis was 1.8%. Tumor thickness greater than 1.5 mm was associated with a cervical metastasis rate of 48%.²³ Spiro *et al.* retrospectively evaluated 105 patients with either tongue or floor of the mouth SCC and found that 2 mm or less of thickness carried a metastasis rate of 7.5% while greater than 2 mm thickness carried a metastasis rate of 38%.²² Byers *et al.* evaluated 91 tongue SCC patients to determine whether preoperative or intraoperative factors including thickness of the specimen, depth of muscle invasion, frozen margin status, perineural invasion, vascular invasion, lymphatic invasion, histologic differentiation, or DNA ploidy could be used to predict lymph node metastasis.¹⁰ In this study the best predictors for nodal metastasis were greater than 4 mm of muscle invasion, double DNA ploidy and histologic differentiation.¹⁰ The DNA ploidy results agree with other studies demonstrating a weak correlation between DNA ploidy status and cervical metastasis in tongue SCC patients.²⁴

Studies which have evaluated tumor thickness highlight some of the pragmatic problems with using tumor thickness as a risk index. The first practical problem is the manner in which tumor thickness is measured. Some studies measured the entire thickness of the carcinoma from the surface of the carcinoma to the deepest point of invasion. Others advocated a “reconstructed” thickness, measuring from a line that approximates the boundary of where the normal mucosa would be to the deepest extent of the tumor. Other approaches focus on depth of invasion, only measuring from the basement membrane to the deepest extent of tumor. The variability in techniques likely contributes to the wide range of thickness cutoff values in the literature that are associated with metastasis.

The second practical problem is one of sampling and how to best determine tumor thickness. All of the studies are retrospective and the majority used paraffin fixed sections from the primary tumor specimen to determine thickness. With this approach, the pathologist has the ability to first grossly examine the entire tumor *ex vivo* and identify the portion containing the deepest extent of invasion. However, the decision to electively treat the neck is generally based on an incisional biopsy before surgical resection and pathologic evaluation of the primary tumor. Therefore, the measured thickness obtained from the incisional biopsy will often be an underestimation of the true maximal tumor thickness. Therefore, applying the measurements based on the incisional biopsy alone will often lead to undertreatment of the neck. Another problem with using recommended cutoffs for tumor thickness is that the majority of studies addressing tumor thickness are based on the argument that failure rates up to 20% are an acceptable risk. Whether this risk is truly acceptable depends on the surgeon and, more importantly, on the patient. Warburton *et al.* approached their determination of a thickness cutoff differently. They attempted to find a point where both the sensitivity and specificity for predicting neck metastasis were maximized. They settled on a threshold of tumor thickness of 2.2 mm which yielded a sensitivity and specificity of 87.5% and 78.9% respectively.¹⁸ Although this is likely one of the most objective ways of creating a useful risk measurement tool, their cutoff at 2.2 mm is small enough to make use of this cutoff value problematic to execute in a clinical practice. A thickness of 2.2 mm would include all but the most superficial T1 tumors.²⁰

Tumor size and staging

The American Joint Commission on Cancer (AJCC) TNM staging protocol must be used to stage all patients with oral cancer.²⁵ Staging is an informative and critical step in determining a patient’s prognosis, as well as a critical administrative process requisite for quality research. There is some debate on the

accuracy of the current staging system for prognosticating, especially with regard to predicting nodal metastasis. Higher stage tumors have been correlated with higher rates of positive margins on resection, higher rates of local recurrence, and lower 5-year survival rates.²⁶ Bell *et al.* have shown that prognosis depends on stage.²⁷ Though advanced stage is used to predict risk of occult metastasis, multivariate analyses have shown that stage is not an independent risk factor for neck involvement when tumor thickness has been factored out.^{11,28} The surgical oncologist must keep in mind that even T1 tongue SCCs are associated with a significant rate (greater than 20%) of cervical metastasis.^{29–35} Management of the neck in patients with oral SCC is discussed more thoroughly later in the chapter. The AJCC system has been criticized for its simplicity. The system is based on surface diameter and excludes other factors that may play a larger role, such as perineural invasion, depth of tumor invasion, and the histopathologic features of the invasive front. Despite this, the simplicity of using the superficial diameter of a lesion as a prognostic tool, especially considering that it is likely to have some relationship to tumor thickness, still makes staging a frequently used measure that is the current standard of care.

Tumor invasive pattern

Relative to tumor thickness there is little information in the literature on the correlation between the tumor invasive pattern and the rate of metastasis; however, the different patterns of tumor invasion have been shown to have varying metastasis rates.^{36,37} SCCs that infiltrate along a broad front are much less likely to metastasize than SCCs comprised of separate packets of invading cells. Woolgar *et al.* have concluded that the pattern of invasion affects the interpretation of margins and even a clear (i.e. 5 mm) margin might be inadequate in the setting of widely separated clusters of invading tumor cells.³⁸ Despite these studies, an evaluation of invasive pattern is generally not used in clinical practice to guide treatment.

Perineural invasion

Some oral SCCs are neurotrophic and expression of nerve cell adhesion molecule (NCAM) on the SCC is associated with perineural invasion.^{39,40} While most studies have demonstrated that perineural invasion is a histopathologic feature that correlates with increased local recurrence and cervical metastasis,^{41–44} perineural invasion has also been shown not to correlate with metastasis. The presence of perineural invasion has been recommended as a potential indication for postoperative radiation therapy. However, the presence of perineural invasion should be considered in the setting of other features, e.g. margin status and lymph node involvement. In some cases perineural invasion can be identified using an

MRI. If perineural invasion is suspected based on preoperative MRI then care should be taken at the time of the operation to obtain a wide margin. Intraoperative frozen margin analysis of the nerve should be used to guide the extent of resection. The use of perineural invasion as an independent factor to predict prognosis and recommend treatment is unclear at this time.

Proliferative verrucous leukoplakia

Most oral SCCs are preceded by clinically evident precancerous oral lesions, including oral epithelial dysplasia, which appear as white (leukoplakia) or red patches (erythroplakia) that are characterized microscopically by varying degrees of dysplasia (from mild to severe).⁴⁵ Other oral SCCs are preceded by proliferative verrucous leukoplakia (PVL), a condition which is persistent, recurrent, and multifocal. Dysplasia has an unpredictable transformation rate to oral SCC that is thought to be approximately 16%,⁴⁶ whereas the transformation rate for PVL is much higher (50–70%).⁴⁷ PVL is a preneoplastic lesion that is recognized for its extremely high recurrence rate following surgical excision, its multifocal development, and its relentless progression to lesions of increasing severity (Fig. 34.2).^{47,48} PVL was first described in 1985 by Hansen.⁴⁸ In contrast to oral dysplasia, PVL is not associated with tobacco or alcohol consumption. In addition, whereas dysplasia is more common in men, the female to male ratio for PVL is about 4 to 1.^{47,48} At initial presentation, PVL lesions are white, irregular areas on the attached gingiva, buccal mucosa, and tongue. Histologically, PVL exhibits no dysplasia or mild dysplasia.⁴⁸ In spite of the bland clinical and histologic appearance of early lesions, PVL is characterized by relentless growth

and irreversible progression through a continuum of lesions, namely, hyperkeratosis, verruciform hyperkeratosis, verrucous hyperplasia, verrucous carcinoma, and ultimately SCC (Fig. 34.3). Repeated surgical excisions of all clinically and histologically appearing abnormal tissue can slow the transformation to carcinoma. However, such treatment results in significant disfigurement and morbidity. Moreover, surgical treatment does not halt the eventual progression to carcinoma.^{47–49} Despite studies which have evaluated the role of HPV, Epstein-Barr virus, p53 expression, transforming growth factor α , *Candida albicans*, and DNA ploidy, the cause of PVL is not known.^{50–55}

Preoperative assessment, staging, and work-up

The work-up of the oral cancer patient begins with a comprehensive history and physical examination. The pathology report should be reviewed to confirm the diagnosis. The histopathology slides should be reviewed by a pathologist with whom the surgeon works closely within their institution. A detailed, well performed history and physical examination can be used to understand the clinical behavior of the oral cancer. Oral cancers are a heterogeneous group and display capricious clinical behavior. Some understanding of the clinical behavior of an oral cancer can be gained by reviewing the history of the cancer and asking the following questions: when was the lesion first noticed, are there any associated symptoms (ear pain, swallowing difficulty, speech problems, limitation of tongue movement), have suspicious lymph nodes increased in size, is the patient having oral pain, is the pain changing and finally does the patient feel the lesion is growing rapidly?



Fig. 34.2 Photograph of a patient with PVL. All three photographs are from the same patient. The patient has extensive involvement of the maxillary and mandibular gingival, buccal mucosa and palate. As depicted here PVL is characterized by extensive verrucous-appearing lesions with areas of ulceration.

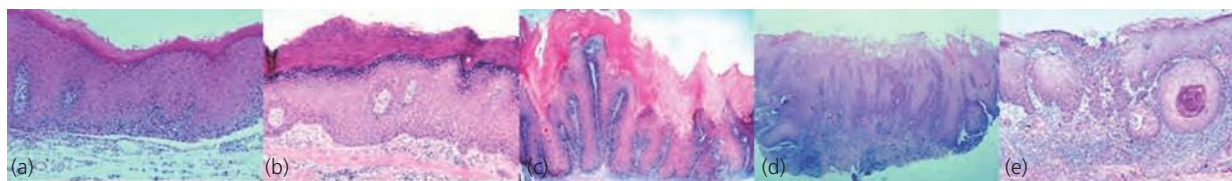


Fig. 34.3 Histologic progression of PVL. The stages of histologic progression of PVL include (a) hyperkeratosis, (b) verruciform hyperkeratosis, (c) verrucous hyperplasia, (d) verrucous carcinoma, and (e) squamous cell carcinoma.

Most oral cancers are painful.⁵⁶ Pain is the most common presenting symptom for patients with oral cancer. For oral cancer patients, pain severely limits their oral function including eating, drinking, and talking.⁵⁶ Pain is rated as the oral cancer patient's worst symptom and is the primary determinant of a poor quality of life.^{56–59} In their final period of life, pain is reported by 85% of oral cancer patients as their most common problem.⁶⁰ Opioids, such as morphine, are the only drugs that have any efficacy for oral cancer pain. Morphine provides minimal relief, does not restore oral function and progressively larger doses are required; therefore, tolerance rapidly develops. Despite these findings and the severe nature of oral cancer pain, surprisingly little research has been done to understand the etiology of pain associated with oral cancer or to discover effective analgesics. The University of California San Francisco has developed and validated a questionnaire specifically for patients with oral cancer pain.^{56,61} This questionnaire can be used to monitor pain control either during the preoperative phase or during the late stage of disease following treatment failure. Pain management should be tailored to provide relief when patients are required to function. Total resection of the oral cancer produces near complete relief.⁶¹ However, patients have severe pain prior to surgery, some oral cancers are unresectable, some patients are too sick to have surgery, and many patients develop a recurrence of the cancer or a second primary oral cancer. Approximately 50% of oral cancer patients will not be cured with surgery, chemotherapy, or radiation therapy. The surgeon, as the head of the multidisciplinary team, must remind members that oral cancer patients experience excruciating pain during their final months of life.

The tobacco history should be reviewed and the patient should be classified as a never smoker, non-smoker, or smoker. If the patient is a smoker or non-smoker the pack-years of use should be calculated. If the patient is a non-smoker the time since the patient's last cigarette should be determined. While there is conflicting data in the literature as to whether marijuana use contributes to oral carcinogenesis,^{62–64} patients should be asked whether and how much marijuana they use.

Systematic palpation of the neck should be performed on every patient at every visit.⁶⁵ Palpation of the neck should be performed on one side of the neck at a time and should always be performed from behind the patient. The sensitivity of clinical exami-

nation to detect metastasis, even by expert surgeons, has been inadequate, with a sensitivity of 51% reported by one major head and neck surgery group.¹⁰ Other groups have also reported on the low sensitivity of clinical exam, ranging from 60–70%.^{66–68}

A panoramic radiograph should be performed in almost all cases of oral cavity SCCs. Even if the carcinoma does not involve the maxilla or the mandible the dentition should be evaluated prior to surgery to determine whether extractions will be required at the time of surgical resection if postoperative radiation therapy is anticipated. Nasopharyngoscopy can be easily and painlessly performed with proper preparation of the nasopharyngeal membranes with local anesthetic and a topical vasoconstrictor. Nasopharyngoscopy can be used to evaluate the nasopharynx, base of tongue, and supraglottic structures.

The significant limitations of imaging for detecting carcinoma need to be considered when evaluating the oral cancer patient, particularly when attempting to identify metastasis in the oral cancer patient who is N0 on clinical exam. Imaging, in the forms of computed tomography (CT), magnetic resonance imaging (MRI), positron emission tomography (PET), ultrasound (US), or now PET/CT, has been used to assess primary tumors. Unfortunately, despite improved resolution and software analysis, all imaging techniques are still insufficiently sensitive for detecting occult neck metastases, with 20–45% of patients staged as N0 using these techniques having occult nodal involvement on pathologic evaluation of the neck.^{10,69,70} In order for imaging techniques to be useful in making treatment decisions, they must be able to detect metastatic nodes that are found in patients with early stage tumors, nodes that are present only 20–45% of the time. These nodes are commonly small, with diameters as small as 3–10 mm. Also, the long axis of these nodes, which can be visualized accurately on pathology, is often not parallel to any of the planes used for anatomic imaging which causes even greater foreshortening.^{70,71} The presence of structural changes, such as cystic changes or necrosis, aids in detection, however these features are rarely present in occult disease. In addition, within this group of patients, these techniques must not only be sufficiently sensitive to detect microscopic disease but also specific enough so that frequent false-positive results do not render universal prescription of elective neck treatment.

Anatomic imaging studies such as MRI and CT have been shown to have similar accuracies, with

sensitivities ranging from 56–85% and specificities from 47–95%.^{72–75} PET has a sensitivity of approximately 75–90% and specificity of 90–100%. One must consider that in most of the studies which have attempted to measure sensitivity and specificity the patients did not exclusively have N0 necks. One would expect the sensitivity and specificity to be much lower for detecting occult nodes in earlier stage disease.

Preoperative imaging of the primary cancer and the head and neck should include CT or MRI. MRI is often more helpful than CT in evaluating the primary cancer because of the high soft tissue resolution



Fig. 34.4 Coronal view of MRI of lateral tongue squamous cell carcinoma. MRI can provide high resolution and anatomic detail which aids in clinical staging and surgical planning.

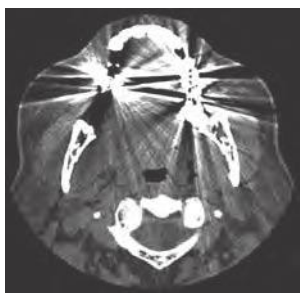


Fig. 34.5 Dental artifact from amalgam obscuring CT. One of the significant drawbacks of CT in the evaluation of an oral cavity primary is the artifact generated by dental materials.

which MRI provides (Fig. 34.4). Moreover, MRI is less obscured by the dental artifact that occurs with high density materials such as dental amalgam. CT can aid in the evaluation of bone involvement; however, its usefulness is often limited by such dental artifact in the area of interest (Fig. 34.5). When reviewing the MRI, all views should be closely examined to help understand the extent of tumor invasion. Depending on the anatomic structure being evaluated, particular views will give more information regarding the extent of invasion. The following recommendations are for interpreting MRI images. Evaluation of oral cavity primary can be difficult and often T1, T2 and gadolinium-enhanced views are required. Fat saturation should be used for postgadolinium and T2 views. The coronal plane is often most helpful for evaluating the primary, particularly the tongue (Fig. 34.4). In evaluating the mandible the T1 images are most helpful to determine if the marrow is involved. T2 and gadolinium-enhanced views, both using fat saturation, provide the most information regarding inferior alveolar nerve involvement (Fig. 34.6). For assessment of mandibular invasion both the axial and coronal planes are critical. Evaluation of cervical nodes is often required in the presurgical setting as well as in the postsurgical setting during follow-up. Cervical nodes are best evaluated with the axial plane views. For presurgical evaluation the T2 view is often the most useful. Following surgery the T1 view can be more helpful due to intensity changes that occur in the nodes following surgery.

PET imaging with fluorine-18 fluorodeoxyglucose (FDG) has been shown to have a higher sensitivity and specificity than traditional CT or MRI in the evaluation of the patient with head and neck SCC.⁷² However, the results of PET scan rarely change the treatment decision in the management of oral SCC. In the case of an N0 neck in a patient with oral SCC the ¹⁸F-FDG PET scan does not have a clear role.^{76,77} The poor resolution of PET/CT makes it difficult to use for surgical planning. Nahmias *et al.* examined the staging capacity of PET/CT on a node by node

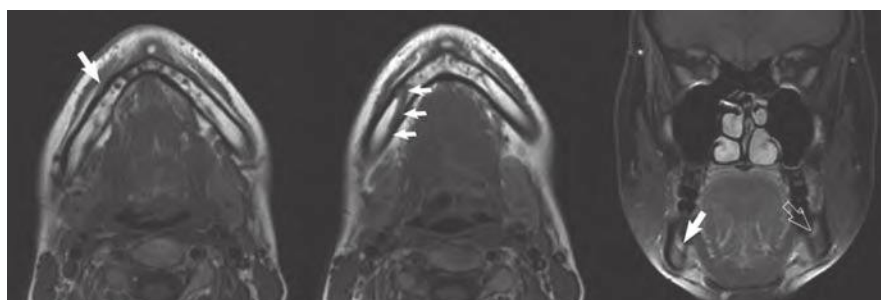


Fig. 34.6 Perineural invasion in a 35-year-old woman with oral SCC of the gingivobuccal sulcus. The patient complained of a numb chin. Axial T1 MRI weighted image (left) shows subtle soft tissue thickening (arrow) in right gingivobuccal sulcus adjacent to the right mental foramen. Adjacent T1 weighted image (center) shows marked thickening of the right inferior alveolar nerve within the mandible (arrows), suspicious for perineural carcinoma involvement. Coronal T1 weighted image (right) with fat saturation shows an asymmetrically thickened and more enhancing right inferior alveolar nerve (arrow) compared to the normal left side (open arrow). Pathologic evaluation of the resected specimen demonstrated carcinoma involving the mental nerve first 3.5 cm from mental foramen at surgery.

basis.⁷⁷ In this carefully performed study, the investigators mapped out each node from a neck dissection and then compared it to the corresponding node that was visualized on the preoperative PET/CT. Their study included a total of 70 patients with oral cancer, 47 of which had N0 necks and 19 of which were clinically N+. In their 47 N0 patients, they found that the sensitivity and specificity of PET/CT for detecting patients that have metastatic nodal involvement was 79% and 82% respectively, with a positive predictive value (PPV) of 68% and a negative predictive value (NPV) of 89%. When analyzing on a node by node basis in N0 patients, the sensitivity and specificity was 26% and 99% respectively, with a PPV of 63% and a NPV of 95%. Nahmias *et al.* concluded that, due to a NPV of 89% and a false-negative rate of 11%, the use of PET/CT was not helpful in ruling out occult nodal metastasis to a degree that would affect a surgeon's decision to electively treat the neck.

In a prospective study of 31 oral cancer patients with N0 necks it was found that the role of ¹⁸F-FDG PET is limited due to the combination of limited sensitivity for small metastatic deposits and a relatively high number of false-positive findings.⁷¹ Schoder *et al.* found, in a cohort of patients with N0 necks, that PET/CT had a sensitivity and specificity of 67% and 85% respectively.⁷¹ They determined, using histopathology, that nodes 3mm or smaller could not reliably be detected by PET/CT. Thus Schoder *et al.* concluded that, despite overall high accuracy, PET/CT has limited clinical utility in this application due to the inadequate sensitivity for small nodes and a high number of false-positives (13%). They also emphasize in their paper that the surgeons in their group had the results of the PET/CT before treatment planning, and that in none of the patients did the information alter the decision to operate.

PET has been proposed to evaluate for distant metastasis but distant metastasis is rare in oral SCC. PET scans are also associated with a false-positive rate which often obligates the clinician to pursue "hot spots" often leading to a delay in treatment. PET scans theoretically could impact on the decision to perform a neck dissection; however, micrometastasis is unlikely to produce a positive PET result. The rate of occult metastasis for almost all oral cavity SCCs dictates that a neck dissection should be performed regardless of the results of a CT, MRI, or PET scan. With the currently available technology a PET scan often gives false security or false worry in the initial assessment of patients with oral cavity SCC. Moreover, indiscriminant use of the PET scan significantly burdens the health care system. The PET scan can be helpful in evaluating oral cancer patients with symptoms suggestive of recurrence when no clinically apparent lesion is found; it also clearly has a role in the patient with an occult primary head and neck cancer.

The neck can also be evaluated with ultrasound. Van den Brekel and Castelijns' group has reported in

multiple studies their success with using ultrasound-guided fine-needle aspiration biopsy with a "wait and see" approach for patients with N0 necks.^{66,78-81} In one such study, Nieuwenhuis *et al.* reported that 21% of their patients using this strategy developed nodal disease during follow-up. These patients were then treated with a therapeutic neck dissection of levels I-V, with a salvage rate of 79% at 5 years.⁷⁸ However, it appears that diagnosing occult metastasis with ultrasound is highly technique-sensitive and user specific, as other studies have not been able to replicate these authors' success.^{10,82}

A chest radiograph is part of the standard work-up recommended by the National Comprehensive Cancer Center Network for head and neck cancer patients. The role of the chest radiograph is for the evaluation of distant metastasis. Approximately 1% of patients with a head and neck SCC will have a synchronous primary in the lung.^{83,84} For patients with oral cavity SCC a chest CT would only be indicated in the setting of advanced cervical involvement or if a suspicious lesion were identified on the chest radiograph.⁸⁵ The use of panendoscopy is dependent on the provider and concern regarding a second primary. The cost effectiveness of panendoscopy is questionable given the relatively low incidence of second primaries.

Following the history and physical examination each patient should be staged according to the AJCC staging system.²⁵ Clinical tumor staging should be based on all clinical, imaging, and pathologic data.²⁵ The surgeon must complete a staging form for each patient and submit this form to the medical center's cancer data management office. Every oral cancer patient deserves to be presented at a multidisciplinary tumor board. Management of oral cavity SCC almost always involves surgical resection with consideration of adjunctive chemotherapy and radiation therapy. Because of the significant, progressive side-effects, radiation therapy should be avoided if possible, as will be discussed below.

In planning for surgery patient performance should be evaluated. The Karnofsky index will give a good estimate of the patient's ability to tolerate surgery, radiotherapy, and chemotherapy.⁸⁶ The patient's goals for treatment should be discussed and considered. The wishes of the patient and family must be weighed heavily when considering treatment options. The question is often raised of what to do with an elderly patient with a limited life expectancy. It is often a mistake to not treat the cancer since many of these patients will live much longer than expected and if the cancer is not treated patients will progress to endure profound pain. Surgical resection offers significant pain relief.⁶¹

Nutritional support has many benefits for the surgical patient, including improved healing following surgery, decreased complications, lean body mass preservation, and improved quality of life. A common belief among clinical oncologists is that the

above mentioned benefits of nutritional support have an inverse negative impact on the malignancy. The surgical oncologist might be surprised that such nutritional support is potentially benefiting the head and neck carcinoma as well. A secondary analysis of Radiation Therapy Oncology Group (RTOG) 90-03, a prospective randomized trial evaluating four definitive radiation therapy fractionation schedules in patients with locally advanced head and neck SCC, prospectively collected data on the nutritional status of the 1073 evaluable patients. Nutritional status of the patients was analyzed against therapy toxicity and outcome.⁸⁷ This analysis is the largest evaluation of prospectively collected nutritional support data and the impact of nutritional support on treatment outcome in patients with cancer participating in a clinical cooperative group trial. Contrary to the result anticipated, nutritional support prior to therapy was shown to be a highly significant independent prognostic factor for increased locoregional failure and death. The reason for this result can possibly be found in a large prospective trial analyzing 92 patients with gastrointestinal cancer and malnutrition. The study evaluated the impact of nutritional support and found that parenteral nutritional support lead to a significant increase in tumor proliferation as measured by cells in S phase, DNA content, and DNA index.⁸⁸ Clearly, the role of nutritional support in the management of the oral cancer patient is potentially controversial and the surgical oncologist must weigh the benefit of nutritional support against the potential negative impact on cancer control.

Meticulous planning prior to surgery will tip the balance in favor of a favorable patient outcome. All studies should be carefully reviewed. Surgical planning should include plans for airway management, discontinuation of coagulants, and necessary preoperative planning with bridging therapy, planned ablation, and reconstruction. Tracheostomy is rarely required in the setting of an oral resection with a neck dissection.⁸⁹ Nasotracheal intubation is generally a safe alternative to tracheostomy. Tracheostomy is strongly favored if a microvascular free flap is planned or if there are concerns for postoperative edema. If the patient requires a return to the operating room for salvage of a dying flap, intubation in the setting of a newly constructed oral cavity and oropharynx can be difficult.

Surgical treatment of oral cancer based on subsite

Nothing impacts on patient outcome more than the oncologic surgeon's attention to detail at the time of primary resection and dissection of the neck lymphatics. Because the structures that are affected by oral cancer impact directly on oral function, for example chewing, a comprehensive understanding of the functional aspects of the anatomy, such as

occlusion, is required to provide the patient with restoration of function. An appropriate margin of normal mucosa must be resected with the primary. The surgeon's focus at the time of surgical resection must be on maintaining such an appropriate margin around the carcinoma and avoiding violation of the carcinoma. The clinical implications of an appropriate cuff of histopathologically normal tissue around a resected cancer have been extensively studied and reported.⁹⁰⁻⁹⁷ The distinctions between clear, close, and involved margins have been variable throughout the literature and arbitrary in designation;^{97,98} however, it is generally agreed that proximity of the cancer to the resection margin is a strong negative prognostic finding. Given the anatomic complexity of the oral cavity, achieving an adequate margin following resection of a cancer is difficult and frequently the histopathologic margins are reported to be significantly short of those measured clinically, the *in situ* margin. The discrepancy between the pathologic margin and the *in situ* margin might explain the high rates of close or positive margins reported in the recent literature.^{38,97,99,100}

To achieve an appropriate margin at the time of surgery, the extent of the primary should be determined based on a review of available imaging prior to surgery and on visual inspection and digital palpation once the patient is under general anesthesia. The primary can then be marked out with a surgical marking pen followed by careful measurement of a 1–1.5 cm margin (Fig. 34.7). The 1 cm margin is arbitrary and there has been no study to demonstrate that this critical cutoff of 1 cm measured around the primary *in situ* shows improved tumor control. It is tempting to suggest that increasing margins of resection universally should be adopted, in light of the increased availability of improved reconstructive options with microvascular free tissue transfer. However, increasing margins of resection from 1 cm to a higher standard has not yet been shown to improve prognosis. While the surface margins guide the initial incision, three-dimensional conceptualization of the cancer is critical as the resection is carried deeper. A 5 mm margin of histopathologically normal mucosa is the accepted designation between clear and close in terms of differences in prognosis.^{90,98} Finger extensions or islands of tumor may invade out of the main mass of tumor resulting in a margin that is closer than anticipated.³⁸ Alternatively, tissue retraction that occurs following resection and pathologic processing of the specimen may cause the margin of tissue to decrease in size.¹⁰⁰ Molecular biologic techniques have shown that even when margins are judged "clear" on histopathologic review, genetic changes often remain in normal-appearing mucosa, changes that increase the risk of local recurrence.¹⁰¹⁻¹⁰³ This potentially wider and invisible area of genetically altered cells with premalignant genetic aberrations makes blindly aggressive resection ineffective. The appropriateness of the final surgical margin var-

ies with the oral subsite resected.¹⁰⁰ As the adequacy of resection margins is paramount to successful treatment, it would be prudent to consider varying the size of the margin of resection based on tumor location, stage, or any other clinical factor that may affect margin status unfavorably. While the discussion below is based on oral cavity subsite, in many cases of oral SCC contiguous structures are involved. In such cases in continuity resection of the involved structures, with the neck dissection if possible, should be performed (Fig. 34.8).

Tongue

The tongue is the most common oral site for SCC. Tongue carcinoma infiltrates the parallel muscle fibers and there is no fascia to resist invasion. The rich lymphatic supply leads to early cervical metastasis. The lymphatic drainage of the lateral tongue, the most common portion of the tongue affected, is into the submandibular and jugulodigastric nodes. The base of tongue is embryologically, anatomically, and biologically distinct from the oral tongue. Base of tongue SCC responds well to chemotherapy and radiation. In clear distinction, surgery, with consideration of postoperative chemoradiotherapy, is the primary treatment for oral tongue SCC. If possible the tongue should be resected with continuity with the neck. However, this approach requires a flap, either microvascular or local, to prevent communication between the oral cavity and the neck. If the primary and neck are resected separately then the intervening lymphatics, within the floor of mouth along the mandible, are at risk for metastasis following surgery. The intervening lymphatics in the floor of mouth and submandibular region are a rare but reported site of failure in patients undergoing partial glossectomy and neck dissection. The tongue anatomy must be carefully considered when resecting the tongue. A common error is to measure and follow a 1 cm margin

on the surface but to not achieve a 1 cm margin in the deep aspect of the tongue. Even for superficial SCCs of the tongue a 1 cm deep margin will almost take the surgeon to the tongue midline. The depth should be established at the lateral margins of the tongue resection prior to carrying the dissection to the deep portion (Fig. 34.7).

Commonly used methods of tongue reconstruction include a split-thickness skin graft or a microvascular free flap. The microvascular free flap adds bulk and can restore articulation. The decision regarding reconstruction must be based on the patient's expectations, requirement for function, planned resection, and health status. For patients who have had a near-total or total glossectomy the need for microvascular reconstruction is clear. However, for resections comprising the hemitongue or less, the remaining tongue compensates significantly. Speech intelligibility scores with tumors in the anterior are significantly lower than those in the middle or posterior tongue. When the tip of the tongue or floor of the mouth can be preserved, intelligibility scores increase. The more anterior portions of the tongue participate in the articulation of consonants including plosive, fricative, affricate, and nasal.^{104,105} These sounds require



Fig. 34.7 Intraoperative measurement of resection margins for oral SCC. At the time of surgical resection great care should be taken to establish the appropriate margin in three dimensions. The lesion should be carefully marked out based on review of the appropriate imaging studies, as well as visualization and palpation of the lesion. The surface surgical margin should be marked out (left). The imaging studies can be used to predict the depth of infiltration. Once the resection is started the appropriate depth, based on estimated depth of infiltration and appropriate surgical margin, should be carefully established and measured prior to dissecting deep to the carcinoma (right).

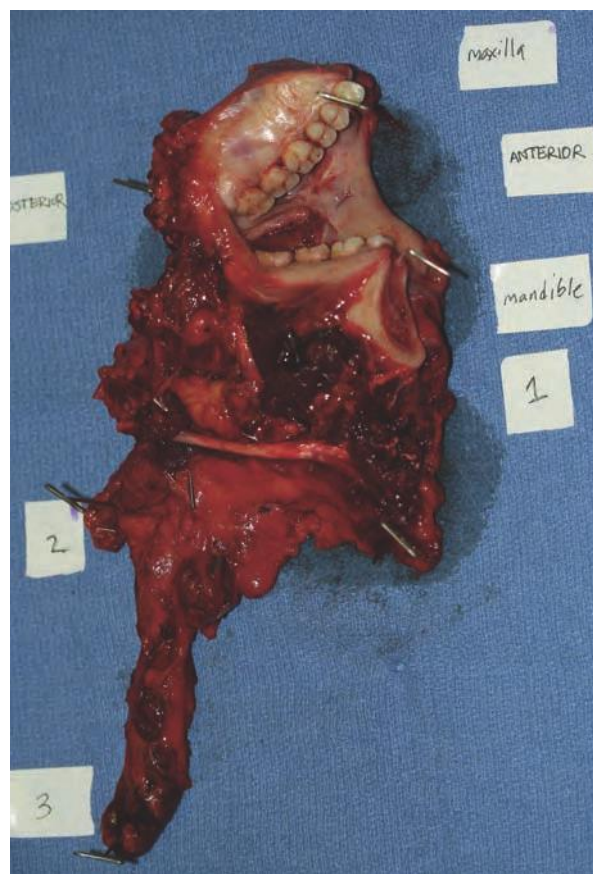


Fig. 34.8 The resected and labeled specimen from a 58-year-old man with an SCC involving the mandibular and maxillary gingiva, oropharynx, and the buccal mucosa. The specimen has been resected in continuity with the ipsilateral neck lymphatics. When possible the entire primary should be resected in continuity with the neck dissection. A dissection that is not performed in continuity places the intervening lymphatics between the primary and cervical lymphatics at risk.

the cooperation of the tongue with surrounding structures such as the palate, teeth, and alveoli. There are very few comparative studies in English on articulation intelligibility in patients that do or do not have microvascular reconstruction. A comparison of the radial forearm free flap with the adjacent tongue dorsal flap demonstrated that for patients having a resection within the range of a hemitongue there was no difference between the two groups.¹⁰⁶ Patients with preservation of the tip or floor of the mouth had higher intelligibility. However, all patients in this study spoke Mandarin Chinese. Urken has shown that excellent sensory function can be maintained when a neural anastomosis with a radial forearm flap is performed; however, this is not commonly performed during microvascular flap reconstruction of the tongue.¹⁰⁷

A comprehensive discussion of management of the neck is presented later in the chapter. Because of the high incidence of oral tongue SCCs, the management of the neck for early tongue SCC will be briefly discussed here. The question as to how to handle the neck in patients with early stage (T1–T2) tongue SCC in the setting of no neck involvement has been addressed in all regions of the world and it is clear that elective surgical management of the neck should be performed.^{108,109} Patients should be treated with an ipsilateral neck dissection at the time of tongue resection. Such an approach leads to improved patient outcome and survival. A review of 380 patients with early stage (T1–T2N0) tongue SCCs demonstrated that patients treated with elective neck dissection have improved neck control rates and overall survival compared to patients that were simply observed.¹¹⁰ Primary radiotherapy has no role in the management of tongue cancer due to poor tumor control rates and the incidence of short- and long-term complications secondary to radiation therapy.¹¹¹

Maxillary gingiva

Presenting an evidence-based approach to the management of patients with maxillary gingival SCC is difficult given that few studies have documented associated metastatic rates and patient outcomes. In particular, there is limited information with regard to occult skip metastasis to the neck, bypassing the parapharyngeal and superficial buccal lymphatic systems.^{112,113} Maxillary gingival oral SCCs are rare compared to other oral cancer sites.¹¹⁴ SCC of this oral site poses complex management issues in terms of resection, reconstruction, and consideration for postoperative radiotherapy.

At initial presentation maxillary palatal, gingival, and alveolar squamous cell carcinomas manifest clinically detectable cervical metastasis at rates in the range 11.5–28.5%.^{115–118} The occult cervical metastatic rate ranges from 15.8–38.0%.^{115–118} Such metastatic rates are comparable to tongue, floor of mouth, and mandibular gingival squamous carcinomas where

the likelihood of occult neck disease reaches 30%.^{108,119–121} Montes and Schmidt have found an occult metastasis rate in patients with maxillary SCC to be 21.4%.¹¹⁸ Ultimately 50% of this patient group will develop regional or distant metastasis.¹¹⁸ The average time to regional failure in the clinically negative neck in patients with maxillary gingival SCC is 9.75 ± 6.37 months.¹¹⁸ Simental *et al.* report regional failures occurring an average of 10.4 months following surgery.¹¹⁶ When the few available studies addressing cervical failure in patients with maxillary SCCs are considered, the failure rate is as high as 50%.^{116–118}

In addition to proper management of cervical lymphatics, ablative accuracy is required for success in treating maxillary SCCs. Compared to all oral sites, maxillary alveolar carcinoma can be one of the most difficult areas to completely resect with proper margins.^{38,100} Maxillary alveolar specimens exhibit a high incidence of positive margins.³⁸ Woolgar *et al.* have proposed that the difficulty in obtaining oncologically proper margins is attributed to: (1) tumor spread along the vestibule; (2) unrecognized infiltration into the submucosal plane and buccinator muscle; and (3) surgeon's desire to limit the morbidity of resection.³⁸ Simental *et al.* have demonstrated a local failure rate of 29.2%.¹¹⁶ The area of greatest concern in terms of infiltration is the pterygoid fossa. Posterior access can be significantly improved with the Weber Fergusson approach. For large maxillary SCCs a combination of the Weber Fergusson and Trotter (subciliary) approaches can be used to increase access and allow for total removal (Fig. 34.9).

Local and regional failure both contribute to poor patient outcome. Patients with maxillary palatal, gin-

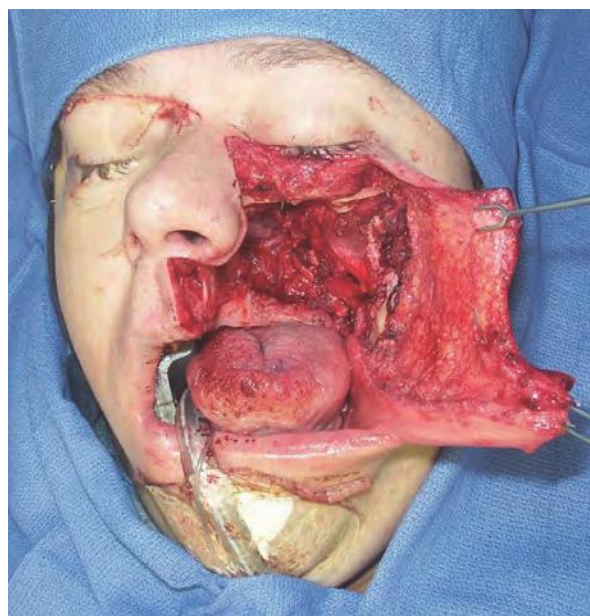


Fig. 34.9 Weber Fergusson approach with Trotter (subciliary) extension for resection of maxillary lesions. The approach can be used to increase access to the posterior pterygomaxillary region. The Trotter extension should only be used if it affords additional and necessary access. Most maxillary lesions can be removed with a transoral or Weber Fergusson approach alone.

gival, and alveolar carcinoma have an overall 5-year survival rate of 59–69%.^{115–117} Salvage success for regional failure of other oral site cancers has been reported to be between 29 and 60%.¹²² For maxillary SCCs the 34.6% regional failure rate is successfully managed in 66% of cases.¹¹⁶ There are no outcome data relative to salvage surgery for local recurrence of maxillary palatal, gingival, and alveolar squamous carcinomas. As with other oral sites, early local recurrence is a poor prognostic indicator.¹²²

Reconstruction of the maxillary defect following tumor resection continues to pose a challenge for the reconstructive oral and maxillofacial surgeon. Currently available maxillary reconstructive techniques include placement of a prosthetic obturator, local and regional flaps, and microvascular free flaps, each with specific indications and advantages depending on the ablative defect, the medical status of the patient, and the patient's prognosis.^{123–126} The meticulously fabricated maxillary obturator has a long history of effectively managing the functional, cosmetic, and psychological problems associated with a maxillectomy defect. However, the lack of retention, support, and stability are significant problems following extensive ablation of retentive maxillary anatomy. In a patient who is reconstructed with a maxillary obturator, a split-thickness skin graft, as well as removal of the inferior turbinate, should be performed in every case (Fig. 34.10). The split-thickness skin graft will aid with obturator insertion and removal, and resection of the inferior turbinate will significantly improve obturator retention.



Fig. 34.10 Maxillectomy defect reconstructed with a split-thickness skin graft. If the planned reconstruction of a maxillectomy defect involves a maxillary obturator then a split-thickness skin graft should always be placed. The skin graft will aid in the comfort of obturator insertion and removal. The inferior turbinate should also be removed to prevent displacement of the obturator.

There are few reconstructive options for patients with near total or total maxillectomy defects. Schmidt *et al.* have described the use of zygoma (zygomaticus) implants for retention of the maxillary defect in these patients.^{127,128} Zygoma implants were originally designed for reconstruction of the atrophic, edentulous maxilla;¹²⁹ however, the combination of zygoma and standard endosseous implants can be used to retain and support a maxillary obturator following extensive resection of the maxilla. Twenty-one percent of zygoma and 30% of standard endosseous implants fail in patients with extensive maxillary resections.^{127,128} A higher failure rate than is traditionally encountered should be expected, most likely due to radiation therapy that is often required in this patient group. Radiation has a clearly negative impact on the reparative capacity of bone.¹³⁰ The success rate for standard endosseous implants is lower in the irradiated oral and maxillofacial region.¹³¹ Parel *et al.* reported a success rate of only 61.1% for craniofacial implants placed in irradiated bone.¹³² The use of hyperbaric oxygen therapy in patients having implant reconstruction following radiation therapy is controversial.¹³³

The biomechanical forces placed on zygoma and standard implants following an extensive maxillectomy are significantly greater than the forces observed in a conventional implant reconstructive case. The quality and quantity of remaining bone available for osseointegration following an extensive maxillectomy are compromised. The lever arm placed on zygoma implants is significantly greater than the lever arm placed on standard endosseous implants. Resection of all palatal and alveolar bone might be required in a patient requiring a maxillectomy. In this case, the only bone available for integration of the zygoma implant is the zygomatic bone at the junction of the temporal and frontal process. Therefore, zygoma implants are at a 30–60° angle relative to the occlusal force and only have one site distal to the occlusal force available for integration. Because of these biomechanical requirements such cases should be over-engineered and as many zygoma and standard implants should be placed as possible (Fig. 34.11).

Zygoma implants should be placed at the time of resection given that most patients will require radiation therapy within 2–3 weeks of maxillary resection. Zygoma implants are significantly more difficult to place if not placed at the time of resection. Placement of zygoma implants at the time of resection requires meticulous planning by the maxillofacial prosthodontist and surgeon. Despite these problems patients reconstructed with this method have highly favorable speech and esthetics and are able to drink and eat without nasal leakage.

Microvascular free flap reconstruction can be used for patients requiring a maxillectomy. There are few studies available which have evaluated quality of life following maxillary reconstruction. There is a single quality-of-life study which evaluated the outcome of



Fig. 34.11 Reconstruction of a patient requiring near-total maxillectomy for SCC, utilizing bilateral double zygoma implants. There was no remaining bone for standard endosseous implants. Postoperative lateral cephalogram (left). An attempt was made to separate the four zygoma implants as much as possible in three planes of space. Postoperative posteroanterior cephalogram (right). The zygoma implants were placed to capture the maximum amount of bone at the junction of the temporal and frontal processes of the zygoma.

maxillary reconstruction with a maxillary prosthetic obturator.¹²⁴ Rogers *et al.* evaluated quality of life in patients requiring maxillectomy for oncologic reasons who were reconstructed with either a microvascular free flap or a maxillary obturator without implants.¹³⁴ The authors used eight quality-of-life questionnaires, including the Denture Satisfaction¹³⁵ and Obturator Functioning Scale.¹²⁴ There was no difference in quality of life between the patients reconstructed with a microvascular free flap to those reconstructed with a prosthetic obturator. A critical point of this study is that only eight of 18 patients reconstructed with a free flap were able to wear a conventional denture.¹³⁴

Mandibular gingiva

Similar to maxillary gingival SCCs, there are few dedicated studies that have looked at the biologic behavior of the mandibular gingival SCC.¹³⁶ Management of the mandibular SCC almost always requires resection of the mandible and reconstruction. Mandibular reconstruction has been discussed in detail in another chapter in this volume and will not be discussed here.

One of the most difficult aspects of managing mandibular gingival SCC is determining the degree of mandibular invasion, as well as the extent and method of mandibular resection.¹³⁷ For oral cavity SCC with the potential for mandibular involvement determination of the amount of bone involvement and how much mandible should be resected are critical for surgical planning and will directly impact on patient outcome. CT is often used as the study of choice for determining the degree of bone involvement; however, dental artifact can prohibit accurate interpretation of the critical CT views. Oral SCC on MRI has an intermediate signal on T1 weighted

images and appears similar to muscle. MRI views of the mandible can be difficult to interpret due to changes secondary to periodontal disease and other inflammatory changes. MRI can be helpful in assessing marrow involvement as well as possible perineural invasion. One problem with MRI is the potential to overpredict mandibular involvement, leading to resection of more mandible than is required for oncologic principles. A prospective study designed to determine the accuracy of available studies for predicting mandibular invasion showed that bone scintigraphy and MRI overpredict and CT and panoramic radiograph underpredict mandibular invasion.¹³⁸ Panoramic radiograph, MRI, and bone scintigraphy are associated with a high false-negative rate when used to predict mandibular invasion.¹³⁹ Periosteal stripping with direct inspection of the mandible has been proposed to determine both the presence and extent of tumor invasion of the mandible.¹³⁸ Brown recommends using clinical examination, periosteal stripping, and at least two imaging techniques that have complementary specificity and sensitivity when deciding to resect the mandible.¹⁴⁰ Cone-beam CT can be used in certain circumstances to help guide the resection margins.¹⁴¹ While standard oncologic principles should be followed the clinical findings and available studies should be carefully reviewed and surgical treatment tailored to the specific patient and the mandibular lesion.

For most mandibular gingival SCCs segmental resection with a 1 cm margin is the recommended approach. A marginal mandibulotomy can be used to resect a gingival SCC in very select cases;¹⁴² however, technical vigilance is required to not create a thin mandible which can easily fracture under the opposing forces of the pterygomasseteric sling and the suprahyoid muscle. It is often difficult to get an appropriate 1 cm margin and avoid thinning the mandible to the point of pathologic fracture (Fig. 34.12). Marginal mandibulectomies also make it difficult to perform an in-contiguity resection with the neck dissection placing the intervening lymphatics at risk for harboring occult metastasis. Reconstruction of a marginal mandibulectomy defect with a bone graft and ultimately endosseous dental implants is extremely prohibitive. The planned osteotomy should take into consideration the classic, and sometimes conflicting, work of McGregor, MacDonald, and Brown.^{143–147} Gingival SCC can invade the alveolar bone at the site of gingival attachment to the teeth and/or at the point of abutment of the carcinoma to the mandible. The mandibular osteotomy must be below the apices of the teeth.

Buccal mucosa

The buccal mucosa lines the oral cavity from the maxillary to the mandibular attached gingiva and progresses posteriorly to the pterygomandibular raphe. The buccal mucosa drains to the submental, subman-

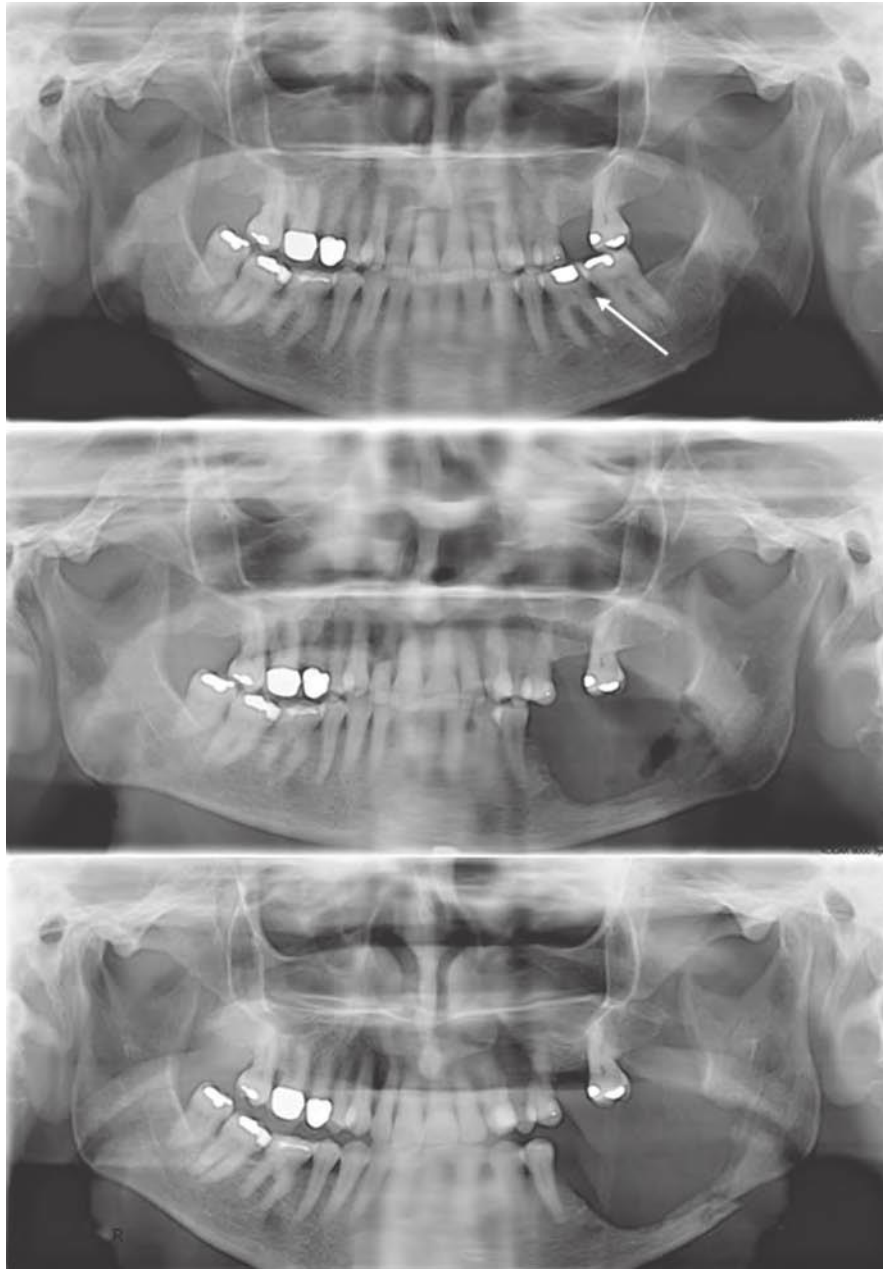


Fig. 34.12 Pathologic fracture in a patient following marginal mandibular resection for a mandibular gingival SCC. Preoperative panoramic radiograph (top) with the area of carcinoma pointed out (white arrow). A marginal mandibulectomy was planned. When a marginal mandibulectomy is planned the resection must be below the apices of the involved teeth. The postoperative panoramic radiograph shows the resection with the appropriate margin (middle). The inferior border is intact but thin. Two weeks following the operation the patient suffered a fracture (bottom). The patient required open reduction and rigid internal fixation.

dibular, and periparotid lymph nodes. Oral SCCs involving the buccal mucosa are typically aggressive in growth and invasion and this invasive pattern is reflected in the poor prognosis of patients with buccal mucosal SCC. The biologic behavior of these SCCs is reflective of the anatomy of the region which is rich in lymphatics and blood supply. The buccal space contains Stensen's duct, buccal fat, facial artery, and facial vein. Buccal mucosal SCCs are prone to buccal fat and muscle invasion, as well as Stensen's duct involvement. There is not an anatomic boundary to prevent spread of carcinoma. Local recurrence rates are very high, ranging from 45–100%.^{148–150} Locoregional failure leads to poor survival.¹⁴⁸ Sieckza

et al. demonstrated that, even for low T-stage carcinomas, negative margins are not adequate predictors of local recurrence which was 56% in this study.¹⁴⁹ Patients with close or positive margins had a 66% local failure rate as compared with 52% when surgical margins were negative (≥ 5 mm following tissue fixation).¹⁴⁹ Patients who had T1 or T2 SCCs had a 40% local failure rate with surgical resection alone. The available studies favor the use of postoperative radiotherapy to improve local control. As discussed in this chapter postoperative radiotherapy is reserved for oral SCC patients that have close (≤ 5 mm following tissue fixation) or positive margins or have evidence of cervical metastasis. There is also a high

tendency towards regional failure.^{148,149} Evaluation of the degree of infiltration can be extremely difficult in patients with buccal mucosal SCCs. CT or MRI is often not helpful given that the buccal mucosa collapses against the alveolus. One method to improve interpretation of the selected scan is to place gauze between the buccal mucosa and alveolus to separate the two structures and allow for evaluation (Fig. 34.13).

Reconstructive options for buccal mucosal SCCs will depend on the size of the defect. The alternatives include a split-thickness skin graft, local flaps including the nasolabial flap and buccal fat, and microvascular reconstruction. For posterior buccal mucosal SCCs in the region of the pterygomandibular raphe, patients will often experience postoperative trismus which is particularly pronounced when radiation therapy is given. Patients should be encouraged to begin jaw-opening exercises as soon as possible following surgery. They should be advised to continue through radiation therapy. Patients will often experience pain with the jaw-opening exercises and analgesics should be taken before the exercises.

Floor of the mouth

Squamous cell carcinoma of the floor of the mouth is associated with a high rate of local recurrence¹⁵¹ and high rates of cervical metastasis, even when the primary is small.¹⁵² Similar to other oral cavity SCC primaries a neck dissection is indicated. Unless the lesion is truly a lateral lesion then a bilateral neck dissection is indicated. Involvement of the sublingual gland is common. Obtaining a 1 cm margin on the deep margin can be difficult without resecting the primary in continuity with the neck dissection. A mandibulotomy significantly improves access and allows for resection of the specimen in continuity with the neck (Fig. 34.14). A parasagittal mandibulotomy can be used to resect floor of mouth, lateral tongue, base of tongue, maxillary, parapharyngeal, and skull base tumors.¹⁵³ If attention is given to strict control of the occlusion, which can be aided by the use of a lingual splint, morbidity associated with the mandibulotomy is minimized.¹⁵³

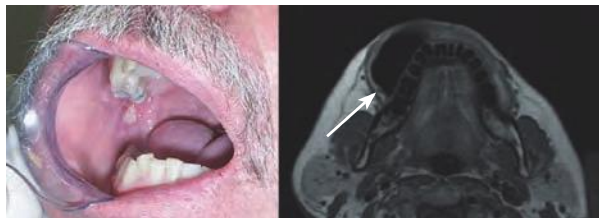


Fig. 34.13 Imaging technique to improve visualization of buccal mucosal lesions. A biopsy-proven buccal mucosal SCC is pictured on the left. CT or MRI imaging of these lesions can be difficult because when the patient closes the buccal mucosa collapses against the teeth and alveolus. Gauze can be placed between the buccal mucosa and teeth during the scan (right) which improves visualization of the lesion (arrow).

Reconstruction of the floor of mouth defect following resection should be based on the following: size of the defect, whether the defect communicates with the neck, planned postoperative radiation therapy, and health of the patient. If there is communication between the oral cavity and neck, the patient's health will tolerate a prolonged general anesthetic, and postoperative radiation therapy is planned, then a microvascular free flap is likely the treatment of choice. Smaller defects where the mylohyoid muscle is maintained can be reconstructed with a split-thickness skin graft and temporary bolster. The inferiorly based nasolabial flap is an excellent alternative to the microvascular free flap. Rose initially described the use of the one-stage arterialized nasolabial island flap for floor of mouth reconstruction.¹⁵⁴ The skin portion of the flap is centered on the nasolabial fold and extends from the medial canthal area to 2 cm superior to the mandibular margin. The nasolabial flap can be harvested to contain the subcutaneous tissue and facial muscles with the associated vessels. Harvesting of the associated vessels made the flap considerably more reliable than the subcutaneous flap. Harvesting of the underlying muscles added to the mobility of the flap. Hagan and Walker formally described the nasolabial musculocutaneous flap.¹⁵⁵ These authors reported that in 15 cases with advancements up to 5 cm there were no cases of necrosis. Bilateral nasolabial flaps can be harvested to cover large floor of mouth defects measuring up to approximately 5 cm in anteroposterior dimension.¹⁵⁶ The advantages of the nasolabial flap for reconstruction of floor of mouth defects include the large amount of tissue the flaps provide and the cosmetic result following closure of the donor site. Potential disadvantages include hair growth from the flap in patients who will not receive radiation therapy and the fact that the flap pedicle must cross over the occlusal table of dentate patients. The flaps should be raised superficial to the facial muscles as two triangular flaps. The flaps can then be tunneled through the cheek and sutured one in front of the other in the floor of the mouth. The flaps are sutured to each other and the margin of the defect. The donor site is closed except for a small area at the base to prevent stricture of the pedicle. The pedicle can be divided and the base closed 2–3 weeks later. With healing of the flaps, the floor of mouth is

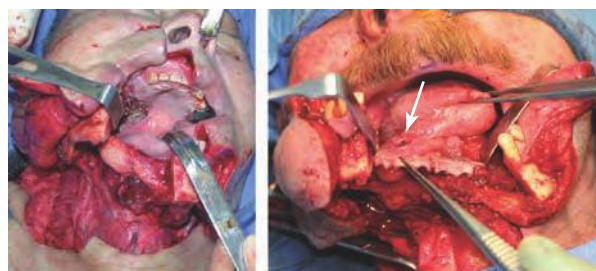


Fig. 34.14 A mandibulotomy can improve access for resection. The mandibulotomy provides surgical access to maxillary (left), tongue, base of tongue, and floor of mouth (right, arrow) SCCs.

flat enough to allow placement of a denture. Ioannides *et al.* reported on the use of nasolabial flaps for reconstruction of the floor of mouth in 16 cases; complications were observed in four cases. The complications included scarring, flap dehiscence in two cases, and a bulky flap, preventing placement of a denture.¹⁵⁶

Lip

Lower lip SCC has similar outcome with surgical resection and radiation therapy. Radiation therapy can be particularly helpful with extensive carcinomas that would require resection of the entire lower lip. However, consideration must be given for mandibular involvement which would prohibit the use of radiation therapy. Moreover, radiation therapy will leave the patient with the life-long effects of radiation therapy. Lower lip SCC can be treated with electrons to treat to very high doses and a superficial depth. Lower lip SCCs are generally ultraviolet light induced. If surgery is chosen as the treatment resection margins should be 1 cm. The primary function of the lower lip function is control of saliva and prevention of sialorrhea. The lower lip also functions for articulation. If possible symmetric coverage of the teeth should be maintained. Restoration of cosmesis and function is best achieved with the use of local flaps.¹⁵⁷ The reconstructive plan must include the skin, lip, and buccal mucosa. Many methods are available to reconstruct the lips following resection. The Bernard flap, which can utilize the buccal mucosa for replacement of the wet portion of the lower lip, is an excellent method to reconstruct large defects of the lower lip (Figs 34.15 and 34.16). For extensive resections a microvascular free flap will be required; however, the final cosmetic result is often compromised.

Management of the neck

In this section an evidence-based approach will be described for managing the neck in patients with oral cavity SCC. A description of the surgical details of neck dissection is not within the scope of this chapter. Detailed, surgeon-oriented texts and articles are available that provide a detailed description of the operative technique for neck dissection.¹⁵⁸ An excellent text describing avoidance and management of complications following neck dissection can be found in Gavilán *et al.*¹⁵⁹ Surgery is the treatment of choice for almost every oral cavity SCC. The data overwhelmingly support elective neck dissection for even small T1 lesions given the high rate of occult metastasis with oral cavity SCCs. The goals of treating the N0 neck are twofold: (1) to therapeutically remove the lymph nodes that might harbor carcinoma; and (2) to histopathologically review the surgically resected neck specimen leading to accurate staging which would potentially warrant adjuvant treatment leading to improved cancer control. The “wait and see” practice is the opposing approach to the elective neck dissection and refers to the practice of closely following patients who are N0 at the time of surgery rather than performing a neck dissection. With this approach once a patient fails in the neck a salvage of the patient is attempted. “Wait and see” approaches for the N0 neck have been associated with high failure rates. Salvage rates for the failed neck in patients with oral SCC are extremely poor as will be discussed below.

Managing the N0 neck

Almost all patients who die from oral cancer will fail because of uncontrolled local recurrence or failure in

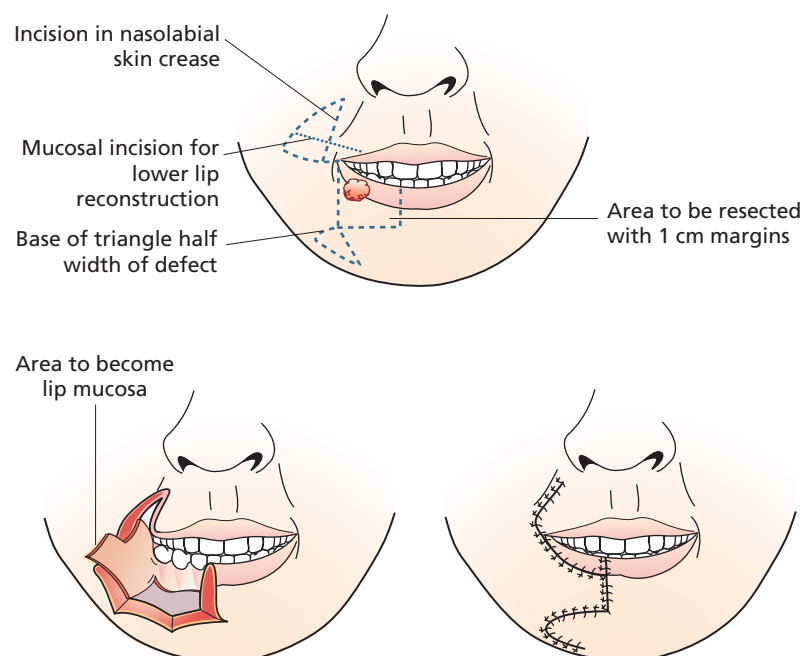


Fig. 34.15 Drawing showing Bernard flap reconstruction of a lower lip following resection of a lower lip SCC. Modified from Closmann *et al.* *J Oral Maxillofac Surg* 2006; 64: 367–74.¹⁵⁷

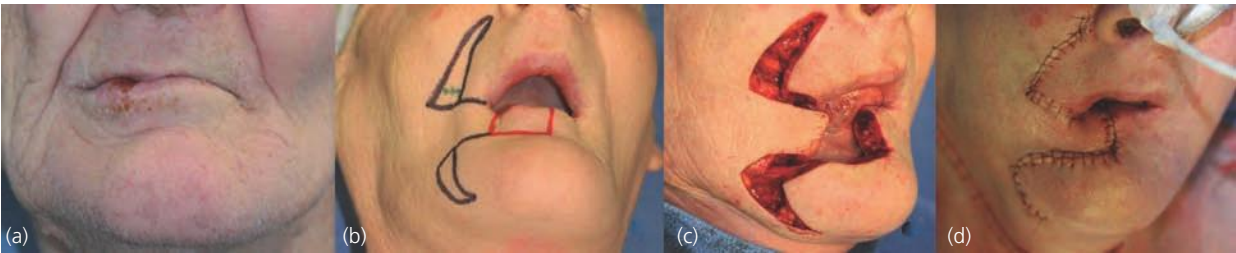


Fig. 34.16 Resection of lower lip using a Bernard flap. (a) SCC of the right lower lip requiring resection of approximately 60% of the lower lip to achieve a 1 cm margin. (b) The planned reconstruction is a unilateral Bernard advancement flap. The area to be resected is drawn in red. Burow's triangles are marked in black. The green line represents the area of buccal mucosa to be used to reconstruct the wet portion of the lower lip. (c) Full-thickness resection of the lesion with 1 cm margins is performed. Burow's triangles of soft tissue are removed, incising through skin, subcutaneous tissue, fat and muscle leaving the mucosa intact. Care is taken to place the medial vertical incision of the upper triangle in the nasolabial fold, and the lower medial vertical incision in the melolabial fold. The base of each triangle is approximately one half the length of the defect. A horizontal incision is then placed lateral to the commissure also one half the length of the defect. This incision is to the level of the mucosa. The mucosa is then incised intraorally 1 cm superior to the horizontal incision to create a mucosal flap for lip mucosal reconstruction. (d) Bernard flap sutured in place in tension-free manner.

the neck. Indeed, regional metastasis reduces the 5-year survival rate by half.^{28,160,161} The treatment of the clinically positive neck is straightforward and the purpose is to extirpate the cervical fibroadipose and lymphatic tissues and remove associated structures.^{162–166} Management of the neck when metastasis is not clinically evident (N0) is less clear, given the unpredictable propensity of oral SCC for occult neck metastasis and the associated grave prognosis.^{10,122,167} The decision regarding the N0 neck has been debated extensively.^{168–170} Occult metastatic rates for oral SCC are very high and range from 20–45% for T1 tongue SCCs (Table 34.1).^{10,69,121,171–174} It is clear that when neck metastasis is detected after initial surgery, surgical salvage rates are poor.^{122,175} The downside of performing an elective neck dissection is that it might subject the patient to an unnecessary major surgery and its associated risks, particularly shoulder syndrome.^{94,160,168,169,176} For oral cavity SCC a selective neck dissection (SND) generally refers to the removal of levels I, II, and III with preservation of the sternocleidomastoid muscle, the internal jugular vein and the spinal accessory nerve. A modified radical neck dissection refers to the removal of one of the key structures preserved with the SND and inclusion of level IV.

An elective neck dissection (i.e. performing a neck dissection when there is no clinical or radiographic

evidence of neck metastasis) is often recommended if the chance for occult metastasis is greater than 20%. This threshold is based on the commonly cited study by Weiss *et al.* which proposes that a 20% risk of occult metastasis should be the threshold for treatment of the neck.¹⁷⁷ An intimate understanding of this landmark study is essential when discussing the management of the N0 neck. In this study, Weiss *et al.* performed a computer-assisted mathematical analysis of the decisions and associated outcomes involved with management of the N0 neck, weighing the outcomes with their likelihood of occurrence and relative cost and benefit to the patient.¹⁷⁷ The authors created a decision tree that begins with the decision to operate, to irradiate, or to closely observe with the associated possible outcomes and subsequent necessary decisions. The most distal branches of the decision tree were given probabilities for occurrence based on data from a review of seven clinical series published from 1971–1984.^{166,178–183} Each of the outcomes, including cure, death, cure with surgery, cure with radiation, and cure with salvage, were weighted based on desirability. Desirability was determined by three head and neck surgeons using a “time tradeoff method”. The “time trade off method” refers to how much time of life these surgeons would sacrifice to have better function. Using this methodology, Weiss *et al.* concluded that the benefits outweigh the costs in prophylactically treating the N0 neck when the risk of occult metastasis exceeds 20%. The threshold of 20% has been used extensively in subsequent studies to identify clinical factors, such as stage, tumor thickness, depth of invasion, and histologic characteristics of the invasive front, as the cutoff to make the decision regarding management of the neck. The limitation of the analysis by Weiss *et al.* is that patients’ perception of quality of life is not considered. The work of Weiss *et al.* is an important component in any algorithm that is being used to make the decision on prescribing elective neck dissection. Only one prospective series has attempted to evaluate the clinical utility of the 20% risk for occult metastasis as a

Table 34.1 Studies on occult metastases in T1 tongue SCCs

Author	Date	Site	Occult neck metastases (%)
Spiro & Strong ^{34,35}	1971–1974	Tongue	29.4
Lee & Litton ³³	1972	Tongue	24
Whitehurst & Droulias ³²	1977	Tongue	24
Johnson <i>et al.</i> ³¹	1980	Tongue	36
Ho <i>et al.</i> ³⁰	1992	Tongue	46
Yuen <i>et al.</i> ²⁹	1999	Tongue	21

threshold for elective treatment of the neck.¹⁷¹ O'Brien *et al.* reported on the application of Weiss *et al.*'s decision tree model using clinical criteria, specifically tumor site, tumor stage, and the necessity of accessing the neck in resecting the primary tumor (i.e. transcervical approach to the primary SCC), to decide when to electively treat the neck.¹⁷¹ In their series of 162 patients with oral or oropharyngeal SCC and N0 necks, 58 patients were observed and 104 patients received elective therapy in the form of a SND, radiation therapy, or a combination of the two. The decision to perform elective treatment of the neck was based on the clinical factors mentioned above, using 20% risk as the threshold for treatment. In their elective treatment group, 30% of the neck dissection specimens had evidence of metastatic disease, and 4% of the group, all of which had positive nodes on initial neck dissection, developed unsalvageable neck recurrences.

Elective neck dissection versus “wait and see” for the N0 patient

There are three prospective randomized trials that have compared the “wait and see” approach to the elective neck dissection and only one of them evaluated the use of the SND.^{166,184,185} Vandenbrouck *et al.* evaluated 75 patients, with primary SCC of the oral cavity and no evidence of nodal metastasis, that were randomly assigned to have either resection of the primary tumor without neck dissection or resection with a traditional radical neck dissection (RND).¹⁶⁶ The patients in both of these groups were kept on a monthly follow-up schedule for 3 years. In their series, they found that 49% had occult metastasis in the elective neck dissection group and that 47% of the patients in the “wait and see” group ultimately developed neck involvement and subsequently required salvage RND and radiation therapy. In the “wait and see” group, there was a higher incidence of extracapsular spread. Fakih *et al.* published a study of 70

patients, with T1 and T2 N0 oral tongue SCC, who were randomized into one of two groups: resection of the primary without elective neck dissection and resection of the primary and a RND.¹⁸⁵ These authors found that 57% of patients in the “wait and see” group developed neck metastasis and 47% of the patients in the RND group had histologic evidence of occult metastasis. At 1 year the disease-free survival (DFS) of the “wait and see” group was 52% while the DFS of the latter group was 63%, although this was not statistically significant.

The only prospective, randomized, controlled trial evaluating SND of levels I, II, and III as elective treatment for the N0 neck as compared to the “wait and see” approach was published in 1994 by Kligerman *et al.*¹⁸⁴ Their series included 67 patients with oral SCC and N0 necks, and they were randomly assigned to either resection of the primary alone or in conjunction with a SND of levels I, II, and III. These patients were followed for a minimum of 3 years postoperatively. The investigators found a rate of 21% occult metastasis in the elective SND group and a 39% rate of recurrence in the neck in the “wait and see” group. Critical to this study is the finding that only 27% were salvageable from the group of patients who developed neck recurrences. Kligerman *et al.* also reported that overall survival at 3 years was significantly improved in the elective neck dissection group.

Many retrospective, non-randomized studies have also reported on the benefits of the SND in treating the neck compared to the “wait and see” strategy.^{78,175,187,188,190} These studies are summarized in Table 34.2. Keski-Santti *et al.* found a significant improvement in survival despite more advanced primary tumors in the elective treatment groups, with 35% of patients in the observation group developing neck recurrences and only a 33% salvage rate.¹⁸⁸ Similarly, Capote *et al.* found an 8% neck recurrence rate in the elective neck dissection group versus a 26.8% rate in the observation group with a 5-year survival rate of 91.7% and 77%, respectively.¹⁷⁵ Again,

Table 34.2 Summary of data from retrospective observational studies on elective treatment of the N0 neck

Study	Elective therapy	Total patients	Occult metastasis on END ^a	Regional involvement on observation	Salvage ^b	Rate of DFS ^c
Khafif <i>et al.</i> 1991 ¹⁹⁰	RND	590	23%	19%		
Nieuwenhuis <i>et al.</i> 2002 ⁷⁸	N/A	161	N/A	21%	(79%)	79% at 12 months
Duvvuri <i>et al.</i> 2004 ¹⁸⁷	SND	359	23% (8%)	27%	Not reported	66% (54%) at 3 years
Keski-Santti <i>et al.</i> 2006 ¹⁸⁸	SND, RT or SND + RT	80	34% (13%)	24%	11% (47%) ^d	82% (81%) at 3 years
Capote <i>et al.</i> 2007 ¹⁷⁵	END	154	Not reported (8%)	26.8%	32% ^e	92.5% (71.2%) at 5 years

^a END, elective neck dissection. Numbers in parentheses indicates neck recurrences following END.

^b Salvage rates in neck recurrences only, unless otherwise noted. Numbers in parentheses indicate salvage rates for neck recurrences in observation group. Minimum follow-up time shown.

^c Numbers in parentheses indicate DFS in observation group.

^d Overall salvage rate for local and regional recurrences.

^e Neck salvage rate for both observation and END groups.

success of salvage therapy was low at 32%. In another non-randomized retrospective review of 359 patients, Duvvuri *et al.* found a 27% regional failure rate compared to 8% in the elective neck dissection group, although there was no difference in survival at 5 years.¹⁸⁷ Elective radiation therapy has also been shown to improve outcomes when used to manage the N0 neck.¹⁸² In a retrospective review of 354 oral cancer patients, Ord *et al.* reported a much higher neck salvage rate than the studies listed above at 50%.¹²²

A few studies have concluded that the “wait and see” approach to the neck is acceptable.^{78,190–192} Khafif *et al.*, in a retrospective analysis of 590 patients, found that patients with occult metastasis who received an elective RND did not gain a survival benefit when compared to those patients who were observed and subsequently developed clinically apparent nodal involvement with subsequent salvage RND.¹⁹⁰ Khafif *et al.* made the incorrect assumption that pathologically N0 (pN0) necks did not harbor occult metastasis. However, as will be discussed below and as is evident in the 7% neck recurrence rate among patients with pN0 necks in their study, this assumption is not accurate. When the elective RND group was analyzed, with both pN+ (pathologically positive for nodal involvement) and pN0 patients included, their survival was significantly improved over the salvage rate in the observation group, 68% with DFS compared to 49% with DFS, respectively. Khafif *et al.*’s recommendation not to electively treat the neck was based on the assumption that elective treatment would be with either RND or radiation therapy, both of which carry significant morbidity. Layland *et al.*, in a retrospective study of 891 patients with oral SCC and N0/N+ necks, showed that DFS was equivalent whether treatment of the neck was given electively or after nodal disease became apparent.¹⁹¹ One criticism of this study is that the observation arm has a higher percentage of early T1 disease as compared to T2–T4 disease, a discrepancy that was not statistically addressed and could have confounded the outcome and the final recommendation.

In consideration of each of these studies the reader must keep in mind that histopathologic review of nodes is not infallible. In most clinical pathology laboratories lymph nodes are bisected and evaluated for metastasis. Several studies have found a high prevalence of micrometastases, that is foci of tumor within lymph nodes smaller than 3 mm, when lymph nodes were serially examined with significantly thinner sections.^{193,194} When lymph nodes were evaluated in this manner micrometastases were identified in 16–25% of previously pathologically staged N0 necks. Therefore, it is likely that previous studies on the prevalence of occult metastasis discovered by elective neck dissections grossly underestimated the actual prevalence of nodal involvement, and that many of these neck dissections are actually therapeutic. This may explain the improved survival rates seen in elective treatment groups in several studies.^{184,188}

Quality of life for patients having a neck dissection

Since the time of Weiss *et al.*’s publication, the use of the SND has supplanted RND as the elective treatment of choice for the neck.^{163,164} Quality-of-life studies using general wellness and shoulder function specific assessments have demonstrated that the SND, when restricted to levels I–III, is not a significant detriment to quality of life, with outcomes in neck dissection patients nearly equal to those in patients who did not receive neck dissections.^{195–199} Patients who are treated with a radical neck dissection or a modified radical neck dissection (MRND) suffer a significant impact on quality of life including long-term dysfunction, disfigurement, and increased shoulder pain.²⁰⁰ Taylor *et al.* used the Neck Dissection Impairment Index to show that the greatest factors that affected quality of life, in descending order of significance, were age and weight, radiation therapy, and the extent of neck dissection.¹⁹⁶ Similarly, Rogers *et al.*, using the University of Washington Quality of Life scale coupled with the Neck Dissection Impairment Index and Shoulder Disability Questionnaire, found that patients treated with MRND or RND had the worst scores while patients who had a SND of levels I–III had similar scores to those patients who had no neck dissection.¹⁹⁵ Therefore, with the relatively low morbidity of SND compared to RND, the desirability values in the decision tree would certainly be different. Quality of life must also be considered for the patient who is followed using the “wait and see” approach and subsequently develops neck metastasis. Salvageable patients will be obligated to undergo a RND, possibly with chemo-radiation therapy.^{165,186,201,202} Thus, when the “wait and see” approach is used, failure will result in decreased survival and quality of life. Of course, elective treatment is only advisable if it effectively prevents recurrences and decreases cancer-related mortality. Most studies on SND demonstrate that it is efficacious in preventing neck recurrence and controlling occult neck metastasis.^{28,184}

Radiation therapy should not be used to treat the N0 neck

Radiation therapy has been proposed as an efficacious method to manage the N0 neck.¹⁸² The advantages of surgery for management of the N0 neck in patients with oral SCC are clear. While radiation therapy has been shown to be effective in treating oropharyngeal, nasopharyngeal, hypopharyngeal, and laryngeal SCC, it has compared unfavorably to surgery as a primary therapy for oral SCC due to its considerable morbidity and poor cancer control rates.^{203,204} Toxicities of radiation therapy include mucositis, xerostomia, and its dental sequelae, erythematous skin changes, subcutaneous fibrosis, carotid artery stenosis, and osteoradionecrosis.

Furthermore, speech and the ability to swallow are often more severely affected with radiation therapy. These adverse reactions lead to a protracted recovery that likely contributes to the lower quality-of-life scores that are correlated with radiation therapy.^{196,205,206} As surgery is the treatment of choice for primary oral SCC tumors and SND is better tolerated by patients when compared to radiation therapy, it is logical to prescribe SND rather than radiation therapy for the N0 neck in patients with oral SCC.

Despite treatment with curative intent, oral SCC has a relatively high recurrence rate. The use of radiation therapy as a primary therapy precludes its future use as adjunctive or salvage therapy. Operating in an irradiated field is technically challenging, making the salvage surgery more difficult with increased risk for complications. Radiation therapy should therefore be reserved for adjunctive or second line therapy in patients with oral SCC.

One of the prime reasons to choose surgery over radiation therapy for treatment of the N0 neck is the staging information that histopathologic review of the neck specimen provides.^{119,207} Although there are limitations to the accuracy of lymph node staging, as mentioned above, the histopathologic review of the neck is invaluable. Identification of extracapsular spread (ECS) and multiple occult nodes can significantly alter prognosis and signals the need for adjuvant treatment.^{167,173}

Extent of neck dissection for the N0 neck

When oral cancer metastasizes to the cervical region it metastasizes predictably to certain lymph node

groups. Since Crile's description of the neck dissection in 1906, treatment of the neck has evolved to become less radical and more conservative, including the sparing of non-lymphatic structures such as the spinal accessory nerve, the sternocleidomastoid muscle, and the internal jugular vein. Also, the number of lymph node levels that are dissected has decreased with no change in outcome.^{163,179,208} Limiting the dissection to the node levels and structures that are at greatest risk has led to a marked reduction in postoperative morbidity, particularly with avoiding sacrifice of the spinal accessory nerve and avoiding trauma to this nerve by foregoing dissection of the posterior triangle.¹⁹⁸ For the N0 oral SCC patient levels I–III should be removed.¹⁶⁴ Fig. 34.17 illustrates the levels of neck as classified in 2002 by Robbins *et al.*²⁰⁹

The anatomic limits of the SND are based on studies that have shown that oral SCC follows predictable patterns of lymphatic metastasis.^{28,164,210–213} Lindberg's classic study of 1972 demonstrated that oral cavity SCCs metastasize to the submandibular triangle, the submental triangle, the upper jugular chain, and the mid-jugular chain of lymph nodes.²¹² Lindberg also described the prevalence of "skip metastases" in his series, where metastatic tumor could "skip" the upper echelon of nodes, i.e. the jugulodigastric or submandibular nodes, and be found in the mid-jugular chain. These skip metastases were rarely found in the lower jugular chain (level IV) or in the posterior triangle of the neck (level V). Several subsequent studies have confirmed Lindberg's observations. Shah *et al.* found in a series of 1119 RNDs for primary oral SCC tumors, level V nodes were never involved when levels I–IV were negative.¹⁶⁴ Davidson

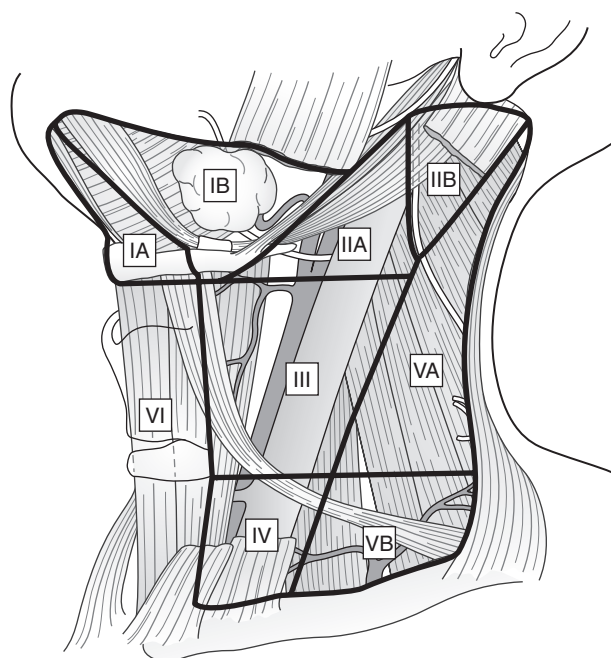


Fig. 34.17 Illustration of the levels and sub-levels of the neck. Notice that level II is divided into IIA and IIB by the spinal accessory nerve and level V is divided by a horizontal plane from the inferior border of the cricoid arch into VA and VB.

et al., in a cohort of 1123 patients with head and neck SCC, reported that only 1% of their patients staged clinically as N0 had metastasis to level V.²¹⁰ In 2004, Dias *et al.* published a series of 339 patients in which only 1.5% of patients with N0 necks had skip metastasis to level IV, and no patients had skip metastases to level V.²¹¹ The efficacy of SND relative to RND has been studied and the two modes of dissection have similar efficacy.^{28,169,174,184,187,214}

The reclassification of the neck levels by Robbins *et al.* divided level II into IIA and IIB using the spinal accessory nerve as the dividing line, and level V into VA and VB using a horizontal plane that crosses the inferior border of the anterior cricoid arch as a dividing line.²⁰⁹ The purpose of this reclassification was to distinguish sub-levels IIB and VA for potential preservation. The rationale behind this decision was based on the premise that IIB nodes (also known as the supraretrospinal triangle, supraspinal accessory lymph node pad, or the submuscular recess) were rarely involved in occult metastasis and that dissection of those nodes may lead to traction of the spinal accessory nerve and subsequent shoulder dysfunction.²⁰⁰ Several studies have confirmed that metastases to level IIB are relatively rare. Silverman *et al.* only found 1.6% of 74 patients with IIB metastasis.²¹⁵ Elshiekh *et al.* found no positive IIB nodes using reverse transcriptase polymerase chain reaction (RT-PCR) molecular techniques, except in SCC of the oral tongue.²¹⁶ In this sub-site, they found 10% of patients had molecular evidence of tumor cells in IIB nodes. Lim *et al.* also only found 5% of metastasis to this level in 74 of their patients.²¹⁷ Level IIB should generally be included in the neck dissection. Metastasis to this sublevel does occur and there are no quality-of-life studies demonstrating the benefits of preservation of level IIB. If neck salvage is required completion neck dissection to include level IIB in the setting of recurrence, previous surgery, and possible radiation will be much more difficult and place the spinal accessory nerve at increased risk than if level IIB had been dissected at the time of the initial surgery. Dissection of level IIB is therefore recommended at the time of elective neck dissection (Fig. 34.18).

Contralateral occult metastasis is also observed, particularly in tumors that encroach on the midline.²¹² It is recommended that a SND be prescribed bilaterally for patients with oral SCC that violates the midline.²⁰⁹ This includes any SCC tumor that requires a wide resection that crosses the midline in order to achieve a 1 cm margin.

Sentinel node biopsy

Perhaps the best example of the shift towards ultra-selectivity in management of the N0 neck is the application of the sentinel node biopsy technique. The sentinel lymph node biopsy technique involves injection of radiolabeled tracer around the periphery of the tumor, allowing the tracer to drain via the lymphatic system to the first echelon of nodes. The sentinel node or nodes are then delineated using a gamma probe, followed by the surgical removal of the node(s) in a manner that is oncologically safe. These nodes are then examined by frozen section. If micrometastases are identified on frozen section, the patient receives a SND. Postoperatively, the nodes from the permanent specimen are then examined thoroughly by serial sectioning, as opposed to bisectioning, which is the technique typically used to sample lymph nodes for histopathologic review. If micrometastases are located in the permanent serial sections, the patient is brought back to the operating room for a conventional SND if it has not already been performed.²¹⁸

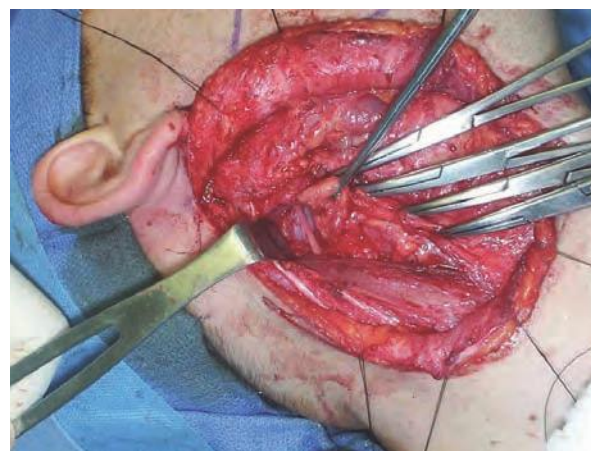


Fig. 34.18 Dissection of neck level IIB. Careful dissection of level IIB with preservation of the spinal accessory nerve should be performed when performing a selective neck dissection. While level IIB is an uncommon site of metastasis from an oral cavity, primary surgical salvage of neck failure involving level IIB is technically arduous and often unsuccessful.

This technique, which has been used effectively in the treatment of melanoma and breast cancer, has been advocated for treatment of the N0 neck in oral SCC. Preliminary results from a multicenter trial in 2004 were favorable.²¹⁹ In addition, a recent clinical trial published by Stoeckli *et al.* demonstrated that the use of sentinel node biopsy had very high sensitivity and specificity for detecting occult metastases, with a false-negative rate of only 6%.²¹⁸ In addition, in nine patients (13%) with laterally located floor of mouth and oral tongue SCC, the use of sentinel node biopsy identified drainage to contralateral nodes and level IV nodes, regions that would not have been addressed by a conventional SND. There have also been some concerns regarding the use of sentinel node biopsy in the management of the N0 neck. The radiolabeled tracer which is injected into the oral SCC tumor, especially tumors involving the floor of mouth and oral tongue, would obscure gamma probe readings in the first echelon nodes due to their proximity to the primary tumor, negating any benefit from the exercise. Stoeckli *et al.* attempted to circumvent this problem by going against conventional practice and excising the primary tumor first, before the sentinel nodes

were marked out and addressed.²¹⁸ Another criticism of the sentinel lymph node technique is that if micro-metastasis is detected later after serial sectioning of the permanent specimen, the patient would be required to undergo a second surgery. Knowing the high rates of occult metastasis, this would likely result in 20–45% of patients requiring second surgeries. The studies that have been published evaluating the efficacy of this technique are small. More studies will need to be done before the efficacy of sentinel node biopsy can be adequately compared to the conventional SND with its attendant low morbidity. Removing level I and II nodes is not significantly less morbid than removing levels I–III; however, performing two separate operations instead of one certainly is more morbid. Although the sentinel node biopsy warrants further investigation, it is still too early to recommend it as a treatment for the N0 neck.

Managing the N+ neck

For the patient with clinically palpable nodes a fine-needle aspiration can be performed to determine if there is malignant involvement of the neck. If neck involvement is either suspected or confirmed then a MRND with preservation of the spinal accessory nerve should be performed. Appropriate muscle flap coverage of the carotid artery with a scalene muscle should be considered, especially if the patient is to receive postoperative radiation therapy. Preservation of the internal jugular might be required for free flap reconstruction. Sacrificing the spinal accessory nerve should be reserved for cases where there is direct involvement of the nerve with carcinoma. Shoulder dysfunction is a common complaint following neck dissection. Complications following neck dissection, while rare, include hematoma, lymphalocele, ear numbness, and shoulder dysfunction. Given that ear numbness is one of the primary complaints following neck dissection, the greater auricular nerve should be preserved if possible.

There is no standard in terms of number of lymph nodes harvested for a neck dissection. The count will depend on the levels dissected and the attention to detail of the surgeon as well as the pathologist. What must be considered when reviewing the pathologic status of lymph nodes is that in many centers the lymph nodes are only bisected and the chance of missing metastasis is high.

Postsurgical management

Histopathologic evaluation of the specimen

Following the resection the primary oral SCC specimen and neck should be pinned out and oriented for the pathologist. Such orientation will help with pathologic evaluation of the margins, as well as the

neck (Fig. 34.19). Following surgery the pathology report should be carefully reviewed and the critical features to evaluate are marginal status, perineural infiltration, lymphovascular involvement, lymph nodes involved, and extracapsular spread of involved nodes. Consideration should be given to margin shrinkage that occurs with processing.¹⁰⁰ The surgeon is sometimes faced with the uncomfortable situation of how to handle a truly positive margin (carcinoma at the resection margin). Further resection will often produce a negative pathology report.

The decision for postoperative radiotherapy will be based on both margin and neck status. As discussed previously there is not a clear definition among clinicians, pathologists and investigators regarding what is meant by “clear” and “close” margins, as well as the distinction between mucosal and deep margins.^{38,90,95,97} While surgeons typically try to resect oral cancer with a 1 cm margin of clinically normal tissue, pathologic evaluation of the specimen almost always demonstrates significantly less normal tissue surrounding the cancer. This reduced margin can partially be explained by tissue shrinkage that occurs with specimen processing. A study in dogs demonstrated that 30–50% tissue shrinkage (from clinical to histologic margin) occurs with specimen processing.²²⁰ Another issue that can compound the interpretation of margins and studies evaluating margins in oral cancer is the site of the primary cancer. Woolgar *et al.* has demonstrated that the oral cancer sub-site significantly impacted on the status of the margins.³⁸ Using the definition of 1 mm margin as an involved margin these authors showed that the percentage of involved margins was highest in maxillary alveolar (45%), retromolar (38%), buccal mucosa (33%), mandibular alveolus (17%), and tongue SCC (11%).³⁸ Not surprisingly, the incidence of involved margins increased with the tumor T stage in this study.³⁸ Even when histologic margins are clear, recurrences occur and are most likely secondary to

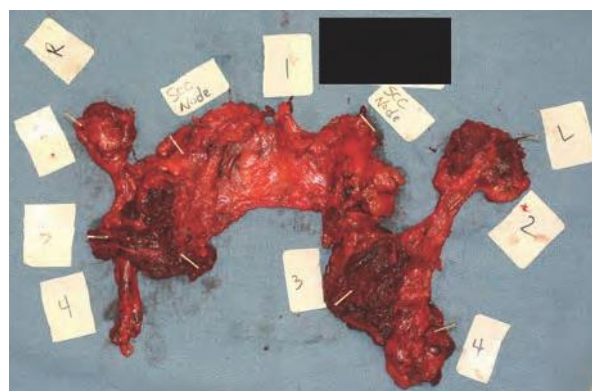


Fig. 34.19 Bilateral resected neck specimen pinned out and labeled prior to pathologic processing. Labels have been placed to designate levels 1 through 4. Nodes that are clinically suspicious for harboring SCC are marked out. By orienting the specimen the pathologist is able to more accurately determine which cervical levels are involved. Precise pathologic information is key to planning postoperative radiation therapy.

retained histologically normal but genetically altered mucosa.¹⁰¹ As molecular technology advances in terms of efficiency and sensitivity, it is anticipated that molecular margin analysis will significantly improve surgeons' ability to obtain both histologically and genetically clear margins.

Postoperative complications

A retrospective study on 242 patients older than 70 showed that in older patients predictive factors for postoperative complications include male sex, bilateral neck dissection, two or more comorbidities, and clinical stage IV.²²¹ Patient's age alone is not an indicator of surgical outcome, rather comorbidities and time under general anesthesia are predictors of postoperative complications and hospital length of stay.²²² A study focused on oral cancer found that the operative death rate was slightly higher than 3.2%. Factors which could be used to predict postoperative complications included pre-existing cardiovascular and respiratory disease, alcohol consumption, stage of disease, scale and duration of surgery, tracheostomies, poor differentiation of tumor, and presence of extracapsular spread of carcinoma.²²³ Perioperative stroke in patients having a neck dissection is rare.²²⁴ While studies on head and neck cancer give us less specific information regarding perioperative complications the results can be helpful in preventing complications and improving patient outcome. A review of 3932 patients undergoing head and neck surgical procedures showed that the overall medical morbidity and mortality rates were 5.65% and 2.98%, respectively.²²⁵ The presence of a major medical complication increased the odds of death by 5.65. Postoperative pneumonia was the most common medical complication. Acute myocardial infarction and stroke were rare and were not associated with increased mortality. Prophylactic antibiotics should cover aerobic, anaerobic, and Gram-negative bacteria. Methicillin-resistant *Staphylococcus aureus* infection is devastating in patients undergoing major head and neck surgery.²²⁶ Perioperative complications can be minimized by careful control of blood pressure and heart rate, deep venous thrombosis prophylaxis, and early mobilization.

Surgery and postoperative radiation

The role of radiation therapy in the management of oral cancer is adjuvant. As discussed previously oral cavity SCCs are poorly responsive to radiotherapy as primary treatment.^{151,204,227} Surgical salvage, frequently required following radiation therapy, is extremely difficult and postoperative complications are more frequent and more difficult to manage. Preoperative radiation therapy is not given for oral cavity carcinoma. In certain cases preoperative radiation therapy, in the dose range of 50 Gy, is used for sinonasal or paranasal carcinomas to improve resect-

ability. Preoperative radiation therapy is associated with wound healing complications. The surgical margins would not change following radiation therapy for oral cavity carcinoma. If the well informed patient refuses surgery or is deemed not to be able to tolerate surgical resection, primary radiation therapy can be considered for palliation. The patient should be involved in the decision regarding radiation therapy and it is critical for the patient to be aware of the progressive, life-long side-effects that result from radiation therapy.

Possible indications for postoperative radiation therapy include positive or close margins, perineural invasion, extensive bone invasion, and neck involvement (strong indication for more than one lymph node involved or extracapsular spread). While these recommendations are commonly used, each case must be evaluated independently and there is not universal agreement on these indications.²²⁸ For patients with stage III and IV oral SCC, postoperative radiation therapy improves control of the carcinoma, particularly in the setting of positive margins. A classic paper demonstrating improved disease control was published by Vikram *et al.* in 1984.⁹¹ Even when the authors viewed the margins as satisfactory, recurrence occurred in 39% of the cases compared with 73% of cases when the margins were positive. Recurrence rates were significantly reduced to 2% and 10.5%, respectively, with postoperative radiation therapy. However, the study suffers from a lack of control of variables. Some of the patients received postoperative chemotherapy. The study also included SCCs from multiple head and neck sites, including oral cavity, oropharynx, hypopharynx, and larynx. Loree *et al.* retrospectively evaluated 398 patients treated for SCC and evaluated the impact of surgical margin status, with and without postoperative radiotherapy, on recurrence.⁹⁵ Postoperative radiotherapy did not significantly lower recurrence rates in patients with positive surgical margins. This study concluded that postoperative radiotherapy is relatively ineffective in local control in patients with positive surgical margins. A more recent study looking at 425 patients with oral cavity SCC showed that positive surgical margins increased the risk of death at 5 years by 90%, after controlling for significant prognostic factors.²²⁹ These studies confirm that of all the factors impacting oral cancer patients' outcome, the surgical margin, which is under the direct control of the surgeon, is one of the most critical.

Intensity modulated radiation therapy (IMRT) represents a significant technological step forward in the management of head and neck SCC. IMRT with parotid sparing has been shown to result in less xerostomia, as measured by a validated xerostomia questionnaire, compared to conventional radiation therapy.²³⁰ This study, however, combined multiple head and neck sites with the oropharynx as the single most common site in this study. Radiation therapy in

the form of electrons can be used to boost the neck. This approach treats superficial tissue in the neck and avoids damage to the spinal cord.

Timing of radiation therapy is critical and should be started as soon as possible following healing. Multiple studies have demonstrated that recurrence rates increase when there is a delay in starting radiation therapy. A delay of greater than 6 weeks after surgery leads to increased recurrence rates.⁹¹ The surgeon needs to not only consider the interval between surgery and radiation therapy but the overall duration of the “treatment package”. Locoregional control of oral cavity cancer improves if the duration of all treatment is less than 100 days.²³¹ Breaks in radiation therapy should be avoided in all cases unless the patient’s health is at significant risk. Patients will often require diligent care of the surgeon and radiation oncologist to get them through radiation therapy without a break.

Chemotherapy and radiation therapy

The addition of chemotherapy to radiation therapy has improved both locoregional tumor control and survival rates. The Radiation Therapy Oncology Group 9501 was designed to determine whether concurrent cisplatin therapy and postoperative radiotherapy improve the rates of local and regional control among patients who have high-risk operable head and neck cancer.²⁰² High risk was defined as having any of the following: histologic evidence of invasion of two or more regional lymph nodes, extracapsular extension of nodal disease, and microscopically involved mucosal margins of resection. A total of 416 patients were enrolled. A significant increase in severe adverse side-effects (primarily hematologic, mucous-membrane, and gastrointestinal) was observed. Most importantly, concurrent postoperative chemotherapy and radiotherapy significantly improved the rates of local and regional control and disease-free survival.

A parallel, randomized trial consisting of 334 patients by the European Organization for Research and Treatment for Cancer (EORTC) commenced to test whether adjuvant chemotherapy and radiotherapy improves progression-free survival, overall survival, and local or regional control more than does radiotherapy alone in patients with stage III or stage IV head and neck cancer.²⁰¹ Patients with oral cavity SCCs comprised 87 of the subjects. This trial demonstrated that high-dose cisplatin with radiotherapy significantly increased the rates of local control, disease-specific survival, and overall survival. There was not an increase in late adverse effects.

In each of the above trials cisplatin was the chemotherapeutic agent. Cisplatin is delivered intravenously over 1 to a few hours. It is typically prescribed in a dose of 100 mg/m² of body surface area. In the above trials cisplatin was given three times during radiotherapy (days 1, 22, and 43). The primary complica-

tion is nephrotoxicity but is prevented with hydration before and after treatment.

Follow-up

Most recurrences and locoregional failures will occur within the first 2 years. Follow-up appointments are recommended every month for the first year, every 2 months for the second year, every 3 months for the third year and every 6 months thereafter. There is little available data to guide the surgeon in making evidence-based decisions regarding interval imaging. This decision should be made by the surgeon based on the clinical behavior of the cancer and the pathologic findings. Patients with oral cancer are at risk for second primaries. The risk for a synchronous or metachronous second head and neck primary in patients with oral SCC ranges is 21%, a rate that is higher than patients with pharyngeal or laryngeal SCC.²³² A long-term, large-scale phase III trial evaluating low-dose isotretinoin had no effect on the rates of second primary tumors or smoking-related second primary cancers.²³³ This study did show a substantial impact of smoking on the development of second primary tumors and survival.²³³ Therefore, an aggressive smoking cessation program should be implemented with oral cancer patients who continue to smoke. A 6-monthly chest radiograph as a routine follow-up examination for patients with oral SCC has no clear benefits.²³⁴

Non-resectable SCC or poor surgical candidates

The question is often raised of what to do with an elderly patient with a limited life expectancy. It is often a mistake to not treat the cancer since many of these patients will live much longer than expected and if the cancer is not treated patients will develop significant pain. Surgical resection provides significant relief of the pain.⁶¹ Palliative chemotherapy or chemoradiation should always be considered. A randomized phase III trial evaluated head and neck cancer patients who had stage III or IV disease with no distant metastasis and had tumors considered unresectable.²³⁵ For patients with unresectable head and neck SCC, induction chemotherapy (cisplatin, fluorouracil and docetaxel) followed by chemoradiation (with weekly carboplatin) improves overall survival.²³⁵

Summary

Oral cancer is a surgical disease. Quality-of-life studies have now clearly demonstrated that our patients’ perception of success is different than ours as surgeons. Patient survival is a facile endpoint which is no longer acceptable. Patients must be restored to

near complete function. A thorough and comprehensive understanding of oral function, particularly dental expertise with appropriate training to preserve and restore the preoperative occlusion and jaw relationships, will provide patients the outcome that they are seeking. Cancer resection should be driven by oncologic principles of clear margins. In terms of reconstruction patients should only have more complex reconstruction if it has a clear improvement of function. For example, microvascular reconstruction of a partial glossectomy defect after surgical resection of a T1 tongue SCC must provide the patient with a meaningful improvement in function over a split-thickness skin graft. Austere attention to detail with preoperative planning, intraoperative decision making, and postoperative care, as well as a rigorous, life-long devotion to the care of his or her patients, is the minimum standard expected of the oral and maxillofacial surgical oncologist. The surgeon must assume the role as director of the multidisciplinary team in the management of the oral cancer patient. This role carries with it the responsibility to maintain a contemporaneous, functional fund of knowledge in all areas of oral cancer management including cancer detection, adjuvant therapy, medical management of the postsurgical patient, and molecular biology. The discovery of new knowledge, which should be surgeon driven, will be the most effective method to reduce, and ultimately eliminate, the burden of disease borne by the oral cancer patient.

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Chapter 35

Management of Patients Undergoing Radiation and Chemotherapy

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This chapter describes the types of tumor and the criteria for treating them with radiation therapy alone, radiation therapy combined with surgery, radiation therapy combined with chemotherapy, and radiation therapy combined with both chemotherapy and surgery. The different protocols available for the different types of tumor will be described, including the different order in which they may be given. The side-effects of radiation therapy and chemotherapy are described as well as the long-term oral complications from both types of therapy.

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Cancer in the head and neck region is relatively uncommon, although approximately 644 000 new such cancers are diagnosed each year on a worldwide basis.¹ Approximately two thirds of these cancers occur in the developing world. In the US head and neck cancer accounts for around 3.2% of all new cancers and 2.2% of all cancer deaths.¹ The incidence of head and neck cancer is about three times higher in men than in women.²

Lesions of squamous cell origin are the most common (>90%) of all head and neck cancers, and they are found in the lips, oral cavity, naso-, oro-, and hypopharynx, as well as in the larynx. Notably, during the last decade, cancer in the tonsils and the base of the tongue has increased in frequency, especially in the younger population.³ Cells with origin in the salivary glands may also be observed among head and neck cancers, resulting in adenocarcinomas, mucoepidermoid carcinomas, adenoid cystic carcinoma,

mas, acinic cell cancer, and malignant pleomorphic adenoma. Malignant melanomas and other tumor types are rarely seen.

Among the known etiological factors for oral cancer, smoking and alcohol abuse are the most common. A heavy smoker has a 5–25-fold higher risk of developing cancer in the head and neck region, compared to non-smokers.⁴ An increasing number of reports also indicate that different types of viruses, e.g. human papilloma virus (HPV) and Epstein–Barr virus (EBV), may cause malignancies.

During the treatment of head and neck cancers, many medical disciplines may be involved, i.e. ENT (head and neck) surgeons, maxillofacial surgeons, and oncologists. The treatment program for head and neck cancers differs between different centers worldwide depending on available techniques, tradition, and results. However, the primary goal of any treatment is survival, and the secondary goal is restoration of function and esthetics.

Prior to the decision of which treatment each unique cancer should be subjected to, several investigations are usually performed. Among them, histological examination of samples of the tumor is the most important. Cytological examination of lymph nodes may be a complement to the histology. Computed tomography (CT) scan and magnetic resonance imaging (MRI) are also important to elucidate the extent of the tumor as well as of any regional or distant spread of the cancer. Sometimes palpation of the area as well as inspection using some type of optical instrument may be necessary. A positron emission tomography (PET) scan may be of use to identify a primary tumor in cases where regional metastasis is the first sign of the disease, as well as to detect any recurrences. It can also be used to evaluate the result of treatment. The results are used for classification of the respective carcinoma (Tables 35.1 and 35.2), and also for treatment planning according to the principles at each center or in each country.

An evaluation of the respective patient's general health is also essential in order to evaluate which treatment is the most suitable.

Table 35.1 TNM classification for head and neck cancer. T staging is, however, not valid for nasopharynx, salivary gland carcinoma and laryngeal carcinoma.

Class	Primary tumor (T)	Regional nodal (N)	Distant metastasis (M)
X	Primary tumor cannot be assessed		
0	No evidence of primary tumor	No regional nodes can be assessed	No distant metastasis can be assessed
1	Tumor ≤ 2 cm in the greatest dimension	Single ipsilateral lymph node, less than ≤ 3 cm	Distant metastasis
2	Tumor > 2 cm but < 4 cm in the greatest dimension	a: Single ipsilateral 3–6 cm b: Multiple ipsilateral ≤ 6 cm c: Bilateral or contralateral lymph node ≤ 6 cm	
3	Tumor > 4 cm in the greatest dimension	Ipsilateral or contralateral lymph node > 6 cm	
4	Tumor invades adjacent structures		

Table 35.2 Staging of carcinoma.

Stages	
I	T1N0M0
II	T2N0M0
III	T3N0M0/T1–3N1M0
IV	T4N0M0/T4N1M0/T1–4N2–3M0/T1–4N0–3M1

Generally, however, it can be stated that early stage cancers (I and II) can be treated with either surgery or radiotherapy with comparable results. The anatomic location of the cancer may be the factor that determines if radiotherapy or surgery is to be used; the opportunities for reconstruction and the patient's quality of life must also be considered. For instance, a T1 or T2 tumor of the nose, external ear or the lip may be better treated with radiotherapy rather than surgical resection for esthetic reasons.

A combined approach of both surgery and radiation therapy may be preferable for intermediate stage (III) carcinomas. The order of treatment, i.e. if the radiotherapy is pre- or postoperative, differs between countries and centers on a worldwide basis.

In patients with local late-stage tumors (IV), a combination of chemotherapy, radiotherapy, and surgery may be the treatment of choice. If the cancer is not resectable or if an organ is to be preserved, chemotherapy and radiotherapy are frequently used initially and then surgery may be used either to complement the treatment or as salvage treatment. In other cases, when the tumor is resectable while preserving an acceptable level of function and esthetics, or when reconstruction is possible, the treatment can begin with surgical resection, to be followed with radiotherapy.

Radiotherapy

The aim and purpose of radiation therapy in the treatment of head and neck cancer may be two-fold:

- the target (cancer) is either a primary tumor or with regional spread to the regional lymph nodes;
- the target is clinically and radiologically tumor-free lymph nodes of the neck, but with a high risk of micrometastasis.

Occasionally radiotherapy may also be used to treat benign tumors, such as ameloblastomas or pleomorphic adenomas, when other treatments may be detrimental.

The intention of radiotherapy may either be curative or palliative. When the intention is to cure the patient from the disease, the radiation may be given either as a single modality if the cancer is small, i.e. stage I and II, or as a complement to surgery. When combined with surgery, it may be given prior to, or after surgery. Radiotherapy can also be given in combination with chemotherapy, with a curative intention. When the radiotherapy has a curative intention, the radiation is given at small doses (1–2 Gy) once or twice a day for a period of 3–7 weeks.

When the cancer is not curable, the intention of radiotherapy may be palliative, i.e. it can be used to reduce the symptoms of the disease, e.g. reducing pain emanating from skeletal metastasis, relieving pressure on vital organ systems. When the radiotherapy has a palliative intention, higher doses are given each time, to reach an effect faster. Palliative radiotherapy can improve the quality of life during the last months of life of a patient with an incurable cancer.

Radiation involves the transport of energy, either as electromagnetic waves (photons) or as a beam of particles (electrons or protons). The position of the tumor determines which type of radiation to be used. Particle beams normally have a shorter range, implying that such radiation is used when the cancer is located relatively superficially in the tissues. In contrast, photon radiation is used when the tumor is located more deeply in the tissues.

When radiotherapy is given as a single modality therapy, a lower dose is given to local lymph nodes in order to prevent and sterilize micrometastasis and a

higher dose is administered to the known primary tumor, i.e. the cancer. If, on the other hand, radiation is combined with surgery, a smaller dose is given with the aim of sterilizing potential micrometastasis in local lymph nodes.

Conventional radiotherapy implies that the primary tumor as well as the larger lymph nodes of the neck are irradiated with up to 70Gy. The radiation is given in single fractions of 1.8–2.0Gy on a daily basis for 6–7 weeks.

Altered fractions have recently been shown to achieve better control both locally and regionally. Hyperfractionation is where 1.2Gy is given twice a day for about 6–7 weeks, and accelerated fractionation means that 1.6Gy is given twice a day for about 6 weeks. A third alternative may be accelerated fractionation with a concomitant boost given as 1.8Gy a day for 6 weeks and during the last 12 days an additional 1.5Gy is given daily as the boost.

Recently a new form of radiotherapy has been introduced, IMRT (intense modulated radiotherapy). This technique is built on a computer-generated three-dimensional image which allows delivery of high doses of radiotherapy to clinical target volumes, still preserving some critical normal structures, for instance salivary glands.

Brachytherapy

Interstitial radiotherapy, or brachytherapy, is administration of irradiation by implantation of a radioactive material very close to, or inside, the primary tumor. A frequently used substance is iridium (^{192}Ir). The time-span of the treatment may vary between a few seconds to a few days, depending on the type of cancer.

Small tumors, without a risk of spreading to the regional nodes, may be treated with brachytherapy only. In cases where the tumor is of larger size or invades surrounding tissues or structures, as well as when there is a risk of spread to regional lymph nodes, brachytherapy is used to deliver a final boost to the primary tumor after an initial more moderate external dose. Brachytherapy is frequently used as the final part in the treatment of cancer in the base of the tongue and in the tonsils. Brachytherapy can also be used when there is a palliative intention, for instance in the treatment of laryngeal cancer.

Chemotherapy

The use of drugs in the treatment of head and neck cancer is still a matter of debate. Despite considerable research, there is no drug or combination of drugs available today that can cure solid cancers in the head and neck region. In general oncology, chemotherapy is given either to cure cancer, or to reduce the speed of progression, or to ameliorate the symptoms. Sometimes

chemotherapy is also administered to reduce the risk of recurrences, so-called adjuvant treatment.

Chemotherapy is generally used prior to (induction), or parallel (concomitant) to radiotherapy. The purpose of chemotherapy is to kill the cancer cells, or at least impede their growth.

Delivery of chemotherapy is made either by intravenous or oral administration. Different drugs are normally combined in different ways depending on the type of cancer to be treated. The drugs are normally given every second to every fourth week. Drugs frequently used are cisplatin, 5-fluorouracil (5-FU), and methotrexate.

It is necessary to repeat the treatment since all cancer cells are not killed at the same time. During the treatment of cancer in the head and neck region, it is normal to administer the drugs two to three times prior to radiotherapy, where the third treatment frequently is concomitant to radiotherapy. Prior to a new administration of drugs, blood counts are always analyzed. After a series of administrations of drugs, an analysis of the treatment is performed, often by a CT scan.

The drugs used in chemotherapy affect the cells in different ways. Some drugs are designed to affect the DNA. Other drugs interfere with the intracellular transporting systems. The aim of all drugs is, however, to result in apoptosis of the cancer cells. To achieve maximum effect, with as few side-effects as possible, different combinations of drugs are normally used.

An advantage with the use of chemotherapy is that the drug, as it is administered via the bloodstream, is able to affect cancer cells on their way to producing metastasis somewhere distant from the original tumor. A disadvantage with the use of chemotherapy is that none of today's available drugs are able to distinguish between cancer cells and normal, healthy cells, which means they also have effects on normal tissues. Among the normal tissues most affected by chemotherapy are tissues with a high turnover rate, i.e. bone marrow, hair, and mucosa, resulting in decreased blood counts, loss of hair, and gastrointestinal problems (nausea and diarrhea). Fortunately, the normal and healthy cells have a great capacity for recovery and repair of the DNA, resulting in complete or almost complete remission of the side-effects after termination of chemotherapy. This is the reason for interruptions in the chemotherapy schedule.

Side-effects

Radiotherapy

The side-effects of radiation therapy can be divided into acute, but mostly reversible effects, and late, more commonly irreversible conditions. The acute side-effects almost always decline within 4–6 weeks after completion of radiation, whereas the late effects

can develop after different periods of time and they will also almost always last for the rest of the patient's life.

Among the acute effects, an inflammatory reaction in the mucosa (Fig. 35.1) of the oral and pharyngeal mucosa – mucositis – is the most common. The appearance of the mucosa can vary from an increased redness, to an intense reddish appearance, to white necrotic lesions. The damage to the oral mucosa can also make it more susceptible to both bacterial and fungal infections. These conditions are almost always associated with varying degrees of pain, and may need relatively strong analgesics.

The skin reactions can vary between a tan-like appearance to almost burn-like (Fig. 35.2) wounds. Softening and lubricating lotions are recommended for the affected skin. Males should be recommended to use an electrical shaver in order to spare the skin as well as not to use aftershave, since the alcohol may add to dryness of the skin.

An increased production of sticky mucus can also be observed after radiation of cancers in the pharyngeal region. Both still and sparkling water seem to be effective in dissolving the mucus. The use of milk products should be minimized.



Fig. 35.1 Intraoral mucositis, degree 3–4, observed at the later stages of external, and after interstitial, radiotherapy.



Fig. 35.2 Clinical appearance of a burn-like wound of the skin after termination of external radiation therapy.

Nutritional problems are frequently seen, and are almost always a result of local reactions in the mucosa as well as of the pain. A loss of appetite may also be a result of loss of, or change of, taste. A majority of the patients undergoing radiotherapy to the head and neck region need professional assistance with nutrition. Some people may need some type of gastric tube to be able to feed themselves.

Tiredness and nausea are other side-effects which are frequently observed during radiotherapy.

Among the irreversible side effects observed after radiotherapy, xerostomia is the most common. The degree differs between different patients. A majority of all saliva is secreted by the large salivary glands, and frequently these glands are within the field of irradiation. A secondary effect of increased oral dryness may be an altered taste and increased sensitivity to different tastes. Some patients need to avoid some type of spices, for instance pepper. The mucosa can also be more sensitive to different types of food, i.e. its consistency. Xerostomia carries an increased risk for development of dental caries and other dental problems.

Radiation also decreases the elasticity of the tissues, which can result in both dysphagia and trismus. The negative effect of radiation on vessels is also well recognised; decreased vascularity can lead to necrosis of both the soft tissues and the bone within the irradiated area.

Chemotherapy

Chemotherapy is also associated with a number of side-effects. Loss of hair is the most well known side-effect of chemotherapy and is usually observed between 1–2 weeks after the treatment.

All mucous membranes are susceptible to chemotherapy due to the quick turnover of cells. Wounds and dryness of the mouth, nausea, and diarrhea may be the result of chemotherapy. Nausea is observed more often in women and younger people. The condition can, however, be reduced effectively with medications.

Loss of appetite is another side-effect, resulting in loss of weight. Professional nutritional counseling is frequently needed. It is, however, not unusual for a patient to gain weight during chemotherapy due to the fact that they may feel better with food in the stomach.

Another vital organ affected by chemotherapy is the bone marrow. The effect may be low blood counts for all types of blood cells, resulting in anemia, increased susceptibility to infections, and increased risk of spontaneous bleeding, e.g. nose bleeds and gingival bleeding. Transfusions may be necessary during chemotherapy.

Tiredness is also a frequently seen side-effect. Lack of energy and problems with concentration may be related to the tiredness. It is of great importance to

rest when necessary, to eat as good and well balanced food as possible, and to exercise.

The most positive fact related to chemotherapy is, however, that all side-effects almost always subside after termination of the treatment.

Management of oral health during radiation

Prior to the start of radiation therapy for treatment of head and neck cancers, it is of great importance to survey the oral health of the patient. A thorough oral examination, including radiographs, with special attention to marginal and apical periodontitis, calculus, and dental caries, is necessary. All infections should be treated, i.e. teeth may be either extracted or treated endodontically. Calculus should be removed, i.e. the teeth should be scaled and cleaned professionally. All caries should be excavated and the teeth should, at least, be supplied with temporary fillings. Daily rinsing with sodium fluoride solutions is recommended and prescribed to each patient as a complement to the use of fluoridated toothpaste.

By taking impressions of both the upper and the lower jaw, the dental technician can make casts on which different devices can be produced. These devices, the mouth-opening device (Fig. 35.3) and the mandibular protection device (Fig. 35.4), are used in order to reduce the effect of irradiation on, or to protect, the surrounding tissues not in need of treatment. The casts can also be used to produce trays (Fig. 35.5) to administer high-concentration (0.2%) sodium fluoride gel on a daily basis.

The mouth-opening device can be designed with and without a tongue depressor. A device without a tongue depressor is used during the radiotherapy when the cancer is located in the floor of the mouth, in the submandibular gland, or in the chin. In contrast, a device supplied with a depressor is used when the cancer is located in the tongue, in the base of the tongue, in the maxillary sinus, in the nose, in the



Fig. 35.3 A clinical picture of a patient with a mouth-opening device used to protect parts of the head in no need of radiation.

upper lip, in the palate, and sometimes also in the floor of the mouth.

The mandibular protection device is used during interstitial radiotherapy to displace the tongue, the cheek or the lip away from the implanted radiation source. It also protects the mandibular bone. This device is used during interstitial radiation of carcinomas in the tongue, the base of the tongue, the tonsils, the floor of the mouth, and the lips. The mandibular protection device is made of approximately 6 mm thick plastic with 2 mm of lead included. The device can also be used to protect the bone of the maxilla, although it is most frequently used to protect the mandible.

During the period of radiation, at least weekly professional cleaning of the teeth and mouth is recommended. As treatment continues, i.e. radiation accumulates within the tissues, the patient will have more discomfort, making it hard to maintain proper oral hygiene.

Management of oral health during chemotherapy

As with patients scheduled for radiotherapy, it is important to thoroughly examine the oral health of



Fig. 35.4 A clinical picture of a patient wearing a mandibular protection device used during interstitial radiation (brachytherapy).



Fig. 35.5 A clinical picture of a patient wearing fluoride gel trays.

patients who will receive chemotherapy. An individual treatment plan should be determined for each patient. The importance of maintaining good oral hygiene during the period of chemotherapy cannot be overstressed. Frequently, the patient may need professional help to achieve optimal oral hygiene.

It is, if possible, desirable to manage the most acute oral problems prior to the start of chemotherapy, as for patients subjected to radiotherapy. However, this is often impossible since chemotherapy is initiated rather quickly after diagnosis. Management of oral health is then scheduled to be performed just before the beginning of the second or third cycle of chemotherapy. It is important to obtain a current blood count before any invasive dental treatment, to prevent development of infections, and any undesirable postoperative bleeding. A leukocyte count of $<0.5 \times 10^9/l$ (neutropenia) is considered to represent a high risk of infection. A count of $0.5\text{--}1.0 \times 10^9/l$ should be accompanied by antibiotic treatment for 7–10 days postoperatively, whereas a leukocyte count of $>1.0 \times 10^9/l$ only should be followed by a single dose of antibiotics prior to any kind of surgery or other invasive dental treatment, i.e. scaling or endodontics. Extractions and endodontic treatment should be performed at the latest 10 days before an expected leukocyte count of less than $0.5 \times 10^9/l$.

Mucositis normally develops after 5–10 days of chemotherapy. At later stages, ulcers can develop. The ulcers can be infected secondarily with bacteria, viruses or fungi. They can also be very painful, resulting in nutritional problems.

Emotional well-being

For most patients, the term cancer is equivalent to a death sentence. It is of great importance to thoroughly inform each patient as an individual as well as on a general basis with regard to the treatment opportunities that are available, the prognosis of the desired treatment, and expected side-effects. It is also of great importance to stress the fact that many patients today can be completely cured from a malignant disease. All information should be given to both the patient and to relatives. Often, reinformation is needed.

Professional psychological care is frequently indicated in order to assist the patient through the life crises that a cancer diagnosis implies. Psychological assistance can, to some extent, be of value for a relatively long period of time after termination of oncological treatment.

Follow-up

It is recommended that patients are followed up on completion of the cancer therapy, irrespective of treatment given to the patient. Follow-up of the patient is usually performed by all treatment disci-

plines involved. One reason is to discover early any signs of cancer recurrences. The earlier a recurrence is observed the better is the prognosis. Early after completion of the oncological treatment, follow-ups are more frequent, but with increased time after the treatment, and if there is no sign of recurrence, the period between each follow-up visit is extended. The patient should be followed for life, but due to mostly socioeconomic factors, follow-ups are terminated after 5 years.

From an odontological point of view, it is important to frequently evaluate the oral as well as the dental health with regards to development of carious lesions, periodontal problems, and early signs of development of both soft tissue necrosis and osteoradionecrosis. Professional dental cleaning can be of great value for a long time after termination of the oncological treatment.

Management of postradiation conditions

As mentioned, a number of conditions can be observed in patients after completion of radiation therapy. Some of these effects can be seen relatively early after radiotherapy, whereas some can occur as late as up to 10 years after. It is not possible to forecast when, or sometimes even if, osteoradionecrosis or soft tissue necrosis will occur.

To reduce the xerostomia the patient should be recommended frequent intake of small amounts of water. Today, there are also a number of saliva replacement products, artificial saliva, and saliva-stimulatory products available on the market, which have many positive effects on oral well-being. The use of such products can also be of benefit for taste. Life-long use of additional sodium fluoride products, i.e. daily rinsing, should be recommended in order to prevent rapid development of dental caries. Frequent and scheduled visits to a general dentist and/or a dental hygienist are desirable.



Fig. 35.6 A clinical view of a severe case of osteoradionecrosis in the anterior part of the mandible.

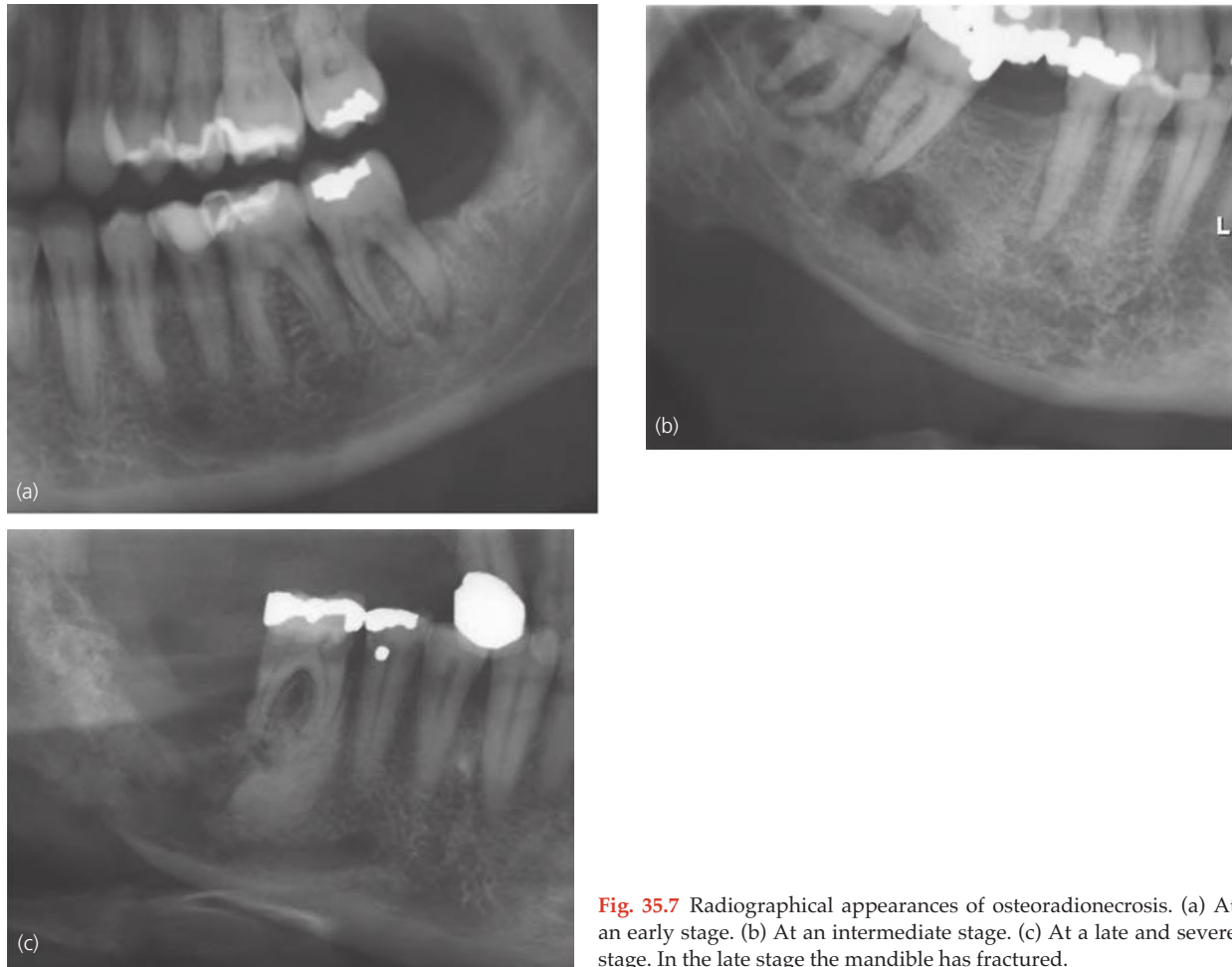


Fig. 35.7 Radiographical appearances of osteoradionecrosis. (a) At an early stage. (b) At an intermediate stage. (c) At a late and severe stage. In the late stage the mandible has fractured.

Reduced mouth-opening capacity is another late side-effect of irradiation. Different training programs are available to prevent early development of this condition. Sometimes it can be of benefit to begin the training already prior to radiation, and continue it as soon as possible after termination of treatment. A very strict and frequent use of these training programs is desirable since the effect is quickly reduced and even eliminated if they are not followed. Today, a number of mouth-training devices are also available on the market.

Soft tissue and bone necrosis (osteoradionecrosis) are the side-effects that usually develop last (Figs 35.6 and 35.7). It is not possible to predict which patient is at risk for development of these conditions, though they rarely occur in tissues subjected to less than 55Gy of radiation. To prevent the development of these conditions it is of benefit to treat dental problems immediately and conservatively, i.e. regular visits to a general dentist to prevent development of both periodontal disease and dental caries. Dental extractions should be avoided as long as possible. Instead, endodontic treatment should be the treatment of choice when apical periodontitis has developed. If extraction is the only alternative, it should be performed surgically with the aim of covering the

area with soft tissue. Systemic use of antibiotics for 7–10 days is recommended.

When and if osteoradionecrosis has developed, it is important to maintain good dental hygiene, to prevent development of infections in the affected area.

Some alternatives are available to reduce the speed of development, although controversy remains. Hyperbaric oxygen treatment has been used to reduce the development of, and also to treat, osteoradionecrosis.⁵ In later stages of osteoradionecrosis it may be necessary to carry out surgical resection, with or without adjunctive hyperbaric oxygen. Microvascular reconstruction, using free flap technology, after surgical resection of the affected area, is today a relatively common treatment in order to restore both function and esthetics in this group of patients.

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Chapter 36

Salivary Gland Disorders

Mark McGurk and Jeremy Sherman

This chapter is devoted to salivary gland disease, which is classified by its clinical presentation as:

- discrete single or multiple masses within a gland;
- diffuse enlargement of a gland;
- intermittent swelling of a gland (obstruction);
- bilateral swelling of glands;
- infection;
- xerostomia (dry mouth);
- drooling.

Emphasis is given to less invasive surgical procedures including endoscopic and non-invasive management of salivary calculi and more localized surgery for many types of salivary gland tumors.

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Diseases of the salivary glands are relatively uncommon but because of the glands' function and position within the complex anatomy of the head and neck they have particular interest for surgeons. Three main disease entities are encountered on a regular basis, namely obstructive disease, salivary tumors, and inflammatory disorders (mainly Sjögren's syndrome), in order of frequency. The remainder are vanishingly scarce and cannot be covered comprehensively in a single chapter on salivary gland disorders.

In terms of development, the subject has remained relatively static throughout the 20th century as disorders of the salivary glands are infrequent. Thus experience has been dissipated over multiple specialties. This resulting dilution of practical knowledge meant that dogma went unchallenged and was recycled from one textbook to another. Now a revolution is under-

way in salivary disease management. Many of the principles on which treatment has been based are being challenged. This chapter aims to highlight these new developments within our area of clinical practice.

Differential diagnosis

Introduction

Salivary gland disorders present to clinicians in several different ways as listed in Table 36.1. These will be used as the template to classify salivary disease as an alternative to the traditional pathological classification (inflammatory, neoplastic, autoimmune, etc.). Table 36.2 lists pathological entities by clinical classification.

Table 36.1 Categorization of salivary pathology by clinical presentation.

Discrete single or multiple masses within a gland
Diffuse enlargement of a gland
Intermittent swelling of gland (obstruction)
Bilateral swelling of glands
Infections
Xerostomia (dry mouth)
Drooling

Table 36.2 Pathological entities by clinical classification.**Discrete single or multiple masses within a salivary gland****Granulomas and chronic infections**

Tuberculosis (TB)
 Non-TB mycobacterial infections
 Actinomycosis
 Cat scratch disease
 Syphilis
 Hydatid disease

Inflammatory conditions

Kim-Kimura's disease
 Castleman's disease
 Kuttner tumor
 Inflammatory pseudotumor

Cystic lesions

Simple cysts
 HIV-SGD
 Dermoid cysts
 Congenital duct cysts
 Polycystic disease of the parotid gland

The diffusely enlarged gland**The intermittently swollen gland (unilateral)**

The obstructed gland
 Sialolithiasis
 Strictures
 Polyps/mucus plugs
 Impacted foreign bodies
 Pneumoparotid

Persistent swollen gland (unilateral)

Infection
 Acute
 Subacute
 Chronic
 Autoimmune salivary disease
 Benign lymphoepithelial lesions
 Sjögren's syndrome
 Wegener's granulomatosis
 Graft-versus-host disease
 Non-autoimmune salivary gland disease
 Amyloid
 Sarcoidosis
 Churg–Strauss syndrome
 Metabolic salivary gland disease
 Endocrine
 Liver, gastrointestinal disease and malnutrition
 Bulimia nervosa
 Cystic fibrosis
 Miscellaneous

The full spectrum of salivary gland disease can be seen across all age groups but the frequency of each entity tends to vary with age. In childhood developmental conditions predominate (hemangiomas and lymphangiomas), in middle age the emphasis is on inflammatory/obstructive disorders and to a minor extent neoplasia, whereas in the elderly neoplasia needs to be excluded. Swellings that occur in either the tail of the parotid or in the submandibular triangle fall within the differential diagnosis of a lump in the neck.

Investigations

Salivary glands are easily accessible and open to a wide range of investigative techniques. The first-line investigation is usually high-resolution B-mode ultrasonography. It is now readily available and is becoming part of the basic equipment in the surgical office. It can reliably distinguish between intra- and extraglandular masses, accurately recognize calculi/strictures and can demonstrate changes within the parenchyma. Doppler flow studies can identify vascular pathology. Conventional plain radiographs are virtually obsolete except for the ability to show small (<2 mm diameter) calculi within the ductal orifice. Traditional sialograms are still useful when investigating stones and evaluating strictures, prior to sialendoscopy (see Obstructive disease, later), and have not been superseded by magnetic resonance imaging (MRI) sialograms, except in expense. Salivary masses deserve more detailed evaluation by MRI (with modest superiority over computed tomography (CT)) and recent developments such as diffusion-weighted and dynamic contrast-enhanced MRI may, in the future, permit routine visualization of the facial nerve, although at present this is still a research tool. Salivary gland scintiscans assess glandular function.

The second most useful investigation to the salivary clinician is fine-needle aspiration cytology (FNAC). The prospect of a reliable tap can be improved by ultrasound- or CT-guided aspiration, especially if an experienced cytologist or cytology technician is in attendance. A competent cytological service should be able to distinguish between benign and malignant disease in over 85% of cases.^{1–3} A correct histological diagnosis is more problematic and results cannot be relied on but the technique has particular value when a neoplasm is not suspected and if this is confirmed surgery can be avoided.⁴

Although frequently overlooked, FNAC is a useful way to sample the discrete intraoral mass, as is a punch biopsy (dermatological punch). FNAC has particular relevance in managing the submandibular mass because of the high risk of malignant diseases at this site. A Tru-cut core biopsy may be useful if lymphoma is suspected, and may thus avoid the need for surgery.

Diagnostic message

Disease risk influenced by age
 Mass in the tail of the parotid and submandibular gland falls within the differential diagnosis of a lump in the neck
 FNAC/core biopsy can be used to sample intraoral masses

The discrete salivary mass

The overbearing responsibility in the presence of a discrete lump is to confirm or refute the presence of a neoplastic lesion. Particularly with granulomatous disease, the risk is to mistake the indurated tuberculous mass for an indolent cancer.

Granulomas and chronic infections**Tuberculosis**

Salivary tuberculosis usually manifests as a slowly enlarging painless mass that can masquerade as a neoplasm in about 3% of cases.⁵⁻⁷ In many instances the chest radiograph is normal and there is no lymphadenopathy. Diagnosis requires a high threshold of suspicion (Fig. 36.1) and knowledge of risk factors



Fig. 36.1 Persistent granuloma in an individual recently arrived from Malaysia which proved to be tuberculosis.

(ethnic group, recent travel abroad to endemic countries, old age, malnourished, alcoholism/drug addiction, and HIV infection). The saliva contains few if any bacilli⁸ and diagnosis is usually secured by FNAC or core biopsy. Mantoux or purified protein derivative (PPD) testing is often unhelpful due to previous BCG immunization.⁷ Anti-tuberculosis chemotherapy is the treatment of choice.^{9,10}

Non-tuberculous mycobacterial infection

This condition can be indistinguishable from the traditional tuberculous lesion and typically *Mycobacterium avium intracellulare*, *M. chelonae*, *M. malmoense*, *M. bovis*, and *M. scrofulaceum* organisms may be isolated.^{10,11} Polymerase chain reaction (PCR) assay facilitates rapid identification. Antibiotic therapy (clarithromycin, possibly long term) is the treatment of choice.¹²⁻¹⁵

Cat scratch disease

This self-limiting zoonosis is caused by *Bartonella henselae*.¹⁶ It may present as single or multiple enlarged preauricular facial or upper cervical lymph nodes. Parotid involvement occurs in about 3% of cases.¹⁷ It follows direct feline contact and a small papule forms at the site of inoculation accompanied by low-grade fever and general malaise. Rarely, conjunctivitis, encephalopathy, and cranial nerve palsies may arise. Diagnosis is usually through specialist hematological tests (Hangar-Rose antigen) or nodal biopsy. Treatment is largely symptomatic and co-trimoxazole or penicillin may stop ugly sinus formation.¹⁶⁻¹⁸

Actinomycosis

Salivary gland involvement is rare;¹⁷ anaerobic culture or histopathological analysis of a biopsy specimen clinches the diagnosis. Long-term penicillin therapy achieves a cure.

Hydatid disease

Echinococcus granulosus is rare and limited to rural and sheep-rearing areas.^{9,19} The parotid gland is implicated in 1-19/1000 patients with this disease⁹ and in appropriate circumstances the combination of eosinophilia, a raised erythrocyte sedimentation rate (ESR) (>50 mm/h), and a positive Weinberg complement fixation test establishes the diagnosis.⁹ Treatment is surgical removal but without rupture.²⁰⁻²⁴

Syphilis

Syphilitic parotitis is mainly of historical interest and is confirmed by serology.

Inflammatory conditions

Kim-Kimura's disease

The disease typically presents in young oriental males (male to female ratio 1:5) occurring at any age,^{25–27} as either single or multiple slowly enlarging indolent subcutaneous or salivary nodules²⁶ with or without regional lymph node involvement.^{25,28,29} The condition is endemic in the Far East and has an unknown aetiology.^{30–32}

Histologically the picture is one of lymphoid follicle formation with marked eosinophils and surgery is the only effective treatment.

Castleman's disease

This uncommon disease presents typically as a solitary, painless, slow-growing mediastinal mass (86%);^{33,34} only 2% involve the intraglandular or paraglandular parotid lymph nodes^{35–42} in adults between 15 and 35 years of age.³⁴ The condition is associated with fever and immunological disturbances⁴³ (including HIV,⁴⁴ myasthenia gravis,⁴⁵ severe erosive lichen planus,⁴⁶ pemphigus vulgaris,⁴⁷ monoclonal hypergammaglobulinemia, hypoalbuminemia, refractory anemia,^{34,48–50} or thrombocytopenic purpura.⁵¹ Histology reveals lymph node hyperplasia, either a hyaline vascular form (80%) or plasma-filled type.^{34,52} Treatment is complete surgical excision^{42,53} but supported by radiotherapy and chemotherapy where appropriate.⁵⁰

Kuttner tumor

This is a rare form of chronic sclerosing sialadenitis^{54–56} of the submandibular gland. It is an ill defined and probably not a homogeneous entity.⁵⁶ Diagnosis is difficult and usually relies on excision biopsy.

Inflammatory pseudotumor

This is a rare but real entity. It consists of an autoimmune inflammatory mass with features that mimic cancer in various organs (e.g. especially lung and liver).^{57–62} Presentation includes a unilateral painless induration, cicatrization, trismus, with neurological involvement but without lymphadenopathy.⁵⁸ Diagnosis is by open biopsy and excision may not be practical. It can be controlled by high-dose steroids.

Cystic lesions

Simple cysts (mucocele/ranula)

These are easily diagnosed by their bluish spherical appearance (Fig. 36.2) or aspiration (no risk of infection). The majority (>90%) are extravasation mucoceles.^{63–64} Ranulae arise from the lesser sublingual gland, and the plunging ranula^{65–68} has the distinc-

tion of running into the submandibular space creating a diagnostic problem. Careful ultrasound evaluation will demonstrate a dehiscence in the mylohyoid muscle and provide an aspirate of saliva. As ranulae originate from the lesser sublingual gland⁶⁶ treatment consists of sublingual gland removal. The ranula sac can be left *in situ* (after aspiration of saliva).⁶⁶

HIV-associated salivary gland disease

Human immunodeficiency virus 1-associated salivary gland disease (HIV-SGD) is a term that has been used to define enlargement of one or more major salivary glands (60% bilateral^{69–75}) and/or xerostomia in HIV-1 infected patients.^{69,70} Solitary gland involvement is rare (2%). About 5% of cases develop cosmetically debilitating bilateral parotid cystic enlargement (Fig. 36.3).^{76–79} Symptoms and ultrasound images may mimic Sjögren's syndrome.^{69,80–84} Surgical treatment has been replaced by highly active antiretroviral therapy (HAART). There may be an increased risk of associated salivary gland lymphoma.^{79,80}

Dermoid cysts

Occasionally, a dermoid cyst occurs in the parotid or floor of mouth. They are diagnosed by clinical features and FNAC.⁸⁵

Congenital duct cysts

Congenital duct cysts are retention cysts (Fig. 36.4) of the parotid gland and are rare. Treatment is conser-

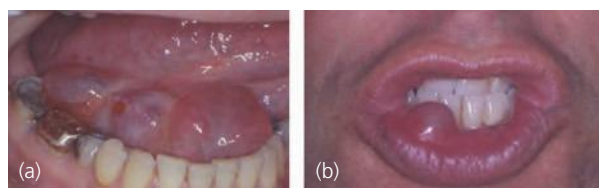


Fig. 36.2 Extravasation cysts on (a) floor of mouth (ranula) and (b) the lip (mucocele).

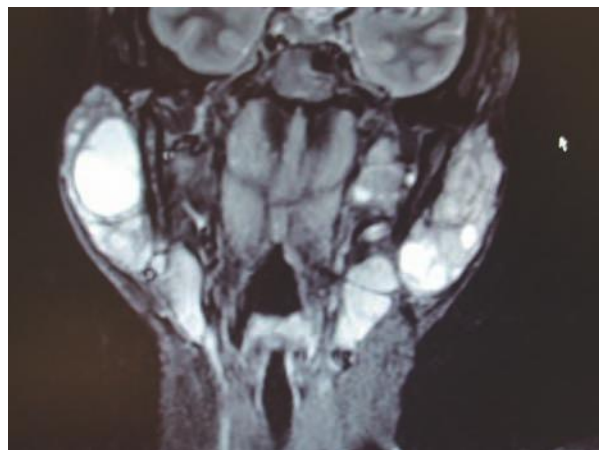


Fig. 36.3 HIV-SGD: multiple cysts involving both parotid and submandibular glands.

vative.^{64,86} Repeated aspirations yield amylase content >15 000 IU/ml, confirming the diagnosis.

Polycystic disease of the parotid gland

This entity is very rare; only eight cases have been reported, affecting only females.^{87–92} The cystic changes usually do not involve the main parotid duct and are demonstrable by sialography or ultrasound. Treatment is conservative.⁸⁹

The intermittently swollen gland

The obstructed gland (see Obstructive disease)

Mucus plugs

The development of thick gelatinous saliva is probably a regular occurrence and goes undetected in normal pliable ducts. Plugs only become symptomatic in the presence of an obstruction. In most cases there is a concurrent stricture. The symptoms are subtly different to mealtime syndrome with nothing found during ultrasonography. Plugs form with stagnation, therefore symptoms occur on waking and after periods of dehydration. Sialogogues and massage are normally sufficient to control symptoms but irrigation via sialendoscopy plus balloon dilatation of the stricture is also an option.

Polyps

Pedunculated lesions arising from the duct lining may remain asymptomatic until picked up on sialendoscopy. Their significance is unknown.

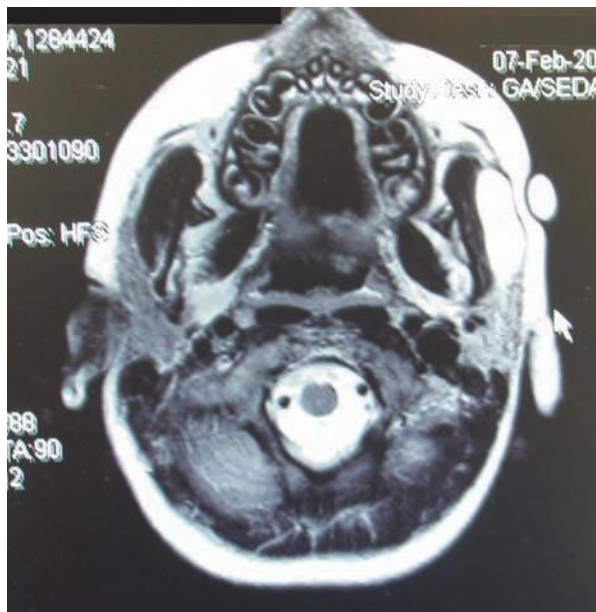


Fig. 36.4 Left parotid duct cyst.

Foreign body impaction

This is a rare occurrence and many reports are anecdotal (e.g. grass seed, fish bones). In the context of salivary gland disorders it is not an important entity.^{93–95}

Pneumoparotid (trumpet blower's syndrome)

Air insufflation of the parotid is a real entity. The parotid duct has a valve-like structure and a tight puctum that stops air passing into the duct. If these are incompetent then air and bacteria pass up the duct. This may be one of the mechanisms involved in "relapsing parotitis of childhood". Symptoms may coincide with playing wind instruments.⁹⁶

The diffusely enlarged gland

Infection (bacterial and viral)

Acute viral sialadenitis

Over a third of all pediatric salivary gland disorders are of an inflammatory nature, with either an obstructive or non-obstructive etiology.⁹⁷

There is a wide range of viral infections that can involve the salivary glands (Coxsackie A⁹⁸ and B3,¹⁰⁰ parainfluenza B,^{101–106} ECHO type 9,¹⁰⁷ and Epstein-Barr^{108–111}). The most common is mumps. It is caused by a highly infectious paramyxovirus with an incubation period of 2–3 weeks.^{112,113} The subject is infectious from 5 days prior and 10 days after the onset.^{113–116}

If the diagnosis is in doubt, the virus can be cultured from saliva^{113,115} or established from rising antibody titres (S-antigen followed by V-antigen). The V-antibody confers life-long immunity.^{116–120}

In adults, the condition may be more severe and complicated by orchitis or oophoritis and varying degrees of meningoencephalitis are relatively common (25%). In rare instances, occurrence may be unilateral.

Juvenile recurrent parotitis

This is another real clinical entity! Juvenile recurrent parotitis (JRP) is a unique non-obstructive, non-suppurative, usually unilateral variant of sialadenitis and represents the second most common parotid disorder amongst young children (Fig. 36.5). Traditionally it is considered a self-limiting condition⁹⁶ but Maynard followed a cohort of children into adulthood and reported continual problems in approximately 30–40% of cases.¹⁷

It is probable there is more than one etiology (see Table 36.3). Incompetent parotid ducts, juvenile Sjögren's syndrome, lymphoma, HIV-SGD amongst



Fig. 36.5 Young boy with a history of recurrent parotid infection that induced systemic symptoms. Parotid gland wash-out (yearly) and long-term antibiotics contained symptoms.

others, congenital ductal malformations, genetic factors, bacterial or viral infection, allergy or autoimmune congenital immunoglobulin deficiency have also been postulated.^{121,122} In Kaban's series of eight patients, 62 episodes of infection were recorded (range 8 weeks to 11 years). A single patient experienced 18 acute attacks.¹²³ The symptom-free interval may be increased following duct lavage.^{124–125} Intermittent bilateral ductal lavage (normal saline, 100mg hydrocortisone and amoxicillin–clavulanic acid 25mg/kg via a sialendoscope) is becoming the treatment of choice.¹²¹

Bacterial sialadenitis

The condition is uncommon nowadays and when it does occur it is invariably related to salivary calculi^{126,127} and/or a predisposing medical condition (immune suppression).^{129–132} It may also arise as an acute exacerbation of chronic sialadenitis (Fig. 36.6). The author's recommended protocol for management is listed in Table 36.4.

Table 36.3 Local or systemic predisposing factors for acute suppurative sialadenitis in childhood.¹²²

Fever and/or dehydration secondary to an upper respiratory tract infection, systemic viral illness or acute glomerulonephritis
Immunosuppression secondary to congenital immunoglobulin deficiency or chemotherapy for acute leukemia
Failure to thrive or respiratory distress syndrome in premature or newborn infants
Previous trauma or mumps involving the parotid gland

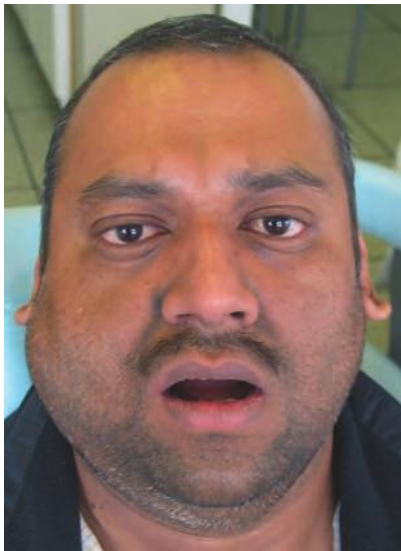


Fig. 36.6 Acute parotitis as a result of a retained stone.

Historically, parotitis was traditionally associated with postsurgical patients, who were often dehydrated, and was frequently the final event of a debilitating illness. Parotitis is uncommon today due to greatly improved standards of perioperative care, especially with respect to maintenance of hydration. Parotitis in the otherwise well patient is now nearly always secondary to a salivary stone and chronic infection can follow radiotherapy. First-line treatment consists of antibiotics targeted at staphylococci and streptococci.^{130,133} Other less commonly implicated microorganisms are listed in Table 36.5.¹³⁴ Surgical intervention (drainage) is seldom required apart from stone removal.

Chronic/subacute sialadenitis

Chronic sialadenitis is usually unilateral, recurrent, and secondary to an underlying obstruction (stone or foreign body).¹²⁶ Approximately 2% of patients with Sjögren's syndrome are affected each year.^{133,135–137} The duct orifice appears inflamed with an accompanying purulent, salty tasting discharge. Maynard demonstrated the sequential architectural changes by parotid sialography (Table 36.6).¹³⁸ Maynard also demonstrated that the salivary secretion rate is significantly reduced in patients with recurrent parotid

Table 36.4 Management of acute bacterial sialadenitis

1. Milk duct orifice and send swab or aspirated material for Gram stain, microscopy, culture, and sensitivity
2. Rehydration (oral ± intravenous fluids), lemon drops
3. Stop antisialogogues
4. Oral hygiene measures and hot salt water mouthwashes
5. Commence antibiotic therapy – initially antistaphylococcal (amoxicillin/erythromycin/cephalosporin)
6. Ultrasound examination, search for a stone, followed by incision and blunt drainage, if above measures fail to produce any clinical improvement within 48 hours

Table 36.5 Microorganisms implicated in acute bacterial infections of the parotid gland and recommended first-line antibiotic therapy.¹³⁴**Gram-positive cocci**

Staphylococcus aureus (flucloxacillin)
 α -hemolytic streptococci: *Streptococcus viridans* (penicillin)
Streptococcus pneumoniae (penicillin)
Micrococcus sp. (flucloxacillin)
Peptostreptococcus sp. (anaerobic) (metronidazole)

Gram-positive bacilli (rods)

Lactobacillus sp. (co-amoxiclav)
Actinomyces (penicillin)

Gram-negative cocci

Neisseria sp. (penicillin)
Veillonella sp. (co-amoxiclav)

Gram-negative bacilli (rods)

Enterobacteriaceae: *Klebsiella* sp (ciprofloxacin)
Salmonella sp (ciprofloxacin)
Pseudomonas sp (ciprofloxacin)
Hemophilus influenza (ampicillin)
Eikenella corrodens (co-amoxiclav)
Fusobacterium nucleatum (anaerobic) (metronidazole)
Bacteroides melanogenicus (metronidazole)

Fungi

Candida sp. (fluconazole)

infection. Interestingly, an apparently normal contralateral gland will exhibit a similar reduction in flow rate in cases of recurrent subacute parotitis, when compared to controls.¹³⁸

Autoimmune salivary disease**Benign lymphoepithelial lesions**

Mikulicz (1888) described painless enlargement of the major salivary glands and lacrimal glands which in hindsight was probably a MALT lymphoma. Benign lymphoepithelial lesions (BLL is an umbrella term)¹³⁹ describes focal collection of lymphocytes which gradually form follicles seemingly identical to mucosa-associated lymphoid tissue (MALT).^{140,141}

Sjögren's syndrome

Primary Sjögren's syndrome has the histological features accompanied by dry eyes and mouth; if accompanied by a connective tissue disorder (systemic lupus erythematosus (SLE), polyarteritis nodosa, CREST (calcinosis, Raynaud phenomenon, esophageal dysmotility, sclerodactyly, and telangiectasia), dermatomyositis, systemic sclerosis) or primary biliary cirrhosis, it is secondary Sjögren's.^{142–145} Other supposed symptom complexes have been identified (sialadenitis, nodal osteoarthritis and xerostomia, SOX).¹⁴⁶ Sjögren's syndrome subjects have a 44 times increased risk of developing lymphoma¹⁴⁷ with an incidence of 5–10% of cases.^{148–150} It is estimated that 1% of the

Table 36.6 Architectural salivary gland changes with increasing severity of chronic infection (after Maynard¹³⁸).

1. Normal sialogram
2. Branch duct changes
3. Branch duct changes with sialectasis (extravasation of contrast medium through weakened walls)
4. Sialectasis with main duct changes
5. Gross main duct changes and complete dissolution of gland architecture

adult population (mean age 50 years: female predominance of 10:1) have Sjögren's syndrome.^{151,152}

The European criteria for establishing Sjögren's syndrome are shown in Table 36.7.¹⁵⁴

Salivary mucosa-associated lymphoid tissue (MALT) lymphomas

These tumors are uncommon (about 1 case per million population per annum) and were first recognized by Isaacson and Wright in 1983.¹⁵⁴ They are the most common lymphoma to affect salivary tissue and the third most common of the non-Hodgkin lymphomas. In the head and neck most occur in the parotid (parotid 88%, submandibular gland 3%, minor glands 9%). Females represent 80% of cases, ranging in age from 14–72 years. It is difficult to decipher when a swollen Sjögren's gland slips into a MALT lymphoma.^{155,157,158} Suspicious features are engorged painful parotid glands (Fig. 36.7). This symptom is usually erroneously interpreted as subacute sialoadenitis but no pus

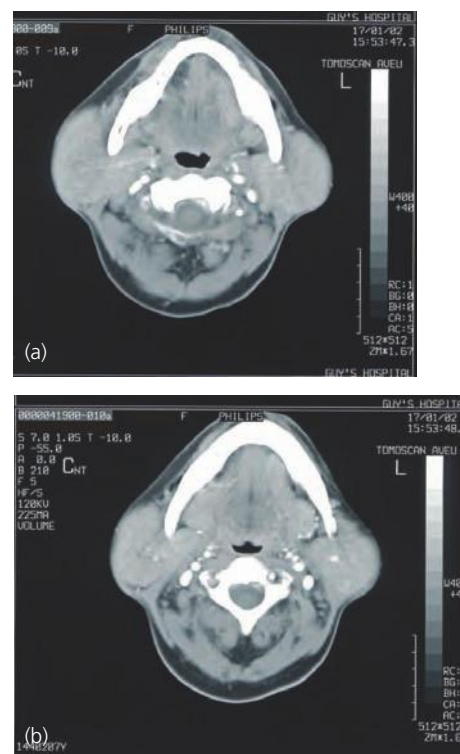
**Fig. 36.7** Long-standing parotid swelling (10 years plus) initially thought to be due to sarcoid. Tail of parotid biopsy confirmed MALT lymphoma.

Table 36.7 European criteria for the classification of Sjögren's syndrome (after Vitali *et al.*¹⁵³).**I Ocular symptoms**

A positive response to at least one of the three selected questions:

- Have you had daily, persistent, troublesome dry eyes for more than 3 months?
- Do you have a recurrent sensation of sand and gravel in the eyes?
- Do you use tear substitutes more than three times a day?

II Oral symptoms

A positive response to at least one of three selected questions:

- Have you had a daily feeling of dry mouth for more than 3 months?
- Have you had recurrently or persistently swollen salivary glands as an adult?
- Do you frequently drink liquids to aid in swallowing dry food?

III Ocular signs

Objective evidence of ocular involvement defined as a positive result in at least one of the following two tests:

- Schirmer's I test (<5 mm in 5 minutes)
- Rose Bengal score (>4 according to van Bijsterveld's scoring system)

IV Histopathology

A focus score >1 in a minor salivary gland biopsy. A focus is defined as an agglomerate of at least 50 mononuclear cells; the focus score is defined as the number of foci in 4 mm³ of glandular tissue

V Salivary gland involvement

Objective evidence of salivary gland involvement defined by a positive result in at least one of the following three diagnostic tests:

- Salivary scintigraphy
- Parotid sialography
- Unstimulated salivary flow (<1.5 ml in 15 minutes)*

VI Auto-antibodies: presence in the serum of the following antibodies:

- Antibodies to Ro(SS-A) or La(SS-B) antigens or both

Rules for classification

In patients without any potentially associated disease the presence of any four of the six items is indicative of primary Sjögren's syndrome but must include positive result from factors IV or VI. In patients with a potentially associated disease (e.g. another connective tissue disease), items I or II plus any two from among items III, IV, V are indicative of secondary Sjögren's syndrome. But if such a patient is positive for ENA antibody then this is a specific marker for primary Sjögren's and even if there is an associated CT disease the patient is classified as primary Sjögren's

Exclusion criteria

Pre-existing lymphoma, acquired immunodeficiency disease (AIDS), sarcoidosis, graft-versus-host disease, sialoadenosis. Use of antidepressant and antihypertensive drugs, neuroleptics, parasympatholytic drugs

* As it has been demonstrated that this test may be reduced in normal subjects older than 60 years, it should be excluded from the criteria or not considered indicative for a diagnosis of Sjögren's syndrome in elderly subjects.

can be milked from the gland. Unexpected vascular events (pulmonary emboli, thrombosis, leg ulcers, and anemia) are also suggestive of lymphoma. Ultrasound examination (multiple cyst formation) with the appropriate history is almost pathognomonic. A definitive diagnosis is dependent on histological examination following Tru-cut core biopsy and an assessment of clonality. This indolent disease can change into a high-grade lymphoma with about 8% succumbing to their disease.¹⁵⁶

Wegener's granulomatosis

This is an immunological disorder and salivary gland enlargement does not occur in isolation but rather with nasal, pulmonary, and renal system problems.^{159–162} The diagnosis is confirmed by circulating anti-neutrophil cytoplasmic autoantibody (ANCA)^{163–165} and biopsy.

Graft-versus-host disease

Xerostomia¹⁶⁶ and submucosal retention cysts¹⁶⁷ have been recorded during immunosuppressive therapy following bone marrow transplantation.

Non-autoimmune salivary gland disease**Amyloid**

Amyloidosis presenting as a salivary mass is exceedingly uncommon.^{168,169} It can be confirmed by biopsy.^{170,171}

Sarcoidosis

Sarcoidosis is a disease of unknown etiology characterized by non-caseating granulomas. It is a systemic illness presenting with general malaise usually between 20 and 40 years of age.^{172–177} In 35% of cases there is either unilateral or bilateral diffuse parotid gland involvement;¹⁷² the disease is usually self-limiting.¹⁷⁸ A subgroup with Heerfordt's uveoparotid fever have a triad of uveitis, bilateral parotid swelling, and cranial nerve involvement. A positive Kveim test, a raised angiotensin converting enzyme (ACE), and a positive gallium scan^{179–180} are indicative of sarcoidosis, which is confirmed through biopsy.¹⁸¹

Churg-Strauss syndrome

The syndrome consists of the triad of allergic rhinitis, asthma, and hypereosinophilia in association with a systemic vasculitis (involving small and medium-sized arteries).¹⁸² It is unmistakably a systemic disorder and not isolated to the salivary glands. Symptoms are controlled by intermittent steroids, but excision of the gland may ultimately be required.

Metabolic salivary gland disease

Endocrine and gastrointestinal disease

This complex lacks scientific scrutiny. Most patients are obese. It is possible in these individuals, that fat is directed to the parotids as well as other select areas of the body (Fig. 36.8). Not surprisingly it has been linked to diabetes mellitus,^{183–186} acromegaly, diabetes insipidus, thyroid disorders,^{187–190} gonad dysfunction,¹⁹¹ and gastrointestinal^{192–196} and liver disease.^{197–199}

Bulimia nervosa

Up to 50% of patients with bulimia nervosa have parotid gland hypertrophy.^{200–204} This is thought to be a physiological response to repeated hypochloremic alkalosis.²⁰² Affected females are painfully thin and the glands stand proud (Fig. 36.9). The consultation is usually for cosmetic reasons.

Cystic fibrosis

In cystic fibrosis, mucus plugging may cause chronic obstruction, resulting in interstitial fibrosis.^{205–207} This is another vanishingly rare problem.

Miscellaneous

Excessive starch ingestion^{208,209} and other sporadic causes of drug-induced diffuse gland enlargement,



Fig. 36.8 A symptomatic parotid swelling with unclear pathogenesis.

xerostomia or ptyalism are listed in Tables 36.8–36.10.^{210–212}

Obstructive salivary gland disease

Introduction

In the last decade a revolution has occurred in the management of obstructive salivary gland disease with the introduction of minimally invasive techniques. Stone clearance is achieved in 80% of cases and gland removal reduced from 100% to 3%.²¹³ Historically the rationale for gland removal was that a calculus induces irreversible parenchyma damage which in turn leads to chronic sialadenitis. However for those stones that migrate to the punctum rather than remain in the proximal duct, the argument is overlooked for they are released by a simple incision. The only reasonable explanation is that the policy has developed mainly around ease of surgery and not risk of subsequent sialoadenitis. There is increasing evidence that salivary glands recover function following stone removal and remain asymptomatic.^{214–218} This is the foundation on which minimally invasive treatment is based.

Investigation

A history of repeated preprandial swelling (mealtime syndrome) is almost pathognomonic of salivary gland obstruction. The exception is a salivary stricture. Stagnant saliva leads to mucus plugs which become impacted in the stricture so symptoms typically



Fig. 36.9 Young woman with a long history of bulimia and grossly enlarged parotid glands.

Table 36.8 Drug-induced xerostomia by classification.²¹⁰

Analgesics	Antipsychotics
Anorexics	Antispasmodics
Anti-acne preparations	Bronchodilators
Anxiolytics	Coronary vasodilators
Anti-arrhythmics	Catecholamine synthesis inhibitors
Anti-arthritis agents	Cough and cold preparations
Anticholinergics	Decongestants
Antidepressants	Expectorants
Tricyclics	Sedatives
Tetracyclics	Digestants and enzyme preparations
MAOIs	Diuretics
Antidiarrheal agents	Muscle relaxants
Anti-emetics	Antiparkinsonian agents
Antihistamines	Benzhexol
Antihyperlipaemics	Biperiden
Antihypertensives	Benztropine
Anti-inflammatory agents	H2 antagonists
Antinauseants	Orphenadrine
Antipruritics	Psychotherapeutic agents
Mesylate	
Cytotoxic agents	
Radiotherapy	

Table 36.9 Drug-induced ptyalism.²¹¹

Ammonium bicarbonate
Ammonium chloride
Anticholinesterases
Bromides
Ethionamide
Iodides
Ipecacuanha
Ketamine
Mercury salts
Niridazole
Parasympathomimetics

follow a period of reduced gland activity such as sleep. The obstruction is frequently released after massage which in turn is followed by a gush of salty saliva. There is a number of investigations that complement clinical assessment (Table 36.11).

Ultrasonography

The main salivary glands (parotid, submandibular, and sublingual) are assessable to sonographic evaluation (frequencies of 7.5–13.5 MHz) which is available in most health care systems. A stone >2 mm diameter is reliably detected as an echo opaque reflection with distinct distal shadowing (Fig. 36.10). A dilated duct proximal to the echo clinches the diagnosis and dilation without an echo suggests the stricture. The

Table 36.10 Drug-induced salivary gland enlargement.²¹²

Corsodyl
Insulin*
Iodides*
Iodine-based contrast media
Isoprenaline*
Methyldopa *
Nitrofurantoin
Nifedipine
Oxyphenbitazone *
Oxyphenbutazone
Phenothiazines*
Phenylbutazone*
Potassium chlorate*
Sulfonamides*
Thiocyanate*
Thiouracil*

* Enlargement associated with pain.

Table 36.11 Optimum imaging modalities for salivary obstruction.

1. Ultrasonography
2. Sialogram
3. Plain film radiograph
4. Sialoendoscopy
5. MRI/CT scans
6. Virtual reality images

parenchyma can also be assessed: a normal salivary gland has a diffuse homogeneous appearance and one with chronic inflammation has a coarse image due to cyst formation and fibrosis.

Consequently ultrasound imaging can be used to monitor recovery following treatment and portable ultrasound machines are an invaluable adjunct to current clinical practice.

Sialography

A sialogram is also a valuable investigation (Fig. 36.11) for it can identify the size, position, and mobility of a stone (the latter has an impact on the choice of treatment) but its main advantage over ultrasound is its ability to provide information on the number and shape of strictures (single, discrete, diffuse, multiple). A sialogram should be avoided when the gland is clinically infected or the patient is allergic to iodine. An air bubble introduced inadvertently during sialography can mimic a salivary stone.

Plain film radiography

The average submandibular duct stone can be easily demonstrated by a mandibular true lower occlusal view, while large hilar stones can be seen on the panoramic or an oblique lateral radiograph. The only real

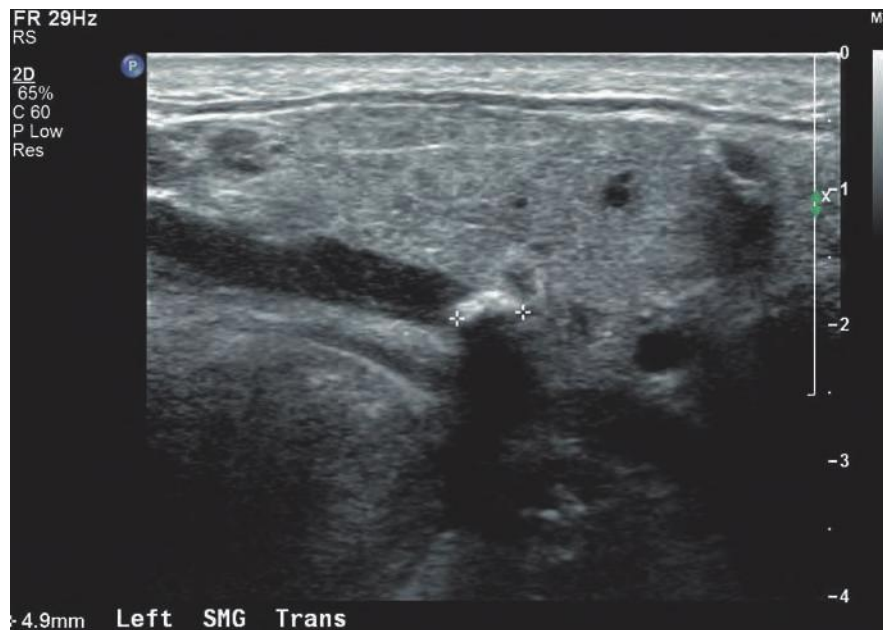


Fig. 36.10 Ultrasound scan demonstrating a 4.9mm calculus at the hilum of the submandibular gland.

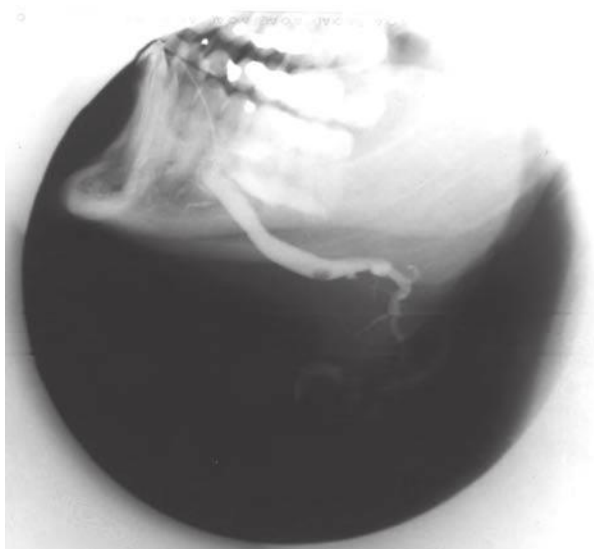


Fig. 36.11 Submandibular sialogram showing calculi. The fact that the duct is dilated along its length suggests the stones are mobile and suitable for basket retrieval.

use of plain radiography is to detect small stones (<2mm) resident in the distal portion of the submandibular or parotid ducts; it is more sensitive if the recommended radiation exposure is reduced for these views. However, this imaging modality has limited application in obstructive disease.

CT and MRI scans

CT and particularly MRI imaging can be used, in the absence of simpler modalities, to demonstrate stones and chronic inflammation²¹⁹ but they should really be reserved for evaluating salivary tumors. MRI has limitations as it is sometimes difficult to distinguish between bone and calcified tissue which, together with blood vessels, appear black. Consequently calcified bodies such as small stones may be over-

looked.^{219,220} The use of 3D rapid spin-echo sequences has improved the detection of stones²²¹ (91%) and strictures (100%) but CT scans frequently produce artefacts and are prone to produce false-positive results. Both modalities can be used with sialography.

One new intriguing application of this technology is the computer manipulation of MRI data to provide virtual endoscopic pictures of the salivary duct.^{222,223} The disadvantages of MRI are its cost, restricted access, and inability to apply it to patients with pacemakers or ferromagnetic implants. In practice there are simpler, easier, and less expensive methods of detecting an obstruction in the parotid duct.

Sialolithiasis

Demographics

The largest published series of salivary stones ($n = 4691$) shows a remarkable similarity in demographic data between five independent salivary units (Table 36.12).²¹³

There is usually a significant delay (greater in the submandibular gland) between the initiation of symptoms and seeking a medical opinion consultation (parotid 4.8 years, submandibular gland 5.4 years) and this correlates with submandibular stones being larger than parotid calculi.²²⁴ The medical consultation is normally triggered by the advent of acute or subacute sialadenitis which signals a move to chronic sialadenitis.

The analysis of hospital admission data in the British NHS system established a hospital admission rate for sialoadentitis and sialolethiasis of 59 cases per million annually. Calculated over a lifetime (70 years) this gives a prevalence of 0.45% for acute symptomatic disease.²²⁵ The anatomical distribution of calculi

Table 36.12 Demographic data relating to $n = 4691$ salivary calculi treated at five centers specializing in minimally invasive therapy.²¹³

Parameter	Erlangen	Israel	London	Milan	Paris
Male/female	1.11	0.98	1.03	1.04	1.02
Mean age	43	45	46	46	46
Mean age parotid	50	46	48	49	44
Mean age submandibular	41	43	45	44	42
Mean size parotid stone (mm)	5.8	5.3	5.8	4.7	5.1
Mean size submandibular stone (mm)	7.7	7.1	7.3	7.0	6.9
Mean duration symptoms parotid (month)	37	24	44	50	34
Mean duration symptom submandibular	23	18	49	43	27

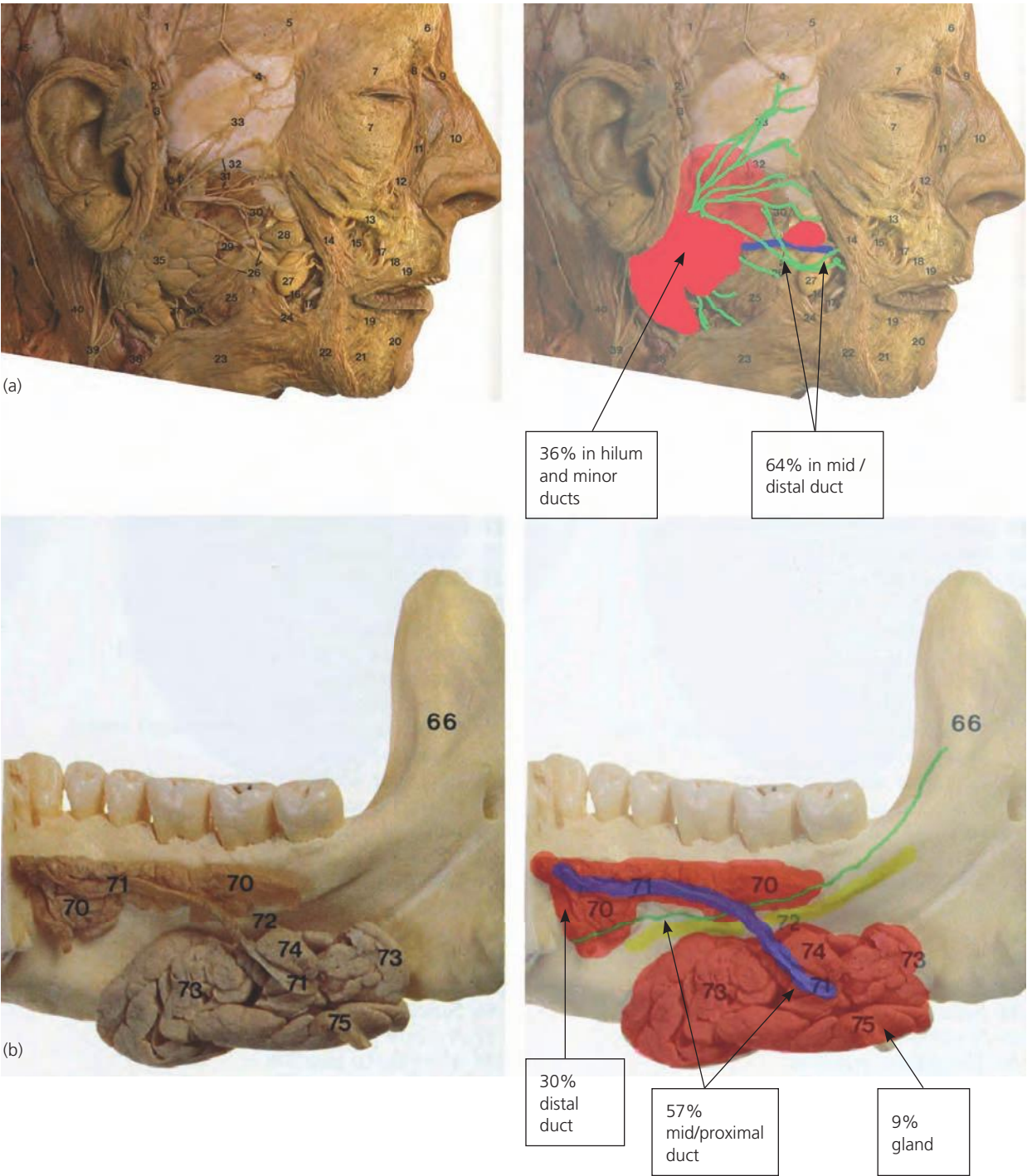


Fig. 36.12 Site distribution of salivary calculi in the parotid and submandibular glands.

shows that 63–94% occur in the submandibular gland and the remainder occur in the parotid gland (Fig. 36.12). The wide variation is due to the reluctance of patients with parotid stones to submit to surgery. Consequently a reservoir of patients with parotid stones can build up within pockets of the community. Multiple stones occur in 7% of parotid and 13% of submandibular glands but stone formation in more than one salivary gland is uncommon (<1%).²²⁵

Pathogenesis of salivary stones

The steps by which salivary gland stones develop have not been fully elucidated. It is probable that this process is multifaceted with one important item being the anatomical configuration of the salivary ducts. Both duct systems have kinks (90% bend) and stones seem to congregate at these kinks (Fig. 36.13).

It is hypothesized that a calculus forms from a nidus of debris. Calcium-binding proteins are present in both the salivary and the renal systems and a deficit of crystallization inhibitors has been proposed as a cause of kidney stone formation.²²⁶ No such association has been demonstrated in the salivary glands nor is water hardness relevant.²²⁷

Harrison has shown that the salivary glands produce a continual stream of microcalculi²²⁸ which are a protective mechanism, whereby calcium ions neutralize active phospholipids exposed within dam-

aged cells.²²⁹ Harrison holds that the microcalculi cause obstruction followed by focal atrophy which leads to ascending infection and propagation of the cycle. A more likely scenario is that microcalculi are continually washed down the duct system and through a combination of chance and transient local conditions (inflammation, mucus plug, stagnation) the debris comes to lodge at a kink in the duct and subsequently acts as a nidus for calcium precipitation. This would account for the orderly precipitation depicted by calcium rings with a stone (Fig. 36.14).²³⁰

The submandibular stone has a significant inorganic component (80%) and tends to be radiopaque (94%) whereas the parotid sialolith is formed of 50% organic material and 43% are radiopaque.²³²

Pathogenesis of salivary strictures

The origin of salivary strictures is unclear. An analysis of over 1300 consecutive sialograms undertaken for obstructive symptoms²³³ demonstrated that approximately 25% of symptoms were due to stricture and the vast majority of these strictures occurred in the parotid gland (Table 36.13). Strictures can be pinpoint, multiple (giving a sausage-like appearance) or diffuse along the length of the duct (Fig. 36.15).

There is no apparent relationship to autoimmune diseases but strictures do occur following repeat treatment with radio iodine. The sialoendoscopic appearance shows circular bands of pale scar tissue reminiscent of tracheal rings, but, without understanding the pathogenesis, therapeutic intervention remains empirical. Why most strictures occur in the parotid gland remains a mystery.

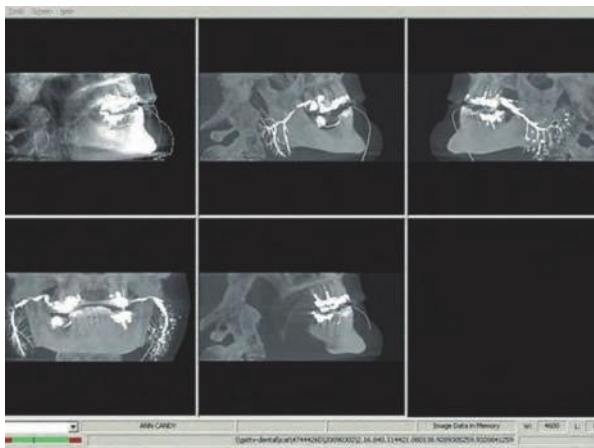


Fig. 36.13 Long cone-beam CT sialogram demonstrating the parotid duct system.



Fig. 36.14 Micrograph of salivary calculus showing rings of calcium precipitation.

Table 36.13 Cause of symptoms in 1362 consecutive sialograms undertaken for obstructive symptoms. In 485 the duct was normal and in 877 there was evidence of obstruction (stones 642, strictures 198, plugs 37).²³³

	% of total	Parotid	Submandibular
Salivary strictures (<i>n</i> = 198 cases)	~24%	78%	22%
Mucus plugs (<i>n</i> = 37)	~3%		
Salivary stones (<i>n</i> = 642)	~73%	28%	72%
Radiopaque		43%	94%
Duration		4.8 years	5.4 years
Size		6.6 mm	8.5 mm

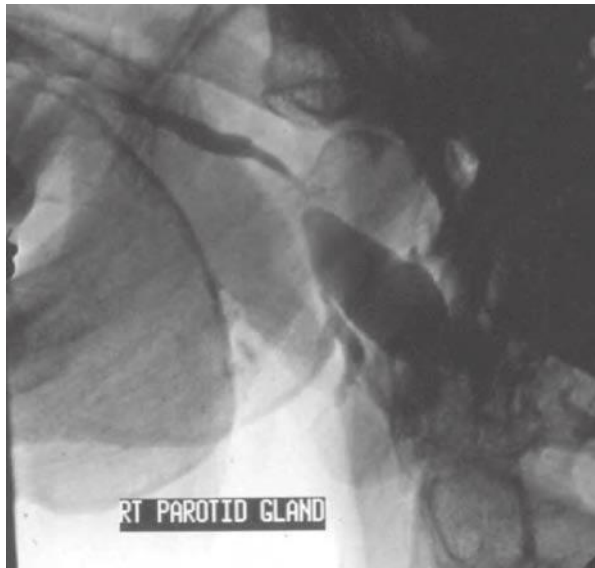


Fig. 36.15 Sialogram showing a point stricture in a parotid duct with gross proximal dilatation.

Modern management of salivary calculi

The advent of minimally invasive salivary therapy was ushered in by innovations in the management of renal calculi. A salivary extracorporeal lithotripter (ECL) became commercially available (Storz Minilith: Storz Medical, Kreuzlingen, Switzerland) in the mid-1990s followed in 2000 by first-generation microendoscopes (Fig. 36.16), which then raised the possibility of intracorporeal lithotripsy (ICL). This proved a false dawn, for the salivary microenviron-

ment could not withstand the energy used in ICL without stricture formation.

Rationale of management

The armamentarium available for minimally invasive therapy includes an ECL (Storz Minilith), salivary endoscopes, microballoons, and an array of wire baskets and microforceps. These instruments should be used in combination and treatment is governed by stone size. Mobile stones <5 mm diameter are amenable to retrieval by basket, forceps or balloon under either radiological or endoscope control.²³⁴ In practice the basket is the easiest method of stone retrieval.

Stones >5 mm or fixed to the duct wall require alternative techniques. ECL is not very effective in eliminating submandibular stones (~30% stone clearance) when compared to the parotid (60%).²³⁵ Consequently large (>5 mm) or fixed submandibular stones are retrieved by endoscope-assisted surgery whereas those in the parotid (6–10 mm) are targeted by ECL. Large parotid stones (>10 mm) do not respond to ECL and similarly have to be removed by endoscope-assisted surgery (Fig. 36.17).

Intervention sialography

The technique commences with a sialogram. The salivary ducts have pressure sensors within their walls and to avoid discomfort the duct should be washed with 1% lidocaine or articaine solution. A dormia basket is advanced under fluoroscopic control until it passes the stone. The basket is then opened, rotated

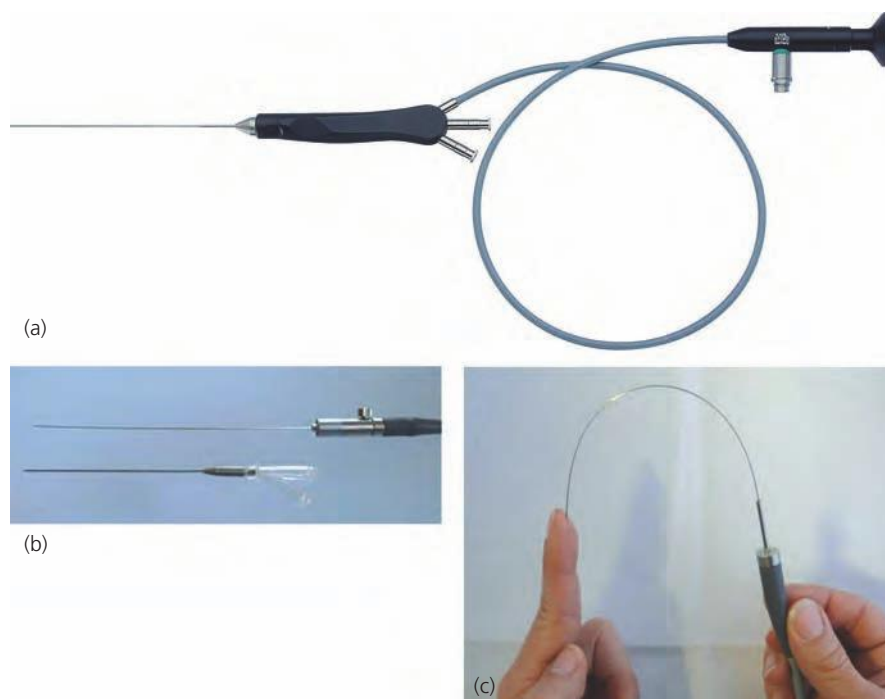


Fig. 36.16 Examples of third generation endoscopes produced by Storz (a) and Polydiagnost (b, c). One is based on a reusable multichannel unit (Storz), the other on a disposable design that minimizes concerns with sterilization. Both are delicate instruments but possess greater flexibility than their predecessors.

	Parotid	SMG
Stone <5 mm	BASKET	BASKET
Stone >5 mm or fixed	Lithotripsy ↓ Failed lithotripsy (10%) ↓ Endoscope-assisted surgery	Endoscope-assisted surgery

Fig. 36.17 Flow diagram demonstrating the management protocol for salivary stones.



Fig. 36.18 Dormia basket retrieval of a SMG stone. Usually the punctum has to be incised to release the calculus.

and withdrawn to engage the calculus, which is withdrawn to the papilla and released (Fig. 36.18). If the selection criteria are used appropriately, the technique of basket retrieval is very effective in eliminating stones (Table 36.14).

In a series of patients treated at Guy's Hospital²³⁸ the clearance rate using current criteria was 86% (submandibular 87%, parotid 86%). Morbidity is minimal but there is a risk of inducing sialoadenitis; it is recommended that those with a history of infection should receive prophylactic antibiotics for 3 days.

Sialoendoscopy

Sialoendoscopy was initially used for diagnostic purposes but it is now routinely used therapeutically. The parotid duct is easy to cannulate (after dilation with a Nettleship dilator) whereas the submandibular punctum is usually incised and sutured open at the end of the procedure. The duct lumen is inflated with pulses of saline solution and the endoscope advanced under direct vision until the stone is identified. The basket is introduced either down the working channel of the endoscope or within the duct lumen parallel to the endoscope. As the basket is withdrawn it should be rotated to engage the stone and then both instruments retrieved together. A contraindication is a distal stenosis that prevents either entry of the

endoscope or retrieval of the stone past the stricture (once secured a stone cannot be released from a basket!). The reported success rate is 82% (Table 36.15).

Morbidity is minimal, but there is a small risk (2–8%) of strictures, subacute sialoadenitis 2–3%, ductal perforation 0–8.5%, basket block 0–6% or ranula formation 0.6–0.9%. Most of these complications are avoidable.

Lithotripsy

The shockwave generated by the ECL (Fig. 36.19) produces a compressive wave which passes through the calculus producing stress fractures. At the same time, the pressure wave induces vapor bubbles at the stone's surface. As the bubble collapses a jet of water strikes the stone drilling tiny holes in its surface.

It has become apparent that the chance of stone clearance is better in the parotid gland and with stones <8mm in diameter.²⁵¹ In 2006, Escudier (Table 36.16: unpublished data) undertook a prospective randomized study to establish factors that predicted for stone clearance. The only independent factor predicting for stone fracture was the size of stone.

The two factors that guide patient selection are the size and site of stone. The stone clearance rate in six published series with over 100 cases in each varied between 29 and 63%. The average clearance was 41% (Table 36.17).

Lithotripsy technique

The Storz sialolithotripter is designed for application in the outpatient setting. It is customary to leave about a week between treatments (due to a tender gland), but if necessary ECL can be undertaken on 2 or 3 consecutive days. The shockwaves are targeted with an ultrasound probe (Storz 7.5MHz) at a

Table 36.14 This series of articles contains an overlap of cases. If repeat counting is removed, the improvement in results over time is apparent.³¹⁶

Authors	n =	Parotid	Submandibular gland	Overall
Drage <i>et al.</i> 2000 ²³⁶	25	2/5 40%	8/20 40%	40%
Brown 2002 ²³⁷	61	20/25 80%	25/35 74%	74%
McGurk <i>et al.</i> 2005 ²³⁵	80	22/26 85%	47/54 87%	86%

Table 36.15 Summary of outcome of 12 studies relating to interventional sialoendoscopy (using semi-rigid endoscopes) for sialolithiasis. The mean clearance rate was 82% with only 6% of glands removed. Figures in parentheses are percentages based on 577 cases for whom data are available. (P, parotid; SM, submandibular; SL, sublingual; NK, not known.)³¹⁶

Author	Year	Number of patients	Gland	Complete success	Residual obstruction	Sialoadenectomy
Marchal <i>et al.</i> ²³⁹	2001	50	P	44 (88.0)	6 (12.0)	1 (2.0)
Marchal <i>et al.</i> ²⁴⁰	2002	106	SM	90 (84.9)	16 (15.1)	5 (4.0)
Nahlieli <i>et al.</i> ²⁴¹	2003	217	151 SM 65 P 1 SL	131 (86.8) 54 (83.1) 1 (100)	20 (13.2) 11 (16.9) 0	15 (6.9)
Chu <i>et al.</i> ²⁴²	2003	13	SM	11 (84.6)	2 (15.4)	0
Nakayama <i>et al.</i> ²⁴³	2003	1	SM	1 (100)	0	0
Zenk <i>et al.</i> ²⁴⁴	2004	13	11 SM 2 P	2 (18.2) 2 (100)	9 (81.8) 0	0
Ziegler <i>et al.</i> ²⁴⁵	2004	54	35 SM 19 P	29 (82.9) 19 (100)	6 (17.1) 0	6 (11.1)
Koch <i>et al.</i> ²⁴⁶	2005	21	10 SM 11 P	5 (50.0) 6 (54.5)	5 (50) 5 (45.5)	NK
Yu <i>et al.</i> ²⁴⁷	2008	4	4 P	3 (75.0)	1 (25.0)	NK
Yu <i>et al.</i> ²⁴⁸	2008	27	27 SM	22 (81.5)	5 (18.5)	5 (18.5)
Papadaki <i>et al.</i> ²⁴⁹	2008	73	NK	62 (84.9)	11 (15.1)	5 (6.8)
Walvekar <i>et al.</i> ²⁵⁰	2008	26	16 SM 10 P	14 (53.8)	12 (46.2)	NK
Mean		605		496 (82.0)	112 (18.0)	36* (6.2)

Table 36.16 Prospective study of factors that predict for stone clearance showed stone size to be the only independent variable. Partly cured cases represent patients who were symptom free but had retained stone fragments in the duct. (Escudier M *et al.* Salivary gland lithotripsy. *Laryngoscope*, in press.)

Variable	Fully cured Multivariate OR (95% CI)	Partially cured Multivariate OR (95% CI)
Age	1.02* (1.00–1.03)	1.01 (0.98–1.03)
Size	0.85** (0.75–0.96)	0.95 (0.85–1.05)
Number of shocks	0.99 (0.92–1.06)	0.98 (0.89–1.09)
Gender	1.06 (0.51–2.22)	0.99 (0.32–3.00)
Duration	0.99 (0.99–1.00)	1.00 (0.99–1.00)
Levels	1.22 (0.73–2.03)	0.39** (0.20–0.76)
Radiodensity	0.73 (0.28–1.89)	3.52 (0.52–23.49)
Site of stone (gland)	1.81 (0.51–6.46)	14.62** (1.74–122.43)

** P < 0.05, * P < 0.10.

frequency of 2 Hz (maximum pressure 36 MPa) usually over an hour (3000–5000 shocks per session) and with no requirement for analgesia.

If stone fragments persist after the initial course of three to four sessions, a second course can be under-



Fig. 36.19 Storz sialo-lithotripter.

taken. The outcome of treatment is usually assessed by ultrasound examination 4–6 months after completion of treatment and the long-term outcome is encouraging. In a 10-year review, patients rendered stone free did not develop recurrent disease (Table 36.18).

Gland-preserving surgery (endoscope-assisted stone retrieval)

Gland-preserving surgery is the treatment of choice for large or fixed submandibular stones and is usually reserved for those 10% of patients with parotid stones that remain symptomatic after ESWL. If no lithotripsy service is available large parotid stones can be reliably retrieved by this technique. The success rate for both techniques is in the region of 95% stone retrieval.

Table 36.17 Overall success rates for extracorporeal shock wave lithotripsy in 6 studies with 100 or more cases of salivary calculi. Stone clearance rate was 41% of cases treated. The differential clearance between parotid and submandibular gland was 53% vs 31%. Figures in parentheses are percentages.³¹⁶

Study	Year	Lithotripter	Number of cases	Degree of success		
				Total	Partial	Failure
Kater <i>et al.</i> ²⁵²	1994	Electromagnetic – Minilith	104	40 (38.4)	19 (18.3)	45 (43.3)
Katz ²⁵³	1998	Electromagnetic – Minilith	200	126 (63.0)	68 (34.0)	6 (3.0)
Escudier <i>et al.</i> ²⁵⁴	2003	Electromagnetic – Minilith	122	40 (32.8)	43 (35.2)	39 (32.0)
Zenk <i>et al.</i> ²¹⁷	2004	Piezoelectric – Piezolith 2500	197	58 (28.9)	139 (71.1)	0.0
Capaccio <i>et al.</i> ²⁵⁵	2004	Electromagnetic – Minilith	322	145 (45.0)	88 (27.3)	89 (27.7)
Schmitz <i>et al.</i> ²⁵⁶	2008	Electromagnetic – Minilith	167	51 (31.0)	57 (34.1)	59 (35.3)
Overall			1112	460 (41.4)	414 (37.2)	238 (21.4)

Table 36.18 Long-term outcome (in three studies at 10 years, 6 years, and 3 years, respectively) following ESWL. Those cured of stone do not suffer further problems. Figures in parentheses are percentages.³¹⁶

	Parotid outcome				Submandibular outcome			
	Number of patients	Cured	Partial success	Failure	Number of patients	Cured	Partial success	Failure
Zenk <i>et al.</i> 2004 ²¹⁷	0	0	0	0	191	67 (35.0)	29 (15.0)	95 (50.0)
Capaccio <i>et al.</i> 2004 ²⁵⁵	88	61 (69.3)	24 (27.3)	3 (3.4)	234	84 (35.9)	64 (27.4)	86 (36.7)
Schmitz <i>et al.</i> 2008 ²⁵⁶	40	18 (45.0)	9 (22.5)	13 (32.5)	127	33 (26.0)	48 (37.8)	46 (36.2)
Overall	128	79 (61.7)	33 (25.8)	16 (12.5)	552	184 (33.3)	141 (25.5)	227 (41.1)

Endoscope-assisted retrieval of submandibular calculi

This technique is reserved for stones in the middle or proximal portion of the submandibular gland. It can be performed in the outpatient or preferably a day-case setting. Two techniques have been developed which are similar except for one feature; the first lays open the duct (marsupialized to the floor of mouth) while the second leaves the duct intact (Fig. 36.20).^{257,238}

Palpable stones in the floor of mouth are ideally suited to this technique²⁵⁸ but repeat surgery is difficult and should be avoided. At the time of surgery an endoscope can be used to guide the surgeon and to check that no additional stones are present in the gland. The mean stone clearance rate is 92.1% with gland removal rate of 2.1% (Table 36.19). This represents a substantial change in clinical practice.

Endoscopic-assisted removal of parotid calculi

The first extra-oral parotid sialolithotomy without parotidectomy was described in 1991 by Baurmash

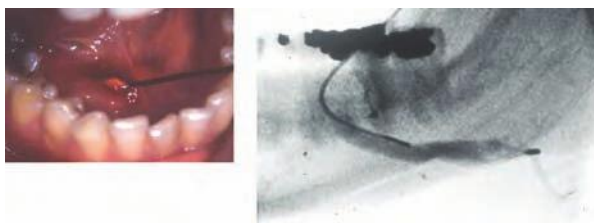


Fig. 36.20 Typical dissection of the floor of mouth to retrieve a stone from the hilum of the submandibular gland.

and Dechiara.²⁶⁷ The sialoendoscope helps to identify the duct for the light at the end of the endoscope acts as a beacon to direct the surgeon on to the stone (Fig. 36.21). The direct transcutaneous technique is applicable to large calculi situated on the anterior edge of the masseter muscle where the parotid duct is superficial. In contrast, proximal stones lie deep in the gland and it is here the endoscope shows its worth. The light at the end of the endoscope is used to mark the position of the stone and a preauricular skin flap is raised to expose the duct and stone. The buccal branch of the facial nerve runs parallel to the parotid cut and is easily visualized in the dissection. Prophylactic antibiotics are prescribed and a stent is optional. The data suggest^{268–270} that over 90% of



Fig. 36.21 The light at the end of the sialoendoscope is used as a guide to identify the duct and the position of the stone prior to surgical release.

Table 36.19 Outcome of 11 studies relating to transoral removal of stones. Figures in parentheses are percentages.

Authors	Number of patients	Stone free	Residual obstruction	Sialoadenectomy
Novotny 1989 ²⁵⁹	17	17 (100)	0 (0)	0 (0)
Zenk 2001 ²⁶⁰	116	102 (87.9)	14 (12.1)	4 (1.7)
Bodner 2002 ²⁶¹	5	5 (100)	0 (0)	(0)
Makdissi <i>et al.</i> 2004 ²¹⁵	43	42 (97.7)	1 (2.3)	4 (9.3)
McGurk <i>et al.</i> 2004 ²⁶²	55	54 (98.2)	1 (1.8)	5 (9.1)
McGurk <i>et al.</i> 2005 ^{a224}	143	137 (95.8)	6 (4.2)	6 (4.2)
Capaccio <i>et al.</i> 2005 ²⁶³	11	9 (81.8)	2 (18.2)	0 (0)
Zenk <i>et al.</i> 2005 ^{b264}	638	583 (91.4)	44 (6.9)	11 (1.7)
Park <i>et al.</i> 2006 ²⁵⁸	172	156 (90.7)	5 (2.9)	3 (1.7)
Kim & Park 2007 ²⁶⁵	13	13 (100)	0 (0)	0 (0)
Roh & Park 2008 ²⁶⁶	59	54 (91.5)	5 (8.5)	2 (3.3)
Overall ^c	1058	974 (92.1)	62 (5.9)	22 (2.1)

^a Included the cases from Makdissi *et al.* (2004)²¹⁵ and McGurk *et al.* (2004).²⁶²

^b Included the cases from Zenk *et al.* (2001).²⁶⁰

^c Excludes duplicate cases.

Table 36.20 Results of two studies relating to endoscopically assisted removal of parotid calculi. Figures in parentheses are percentages.³¹⁶

Authors	Year	Number of patients	Stone free	Residual obstruction	Sialoadenectomy
Nahlieli <i>et al.</i> ²⁶⁸	2002	12	9 (75.0)	1 (8.3)	2 (16.7)
McGurk <i>et al.</i> ²⁶⁹	2006	7	6 (85.7)	0	1 (14.3)
Overall		19	15 (78.9)	1 (5.3)	3 (15.8)

stones can be retrieved successfully from the parotid (Table 36.20). An update of combined results (Guy's Dental Hospital, London, UK, the Barzilai Medical Centre, Ashkelon, Israel, and the Indiana University Medical Center, Indianapolis, USA) in 69 patients treated with the endoscopically-assisted open surgery and with a median follow-up of 25 months (range of 2–81 months) reported a stone clearance rate of 66/67 (98.5%). In general the complication rate is low (4.5%) and includes stricture, retained fragments, and scar formation.²⁶⁹ The downside is that the procedure requires hospital admission and treatment under general anesthesia.

Results of minimally invasive approach to salivary stones

In the last two decades, five centers have pioneered the use of minimally invasive treatment for the management of salivary calculi. One common theme is that no one technique was used in isolation. Treatment protocols have slowly been developed that are broadly similar in the five units. In the period 1990–2004, 5258 patients with 5993 salivary stones were seen in the five salivary centers.²¹³ Those patients who had stones released by a simple intraoral procedure or refused treatment ($n = 241$) were excluded as were those who had complete dental stenosis ($n = 11$), recurrent infective episodes or multiple stones neces-

sitating gland removal ($n = 63$) or they were lost following the initial assessment ($n = 11$). There was a learning curve and a number of patients were treated by minimally invasive techniques which have since been abandoned (intracorporeal lithotripsy). These cases ($n = 237$) were also excluded. The remaining 4691 consecutive cases treated by a minimally invasive approach were evaluated. The results are shown in Fig. 36.22.

Overall salivary stones were successfully removed in 3775/4691 (80.47%) of cases. Partial success (symptom-free but retained stone debris) was achieved in 782/4691 (16.67%) and these patients have not received further treatment or were lost to follow-up. Treatment failed in 134/4691 (2.8%) of cases resulting in salivary gland removal. Currently, if these patients were seen at a non-specialist unit, they would all have the gland removed.

Salivary gland tumors

New developments

- Warthin's tumor is a polyclonal lesion and not a neoplasm.
- Benign parotid tumor can be safely treated by minimal procedures much less than the traditional superficial parotidectomy.

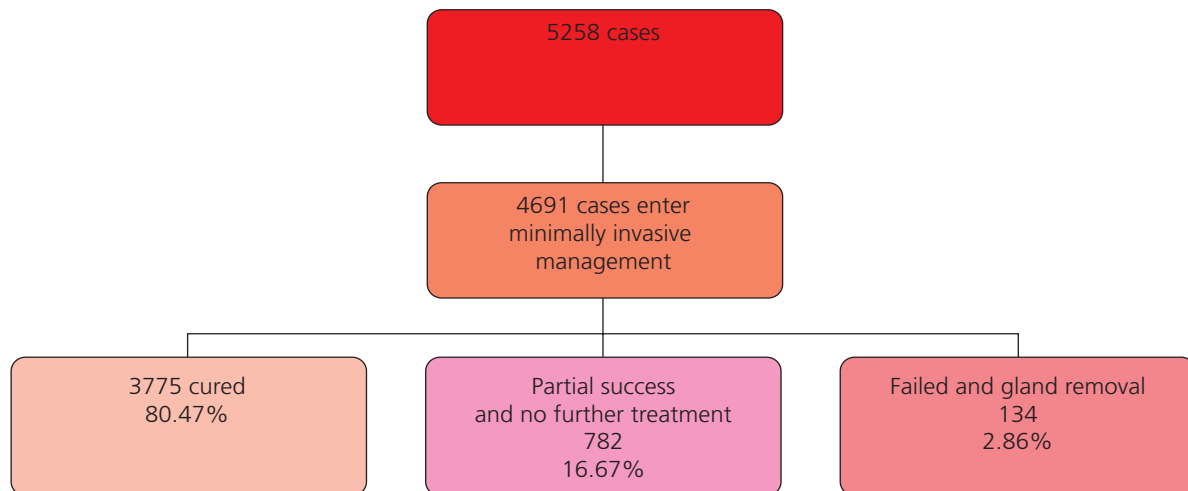


Fig. 36.22 Results of minimally invasive therapy for salivary stones ($n = 4691$).²¹³

- Management of malignant salivary tumors should be governed mainly by stage rather than grade (+ histological type). The 4 cm rule is important.

General epidemiology

Until recently comprehensive population data for salivary gland tumors were incomplete, as benign neoplasms are not recorded by cancer registries and minor salivary gland cancers are coded with oral malignancy. There is supposedly a wide variation in the incidence between countries²⁷¹ with an increase reported in the USA from 6.3% of head and neck cancers in 1974 to 8.1% in 1999.²⁷² It is unclear whether this change is real or a feature of improved registration.

Clarity has arrived with a unique population-based study²⁷³ in a defined UK population of 604200 patients served by only two hospitals. The records for the period 1988–1997 were scrutinized and in total 517 patients with salivary gland tumors identified (Table 36.21).

It is notable that the benign:malignant ratio of tumors at each site did not conform to standard teaching. The proportion for the three main sites – parotid, submandibular, and minor salivary glands – was 6%, 34%, and 24% respectively. Previous data originated from large cancer institutes where a referral bias in favour of malignant disease may have distorted the data. The annual incidence for benign and malignant salivary tumors over the 10-year study period is shown in Table 36.22. The incidence of benign tumor was 7.2 and that for cancer 0.8 per 100 000. Data on malignant disease were comparable to National Cancer Registry returns, suggesting the remaining data are valid.

Etiology and risk factors

The causes of salivary gland tumors are largely unknown. A well established association exists with radiation which has been studied in the Japanese

Table 36.21 The proportions of benign and malignant salivary gland tumors in a defined UK population by anatomic site (1988–1997). The traditional 10:1:1 rule holds true for distribution of tumors between parotid, submandibular, and minor salivary glands. Figures in parentheses are percentages.

	Benign	Malignant	Total
Major salivary glands			
Parotid gland	397	26 (6)	423
Submandibular gland	35	12 (34)	47
Minor salivary glands	32	10 (24)	42
Sublingual gland	0	5 (100)	5
Total	464	53	517

Table 36.22 Annual incidence (per 100 000 population) of salivary gland neoplasms by site in defined UK population.

	Benign	Malignant
Major salivary glands		
Parotid gland	6.20	0.40
Submandibular gland	0.55	0.19
Minor salivary glands	32.00	0.16
Sublingual gland	0.00	0.08
Total	7.20	0.80

population surviving the atomic bomb.²⁷⁴ Assertions^{274–277} have been made that certain tumors (Hodgkin's disease, medulloblastoma, basal cell carcinoma) carry a secondary risk of salivary cancer. What these tumors have in common is that they all originated in the head and neck and were treated by radiation. The environmental factors that drive salivary glands towards neoplastic change remain largely unknown.

Investigation

A careful clinical history and examination are the foundation on which all subsequent investigations are based. As stated before, it is essential that lesions in the tail of the parotid or submandibular gland be

considered within the differential diagnosis of a lump in the neck. Tumors hidden in the retromandibular and submandibular area are more difficult to evaluate. The initial investigation of choice is ultrasonography which will immediately establish if the mass is intrinsic to the salivary gland or related to adjacent lymph nodes. The majority of cancers in the submandibular triangle are not of salivary gland origin and in sunny climates (Australia) the commonest cause of a malignant parotid lump is a metastasis from a tumor of the scalp.

Complex salivary gland masses that run deep to the mandible or are suspected of being malignant should be visualized by CT or preferably MRI scans. MRI offers superior soft tissue contrast and artifacts caused by dental fillings usually play a minor role on scanning. The greatest drawbacks with MRI are the long scan times and the claustrophobic environment that it demands. These imaging modalities should be complemented by the free use of fine-needle aspiration cytology. The latter is operator dependent but experienced cytologists can achieve accuracy rates of 87–96%.²⁷⁸ Best results are obtained when cytologists and radiologists work together which reduces the incidence of negative taps. The application of FNAC varies between clinicians. It has an obvious role in distinguishing benign from malignant disease but in many incidences a malignant disorder will be suspected on clinical grounds alone. In high-grade disease an open or Tru-cut biopsy is the investigation of choice. Paradoxically FNAC has a particular role when tumor is not anticipated. Thus in childhood, many lumps are inflammatory in origin and FNAC may avoid the need for parotid surgery. Similarly it will detect tuberculosis and if a benign tumor in an elderly or infirm individual can be confirmed then surgery can be avoided.⁴

It is important that all submandibular masses undergo FNAC. When compared to the parotid, the risk of encountering a malignant tumor is greater (34–50% vs 6–15%) but more importantly submandibular gland cancers carry 20% worse prognosis for no other reason than they are frequently misdiagnosed and inadequately excised.²⁷⁹

One other specific indication for FNAC lies in children with a suspected parotid tumor. Salivary tumors are uncommon in childhood but the risk of encountering a malignant tumor is greater than in the adult (34% vs 10%). It is imperative to try and avoid adjuvant radiotherapy in this group and if the surgeon is forewarned every attempt can be made to obtain adequate clearance to minimize the need for radiation therapy.

Intraoperative facial nerve monitoring

Superficial parotidectomy (SP) is little more than a dissection of the nerve. It is therefore not surprising that intraoperative neurophysiological monitoring

has been advocated in this type of surgery.²⁸⁰ It seems that for experienced surgeons, the incidence of facial nerve injury is not reduced,²⁸¹ but the technique may be an advantage for those in training. The medico-legal dimension of monitoring cannot be ignored, the equipment is relatively inexpensive, easy to use, minimally invasive, and may on occasion reduce the extent (rather than incidence) of nerve injury through constant feedback during surgery.

Benign parotid tumor

The majority of benign tumors are represented (93%) by two histological types – pleomorphic adenoma (71%) and Warthin's tumor (22%). In the last decade there has been a fundamental reappraisal of these two tumors. Pleomorphic adenomas had a fearful reputation for recurrence, which is not deserved, and the rationale for wide excision (SP) was based on this reputation. Similarly it is now appreciated that Warthin's tumor is a polyclonal lesion and not a neoplasm, which may manifest with synchronous or metachronous lesions. In both instances a more conservative approach to treatment is possible.

The traditional surgical approach to benign parotid tumors is a superficial parotidectomy (SP) and that for deep lobe tumors is a total parotidectomy (TP). Modern modifications include partial superficial parotidectomy and selective deep lobe parotidectomy.²⁸²

Traditional parotidectomy

An essential step in traditional superficial parotidectomy is the identification of the facial nerve and a prerequisite for safe surgery is wide exposure. This is achieved through a variety of preauricular incisions (lazy S: facelift). It is a useful exercise to release the parotid gland from the confines of the deep cervical fascia by freeing the fascia from the sternomastoid muscle, mastoid process, and tragus (the greater auricular nerve is ideally preserved). The increased mobility allows tumors wedged between the mandible and the mastoid to become more accessible. Using the avascular pretragal plane of dissection, the parotid is displaced anteriorly to identify the facial nerve. The main trunk is located halfway between the bony external auditory meatus and the tip of the mastoid at the medial aspect of the tympanomastoid fissure. The extent of the nerve dissection is governed by the location of the tumor. If the tumor is small only a quadrant of the parotid gland need be removed (partial superficial parotidectomy). Parotid surgeons should be equally adept at both retrograde and antegrade nerve dissection.

A total parotidectomy (TP) accompanies tumors deep to the facial nerve where the exposed branches of the nerve are lifted to access the tumor. With this additional stripping of the nerve comes an increased risk of permanent and temporary nerve injury (SP

1–2% permanent, 30% temporary vs TP 4–6% permanent, 60% temporary). Occasionally a parotid tumor extends into the parapharyngeal space and is usually removed following a total parotidectomy. This may necessitate a vertical submandibular osteotomy and division of the stylomandibular ligament or posterior belly of the digastric, either alone or in combination in order to improve access. Once these binding structures are released the tumor can usually be displaced inferiorly, where by necessity it is freed by a form of extracapsular dissection. In experienced hands this is a safe procedure and no adjuvant radiotherapy is warranted.

Extracapsular dissection

The practice of surgery in general is moving down a minimally invasive pathway and in the parotid this has found expression in extracapsular dissection (ECD) of benign parotid tumors. A minimally invasive approach challenges the concepts underpinning traditional parotidectomy and is still contested. The paradox is that as early as 1984, Donovan and Conley²⁸³ noted that 60% of benign salivary tumors came to rest on the facial nerve and when peeled from the tumor surface there was no increased risk of recurrence. The Christie series of 380 patients treated by ECD (median follow-up of 15 years) had a recurrence rate of 1.6% which matches SP but with significantly reduced morbidity.²⁸⁴

ECD should be reserved for discrete benign parotid lumps (>2 cm diameter) in the superficial portion of the parotid gland. The nature of the lump should be confirmed by FNAC for the main risk in ECD is not tumor recurrence but the inadvertent treatment of a low-grade malignant salivary gland tumor masquerading as a benign lump. This risk is greatest in small lesions as they have not had time to express their clinical nature and small lumps can be missed on FNAC.

The initial surgical approach and exposure of the parotid is similar to that for superficial parotidectomy (Fig. 36.23) with the preauricular skin flap raised to a point 1 cm peripheral to the tumor margin. A cruciate incision is made over the surface of the tumor and, by applying tension by way of artery clips attached to the parotid fascia, an areolar plane can be developed by careful blunt dissection at points around the tumor. A careful bloodless field accompanies this surgical plane at 1–2 mm peripheral to the tumor capsule. By moving it slowly from side to side, the tumor is gradually released but the procedure is not based on a nerve dissection so the nerve is not exposed unless it comes into the surgical field. Intraoperative facial nerve monitoring can be used for security. Once the tumor is removed the parotid fascia is reapproximated obliterating the defect, so minimizing the risk of Frey's syndrome, and leaving a normal parotid contour. In the majority of cases (60%) the greater auricular nerve can be preserved. It is advisable to



Fig. 36.23 ECD is a careful dissection, peripheral to the tumor capsule.

apply a pressure dressing for 48 hours post-surgery to reduce the risk of sialocele.

The data relating to ECD at four institutes (Guy's, Erlangen, Christie Hospital, Wythenshawe Hospital) were collated (Table 36.23). Outcome indices (permanent/temporary facial nerve injury, Frey's syndrome) are similar between groups suggesting that results achieved in more recent series are paralleling those with long follow-up. The accumulating data suggest that conservative procedures are safe when treating benign parotid tumors. ECD represents the limit of conservative surgery in the parotid gland.

Recurrences of pleomorphic adenoma

Recurrences of pleomorphic adenoma are nowadays uncommon following the abandonment of enucleation by the surgical community. Rupture of the tumor capsule may occur during surgery, especially if there is a significant myxoid component, or pseudopodia or satellite nodules in the "bare area". However macroscopic spillage carries only an 8% risk of recurrence, compared to 2.5% if no recognizable spillage was noted.^{285,286} Therefore the concept of spillage may not be as important as previously believed. Large tumors with a high S-phase fraction on flow cytometry are possibly more prone to recurrences.²⁸⁷

On average, recurrences occur 8 years after the initial treatment, presenting as either uninodular or multinodular lesions. MRI in combination with mandatory FNAC will confirm the recurrence of benign disease. Single nodules may be safely removed by nerve-preserving additional surgery, with a 15% risk of further recurrence at 10 years. Surgery alone is insufficient for the more common multinodular recurrences, which may be dispersed over the surgical field (Fig. 36.24). Forty-five percent of multinodular recurrences will recur within 10 years of surgical removal; adjuvant postoperative radiotherapy will

significantly reduce this to 4%.²⁸⁸ Malignant progression is very rare.

Revision surgery carries the risk of further trauma to the facial nerve which is proportionately dependent on the extent of the original surgical technique (ECD, SP, partial SP or TP). If this was either a SP or TP, the facial nerve will lie immediately inferior to the subcutaneous tissues within scar tissue. In these circumstances, the risk of permanent injury to the facial nerve is up to 60%. Even with nerve monitoring, facial nerve identification may be difficult. Skin replacement may be necessary for nodules adherent to skin. If on the other hand ECD or partial SP was performed for a tumor located in the parotid tail, the risk would be 15%.²⁸⁸

If the benign nature of the lump has been ascertained, and there is a significant risk of nerve damage, conservative management is acceptable in patients who are over 65 years old and in poor physical health.

Benign tumors of the submandibular and minor salivary glands

A submandibular gland adenoma should be removed in continuity with the gland. The situation to be avoided is an adenectomy for apparent sialoadenitis when the lesion is a malignant tumor.

The commonest intraoral site for benign salivary gland tumors is the junction of the hard and soft palate. Most present as rubbery lumps and the diagnosis can be established by FNAC or ideally a dermatological punch biopsy. The latter retrieves a small core (2–4 mm diameter) of tissue without violating the tumor margins. Those tumors situated on the junction of the soft and hard palate do not require a through-and-through dissection into the nasal cavity but can be safely dissected in a subperiosteal plane. It is advisable to cover the raw area with a dental healing plate.

Table 36.23 Outcome data for 966 tumors treated by ECD at four centers. Results of units commencing ECD parallel those with extended follow-up, suggesting similar outcome of low recurrence and reduced morbidity.

	Guy's	Erlangen	Wythenshawe	Christie
Year	1999–2007	2000–2007	1971–1999	1952–1992
Number of patients	129	279	67	491
Mean age	43.4 years (range 15–72 years)	53.4 years (range 15–90 years)	47.1 years (range 16–83 years)	
Complications:				
temporary weakness	2 (2.58%)	18 (6.45%)	4 (6%)	48 (10%)
permanent weakness	0	0	0	8 (1.6%)
Frey's syndrome	4 (5.16%)	0	1 (1.5%)	25 (5%)
Recurrence	0	0	1 (1.49%)	7 (1.8%)
Follow-up years	0.2–8 Median 4.8	0.3–7.3 Median 5.6	1–21 Median 10.4	5–32 Median 12

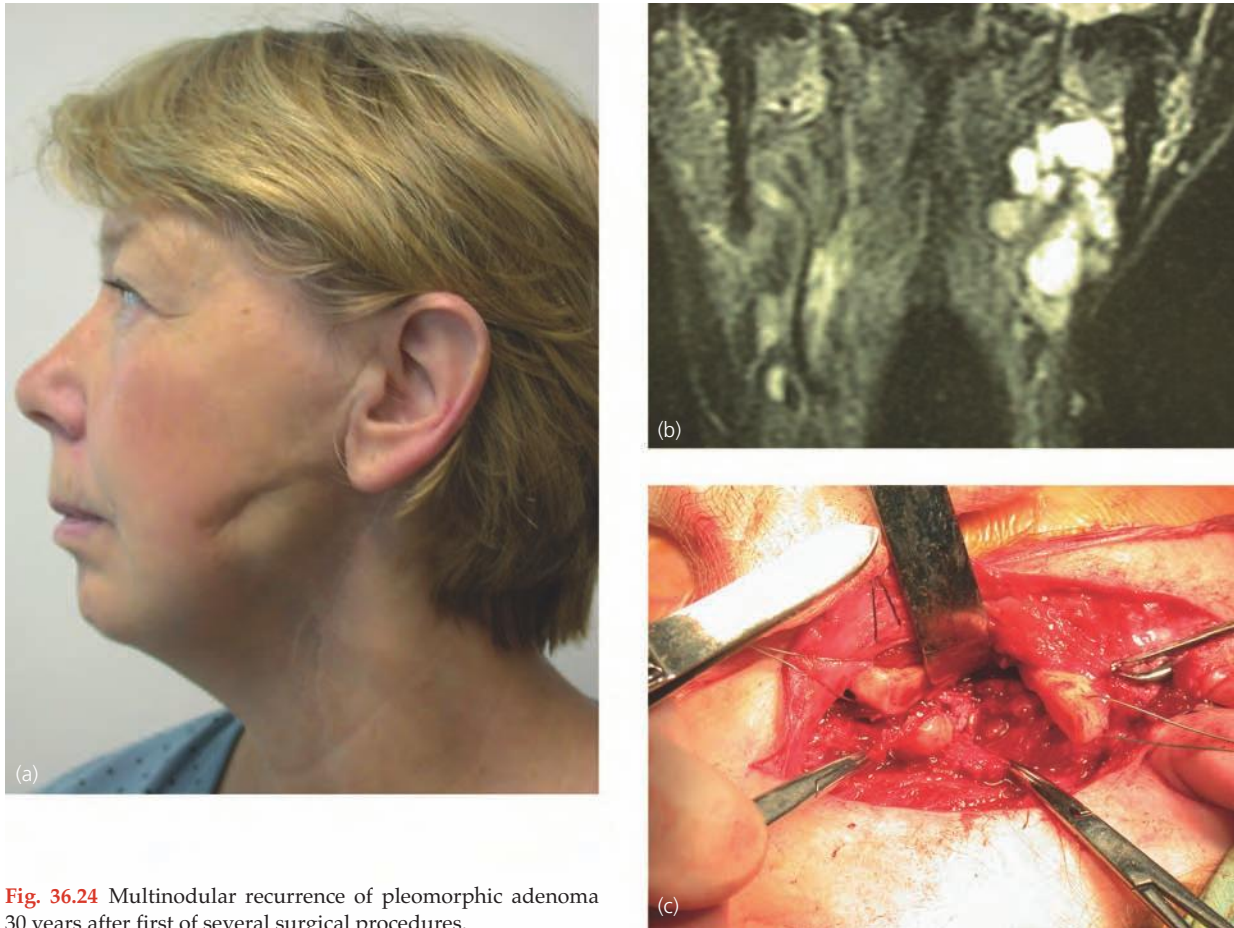


Fig. 36.24 Multinodular recurrence of pleomorphic adenoma 30 years after first of several surgical procedures.

Malignant tumors

In the last two decades there has been a gradual change in philosophy in the management of malignant salivary gland disease based mainly on the pioneering work of Spiro.^{289–291} Management traditionally relied on histological diagnosis (as proxy for grade/prognosis: see box overleaf) when deciding on treat-

ment,²⁹² whereas Spiro focused on the relationship of size rather than grade to outcome. The results were clearly better in patients with low-grade lesions; the presence of a high-grade tumor had no significant impact on survival unless the tumor was large (>4 cm in diameter). This concept revolutionized the approach to malignant salivary gland surgery and outcome data confirm (Table 36.24) that treatment

Table 36.24 Clinical factors linked to outcome on multivariate analysis (P, parotid; SM, submandibular).

	Institute	N	Sites	Prognostic factors							
				T size	Nodes	Fixity	VII nerve	Grade	Age	Male sex	
O'Brien <i>et al.</i> 1986 ²⁹³	Alabama	113	P, SM	+++				++	++		
Theriault & Fitzpatrick 1986 ²⁹⁴	Toronto	271	P only	+++	+++				+		
Spiro <i>et al.</i> 1989 ²⁹⁰	SKMCC	463	P, SM	+++				+++	++		
North <i>et al.</i> 1990 ²⁹⁵	Johns Hopkins	87	P, SM			+	++	+		+	
Frankenthaler <i>et al.</i> 1991 ²⁹⁶	MD Anderson	178	P only	++	++		++	++			
Kane <i>et al.</i> 1991 ²⁹⁷	Mayo Clinic	194	P only	+				+	+	+	
Leverstein <i>et al.</i> 1998 ²⁹⁸	Vrije Universiteit, Amsterdam	65	P only	+	+						
Calearo <i>et al.</i> 1998 ²⁹⁹	Ferrara	167	P only	++							
Vander Poorten <i>et al.</i> 1999	NCI, Amsterdam	151	P only	+	++	+			+		
Renehan <i>et al.</i> 1999 ³⁰⁰	Manchester	103	P only	++	++			++			
Kirkbride <i>et al.</i> 2000 ³⁰¹	Toronto	184	P only	+++					+++		
Luukkaa <i>et al.</i> 2006 ³⁰²	Turku	212	P, SM	+++	++				++	++	
Terhaard <i>et al.</i> 2004 ³⁰³	Utrecht	565	P, SM	+++	+++	++			++	++	

should be governed more by stage rather than histological type. The implication is that treatment has to be individualized for each patient.

Grading of tumor

The grade of a tumor is supposed to reflect its inherent biological nature (aggressive, intermediate or indolent). The term has been used loosely with more than one origin. Salivary carcinomas are classified into histological types or families (adenoid cystic carcinoma, mucoepidermoid carcinoma, etc.). The majority (but not all) of tumors in the family have a similar biological nature and certain families are known to be high-grade or biologically aggressive neoplasms (anaplastic carcinoma, carcinoma ex-pleomorphic adenoma, etc.). Others are recognized as low-grade tumors (acinic cell, low-grade adenoid carcinoma, etc.).

In addition it is possible to use histological features to identify a subgroup of tumors that signify a more aggressive behavior. This is the case for mucoepidermoid carcinoma which can be divided by histological features into high, middle, and low grade.

The last method of determining the biological nature of the tumor and the most reliable is assessment of the clinical features of aggression, such as rapid change in size, infiltration, metastasis. These are currently incorporated into the present staging system of salivary cancer. The latter has taken over from histological type and histological grading as the most important prognostic factor determining treatment.

Malignant parotid tumors

The object of treatment is to excise the tumor with an adequate rim of normal tissue but this may not be possible because of the complex anatomy of the parotid area and emphasizes the practical nature of the 4 cm rule, as adequate surgical clearance of tumor is not possible with large masses.³⁰⁴ It is for this reason that adjuvant radiotherapy plays an important part in the management of salivary gland cancer.³⁰⁵ Wherever possible a functioning facial nerve should be retained except if completely immersed in tumor. The traditional approach to a malignant parotid tumor is a total parotidectomy. With experience this dogma can be softened and small (stage I) well defined tumors, particularly in the tail of the parotid, can be excised with an adequate normal parotid tissue to make a total or even a superficial parotidectomy unnecessary.^{306–308} In contrast if a facial nerve palsy is already present then a radical parotidectomy is a logical option. If the facial nerve has to be resected, ideally it should be reconstructed at the same operation.

Submandibular gland

The literature demonstrates that submandibular gland cancers carry a 20% worse prognosis than malignant tumors in the parotid or mouth.²⁷⁹ Initially this was attributed to a higher incidence of adenoid cystic carcinomas in the submandibular gland. However Spiro showed this not to be the case. An appropriate oncological procedure as the primary

procedure restores the survival rate to that achieved in both the mouth and the parotid.

Minor salivary gland tumors

Minor salivary gland cancers within the oral cavity are managed in the same way as squamous cell carcinomas in the mouth.

Radiotherapy

A single center retrospective series³⁰⁹ and a matched-pair analysis³¹⁰ demonstrated improvement in local control when a combined approach was compared with surgery alone. Analysis of results of the Christie Hospital^{311–313} also showed significant reduced local regional recurrence (16% vs 62%) and significantly improved 5-year survival (63% vs 73%) in tumors >4 cm with adjuvant radiotherapy. It is generally acknowledged that adjuvant radiotherapy reduces local recurrence but does not have a significant impact on overall survival due to distant metastasis. There is emerging evidence, however, that in biologically aggressive tumors survival may be improved by adjuvant radiotherapy.

Neutron beam therapy was used in the 1980s and 1990s in the treatment of head and neck cancer but was subsequently abandoned because of severe tissue reaction and complications following treatment. Radiobiological effectiveness of neutron beam therapy is superior to that of traditional photon therapy for salivary gland tumors.^{314,315} In view of the history of unacceptable normal tissue morbidity following neutron beam therapy the number of operational neutron facilities is limited. Consequently photon therapy is likely to remain the principal form of adjuvant therapy to support surgery.

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Chapter 37

Outcomes of Management of Oral Pathologic Lesions

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This chapter will aim to cover the general outcomes of oral pathologic lesions with particular emphasis placed on oncology. The main outcomes are survival, complications, quality of life, and function. These will be explored in detail to give the reader an understanding of how these impact on the maxillofacial patient.

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the disease, the consequence of surgical intervention, and the patient's perception of this.

Outcomes are fundamental to all aspects of oral and maxillofacial surgery ranging from dentoalveolar, orthognathic, trauma, and oncology. This chapter will focus on outcomes following complex benign and malignant lesions. Although there is a vast range of pathologic conditions, they share a common outcome. The main outcomes discussed are survival, complications, patient-derived (health-related quality of life), and function (Table 37.1). However, there is a notable difference between complex benign and malignant lesions, that of survival. Although unexpected tragic death can occur following surgery for benign disease, in the cancer situation the poignancy of recurrence and death from disease is reality. In oncology survival is of paramount consideration for patients.^{1,2} There is a better outcome with early-stage disease (stage 1 and 2) but this chapter does not intend to address issues related to earlier detection and referral of pathologic lesions.

There are various ways of measuring outcome, both objective (clinician rated) and subjective (patient derived) (Table 37.1). The objectively scored evaluations include measurements from radiographs or study models, or the presence or absence of a specific outcome such as a gastrostomy tube. Specific complication rates for certain operations might also be pertinent and vary depending on the surgery, for example facial nerve weakness following superficial parotidectomy. Indirect indicators may be related to outcome such as length of hospital stay (LOS). The problem with LOS is that, although it might reflect surgical complications, it is influenced by many other factors such as age, comorbidity, family support, and social deprivation. Thus it is essential to incorporate case-mix factors for certain outcomes such as perioperative mortality. The patient-derived assessments (subjective) are usually measured by questionnaires. These can provide quantitative (numbered) data that are easier to handle than qualitative (open interview) approaches. Although difficult to measure, the patient's perspective must be borne in mind, as this

Introduction

The aim of this chapter is to discuss outcomes of both complex benign and malignant lesions of the oral cavity. Pathologic lesions within the oral cavity and adjacent structures can arise from a variety of tissues such as mucosa, salivary glands, bone, and tooth tissue. The outcome relates to the natural history of

Table 37.1 Main outcomes.

Outcome	Examples
Survival	Overall, disease specific, recurrence, locoregional (i.e. around a local region) control rates
Complications	Relapse, deep vein thrombosis (DVT), wound infection, plate removal, free flap success, fistula, etc.
Patient derived	Health-related quality of life, function specific, satisfaction
Function	Paresthesia, movement of tongue, mouth-opening limitation, pain, speech difficulty, swallowing difficulty
Indirect	Length of hospital stay, readmission rates

Table 37.2 Value of patient-derived outcome data.

Person who might find patient-derived outcome of value	Reason for the patient-derived data
Clinician	To demonstrate that the treatment has been effective
Hospital	Successful treatment for groups of patients
Financers for treatment/insurance companies	To include the patient's perspective in the evaluation of the most cost-effective treatment
Government bodies/health improvement agencies	Compare outcomes between different surgeons, departments, and hospitals
Employer	Successful treatment reduces the time taken to recover and be absent from work
Researcher	Statistical comparison of different treatments
Statistician	95% confidence limits, i.e. to show treatment works intentionally and not by chance
Patient/carer	Informed decision making, improvement of symptoms

can be crucial to “success” or “failure” from the patient’s viewpoint. It is recognized that there are patients who have an excellent outcome following surgery yet have a lot of concerns and are far less happy with the result than other patients with a similar objective outcome. Conversely, some patients who have a poor surgical outcome report relatively little dysfunction and high degree of satisfaction. In part this reflects different personalities and expectations.

To fully address the issue of outcome it is worthwhile combining both elements of objective and subjective measures. This is well demonstrated in an early paper by Brown *et al.*³ where the benefit of using a pharyngeal flap in combination with a radial forearm free flap to reconstruct the soft palate after tumor ablation was evaluated. The outcomes include a validated head and neck specific questionnaire (University of Washington Quality of Life Scale), intelligibility

(clarity of speech scoring by both speech therapists and lay listeners), and videofluoroscopy. These multiple modalities combined give a clear indication of the potential benefit of using a pharyngeal flap when the resection involves more than half of the soft palate. As shown later in this chapter a similar detailed approach was used by Devine *et al.*⁴ to compare outcomes following two different surgical oral access procedures: lip split mandibulotomy or visor/dropdown.

Outcome data are useful to many different people (Table 37.2). They help to underpin clinical audits, local protocols, national guidelines, and clinical research. A challenge is that outcome parameters can vary markedly between units and countries, and this makes comparison problematic. It is important for groups and “associations” to agree a workable dataset with common outcome parameters that are reproducible. Ideally the measures should be simple and the process not arduous. There are several examples of nationally agreed datasets such as DAHNO (data for head and neck oncologists) in the UK.

Survival

Relatively few institutions have reported survival data for their patients with oral cancer (squamous cell carcinoma) and this probably reflects the diligence required in collecting accurate data. Survival can be considered as crude (overall) or disease specific (i.e. related specifically to squamous cell carcinoma for example). Some older papers predate the widespread introduction of microvascular reconstructive techniques.^{5,6} Reports exist from various other countries such as Australia, Denmark, Taiwan, USA, Norway, Japan, South Korea, Germany, and the UK.⁷

Survival can be related to prognostic factors such as age, stage, and histopathological criteria.^{8–12} Other factors may also play a role, for example blood transfusion and free tissue transfer¹³ for patients with mandibular invasion¹⁴ and adjuvant radiotherapy for patients at an intermediate risk of recurrence.¹⁵

Accurate survival data are critical for many reasons. These data aid treatment protocols, tumor board discussions, and individual patient and carer decision-making. Data from the authors’ unit (University Hospital Aintree, Liverpool) show that of 541 consecutive patients with oral squamous cell carcinoma seen between 1992 and 2002 only 10% received primary radiotherapy.⁷ In the unit the favored curative treatment is radical primary surgery. Those patients who had primary radiotherapy tended to be older, with additional comorbidity and with more advanced disease. This was reflected in an overall poorer survival at 5 years, 23% (SE 7%). In the 489 patients who had primary curative surgery, 40% received adjuvant radiotherapy. The overall survival (OS) was 56% (SE 2%) and the disease-specific survival (DSS) was 74% (SE 2%). Five year DSS for pT1 tumors was 96%, pT2

was 82%, pT3 was 78%, and pT4 was 57%. There was a local recurrence rate of 10% and the locoregional recurrence rate was 21%. The second primary rate was 7%. Survival figures had improved over the 10-year period from 63% DSS for the first 4 years of the study (1992–1995) to 81% for the last 3 years (2000–2002). In stepwise Cox regression the two predictors selected for DSS were pN status and margins (both $p < 0.001$). Age–sex mortality rates for the north-west of England indicate that 15.0% of the 489 primary surgery patients might have been expected to die within 5 years if they were typical of the general population and the observed difference between all causes and oral cancer specific survival was 18.3%. These data emphasize the value of DSS as an indicator of successful treatment in a cohort that tends to be elderly, from social deprived backgrounds, with life styles and comorbidity that influence overall survival.

Tumor recurrence rates

Both benign and malignant pathologic conditions have the potential to recur. Although uncommon and arising over a long time period, it is important that recurrence rates following surgery for benign disease are recorded as accurately as possible. For example, recurrence is seen with parotid gland neoplasia (pleomorphic adenomas), odontogenic cysts (keratocystic tumors), and odontogenic tumors (ameloblastomas). Likewise local recurrence rates following locally aggressive lesions such as odontogenic tumors (ameloblastoma) are a significant outcome.

With malignant disease recurrence rate usually reflects ongoing disease in terms of local, regional, and metastatic spread.⁷ Regional (neck) recurrence is one of the commonest sites and this is linked with the rate of clinically and radiologically undetected nodes at presentation tends to support the use of selective neck dissection even in relatively early oral cancer. There is some uncertainty, about which patients benefit from adjuvant radiotherapy following primary surgery.¹⁵ As discussed later in the chapter, there are probably more opportunities for salvage in patients treated by surgery alone. Second primary tumors may also arise from an area of field change (adjacent dysplastic tissue). The issue of second primary tumors rates has become of concern as a consequence of the success of primary treatment and increase in longevity in this patient group.

Complications

Complication rates are another key outcome parameter following surgery. These can be considered as immediate, early or late, and are also general or site specific. There are many different complications following surgery to the orofacial region. This is illustrated following parotidectomy where facial nerve

impairment, greater auricular sensory deficit, gustatory sweating (i.e. sweating on salivation), and pain are all pertinent outcome parameters. These can therefore impact on the patients' perception of success.¹⁶ It is usually easier to record complications immediately postoperatively, such as facial nerve weakness in the case of a parotidectomy, when patients are on the ward or at their first outpatient review. With longer-term outcomes patients tend to get lost to follow-up or are missed. It is difficult to get long-term data as assessment at a specific time point is often forgotten. However facial nerve weakness can take many months to improve so long-term outcome data are essential. This chapter will not attempt to list all the possible complications that might make a suitable indicator of outcome for the various different pathologies and interventions; a relatively few general examples (in alphabetical order) are considered for illustrative purposes.

Adult respiratory distress syndrome (ARDS)

This is defined as *"the pulmonary manifestation of a widespread abnormality of cellular metabolic function"* and was first described by Ashbaugh *et al.* in 1967.¹⁷ ARDS is associated with mortality following major surgical procedures. It has been linked with large-volume blood transfused in 48 hours and a return to the operating theater for early complications. Change in perioperative practice in relation to fluid management and mechanical ventilation seems to have all but eliminated this event.^{18,19}

Deep vein thrombosis (DVT)

This is a complication usually related to prolonged operations such as orthognathic and oncology surgery. Blackburn *et al.* carried out an audit of DVTs following osteotomies in 129 patients over 4 years.²⁰ Two cases had DVT confirmed by venography with neither having a pulmonary embolus. Clagett *et al.* showed a 25% incidence of DVT in postoperative patients who had no prophylactic measures.²¹ This highlights the value of prevention by using local measures such as antithrombotic stockings and low molecular weight heparin.

Donor site morbidity

The radial forearm free flap is the commonest source of free tissue transfer for reconstruction of the maxillofacial region. This reflects its relatively easy harvesting and its versatility. It also has a long vascular pedicle with large-caliber vessels if raised with the cephalic vein, and minimal donor site morbidity. It is particularly useful in the oral cavity. There are various complications that have been reported, such as partial loss of the donor-site skin graft, exposure of tendons, fracture of the radius with composite flaps,

and reduction in superficial radial nerve sensation.²² Fracture of the radius is associated with a poor functional outcome. Care is required during the harvesting of bone, and plating and immobilization have been advocated to help prevent this complication.

Free flap loss and flap salvage

Free tissue transfer has an established role in the reconstruction of patients following tumor ablation and tissue loss. The success of the free flap is critical from a functional perspective and for health-related quality of life. A non-viable flap has to be removed and the defect reconstructed by another free flap or alternative method. This leads to additional morbidity and prolonged hospital stay, and tends to delay the timing of adjuvant radiotherapy. This in turn can impact on cure rates. Flap success rates are decreased in patients who have had previous neck surgery or radiotherapy, those with comorbidity, and patients who have composite reconstruction. Site is also a factor, with the maxillary defect being one of the most difficult given its composite nature, the length of pedicle required, and the quality of the feeding artery and draining vein. Early identification of a compromised flap increases the likelihood of successful salvage.²³ Grant *et al.*²⁴ have reported that the acute physiology and chronic health evaluation (APACHE II) score is a good predictor of early postoperative surgical complications but how useful this is in clinical practice is unclear.

Length of stay (LOS)

This can be used as a surrogate indicator of complications. It is, however, influenced by several other factors such as age, comorbidity, and social circumstances. Although LOS is a relatively non-specific outcome it is often used because there are cost implications to hospitals with regards to time spent in a hospital bed and it is an easy outcome to measure. Rogers *et al.* examined the relationship between LOS and health-related quality of life and found increased LOS to be associated with worse health-related quality of life.²⁵ This was confounded by age, which influences the LOS more than health-related quality of life.

Plate infection and removal

For mandibular defects, reconstruction usually involves titanium miniplates or reconstruction plates. If infection occurs the offending plate(s) must be removed. Shaw *et al.* compared complication rates of miniplates versus the larger reconstruction plates in the fixation of vascularized bone grafts into segmental mandibular defects involving 143 vascularized composite free flaps placed over an 8-year period.²⁶ Forty-nine percent had miniplate placement and 51% had reconstruction plates; 27% of miniplates and 30% of reconstruction plates required removal for infec-

tion. There was no statistically significant difference between the plate types.

Readmission

Readmission rates are sometimes used as an outcome parameter. Like LOS they are non-specific as readmission might not necessarily reflect a complication or indicate the need for further surgery or recurrence. Readmission could be for pain control, nutritional support or for other non-surgical reasons.

Wound infection rates

Wound infections, although not often of major significance in terms of mortality, cause delayed and less satisfactory healing. There are many pathogens which can be involved, such as hemolytic streptococci, but it is methicillin-resistant *Staphylococcus aureus* (MRSA) that is currently under the most intense scrutiny. Rogers *et al.* reported an MRSA rate of 1% on a maxillofacial ward.²⁷ The vast majority occurred in oncology patients where the rate was 14% and the significant risk factors were found to be the stage of cancer and whether free flap surgery was involved. Another notable pathogen is *Clostridium difficile* – a toxin-producing, Gram-positive anaerobe that has been implicated as the causative agent of pseudomembranous colitis. This is an acute inflammatory bowel disease that generally occurs in association with antimicrobial therapy.²⁸ This pathogen can have grave implications for patients. Further issues arise regarding cross-infection control, such as hand washing, screening and segregation of patients, inappropriate use of antibiotics, and wound dressings. These all need further evaluation specifically related to a maxillofacial setting and how they affect wound infection rates.

Patient-reported outcomes

It is important to use patient-reported outcomes (PRO) as there can be marked differences in perspectives between doctors and patients. Doctors tend to overestimate the objective symptoms and underestimate more subjective problems.²⁹ Concerns of patients can easily be missed.³⁰ The various ways of measuring patient-derived outcomes are given in Table 37.3. The main difficulties in asking patients to fill in questionnaires are logistical such as lack of resources, time, and manpower as well as inexperience with questionnaire use.³¹ Computers offer a practical solution, using touch screen technology³² to help collect PRO in terms of health-related quality of life outcomes.

Table 37.4 gives examples of the different groups and types of questionnaires available. There is no gold standard questionnaire and each has unique features, strengths, and limitations.^{33,34} Questionnaires

Table 37.3 Methods of measuring patient-derived outcomes.

Approach	Indication
Simple questionnaire	Takes less than 10 minutes for patient self-completion Useful for postal survey with good response rates Ideal for telephone survey
Semi-structured interview	Patients are assisted to complete a battery of questionnaires The questionnaires can be more complicated in structure and thus provide more information Discussion of issues adding weight or importance to the items to develop a patient-generated index (PGI) or trade-off to build a utility value
Open interview	Loose structure to the interview allowing the patient to report many different issues and the opportunity to explore these in more depth Useful for item generation as a first step in questionnaire development Used in conditions where the scope of the issues is very wide, such as palliative care

Table 37.4 Different groups of questionnaires and examples of each. Each abbreviation mentioned is the name of a questionnaire tailored to measure a specific aspect, e.g. cancer or function, etc. There is a plethora of these tools available and those shown here are merely a small sample. For further information please see references 33, 34, 67, 68.

Group	Questionnaire name
Performance	Karnofsky, ECOG
Global/generic	SF-36, HAD, CESD, EQ-5D
General cancer	EORTC-C30, FACT-G
Head and neck cancer specific	EORTC H&N, FACT HN, UW-QOL
Function specific	Appearance (Derriford) Oral rehabilitation (LORQ) Saliva (XEQoLS) Shoulder (NDI, SDI) Speech (VHI, V-RQOL) Swallowing (SWAL-QOL, MDADI)
Others	Body image, coping, distress, emotion, family, fatigue, information, pain, personality, satisfaction, self-esteem, social support, spirituality

must be validated. A commonly used broad head and neck cancer questionnaire is the University of Washington quality of life tool (UW-QOL). The UW-QOL was first published in 1993³⁵ and is currently in its fourth version.³⁶ There have been recent additional questionnaires that have focused on specific issues in more depth, such as swallowing, speech, and dry mouth. There is now a substantial body of published material on the subjective outcomes following oral and oropharyngeal cancer.³⁷ Table 37.5 shows the key issues. Outcomes are associated with patient characteristics such as age, the tumor itself, the patient's

Table 37.5 The key subjective outcomes following head and neck cancer. For further information please see references 33, 34, 67, 68.

Patient-derived outcome
Coping
Dental status
Disfigurement
Emotion
Fear of recurrence
Financial
Function
Intimacy
Oral rehabilitation
Personality
Socio-demographic
Speech
Swallowing
Shoulder
Xerostomia

lifestyle, and factors relating to the surgery and/or radiotherapy.³⁸

It is better to choose a validated questionnaire for audit or a research proposal where possible, as study-specific questions lack validity and comparison with other studies. Questionnaires exist to test nearly every aspect of oral and maxillofacial surgery, including esthetic surgery, cleft lip and palate, craniofacial surgery, dentoalveolar surgery/third molars, distraction osteogenesis, facial pain, oncology, oral medicine and oral mucosal disorders, orthognathic surgery, pre-prosthetic surgery and dental implants, reconstructive surgery, salivary gland disease, skull base surgery, temporomandibular joint, and trauma.

Function

The functional outcome of disease is a key factor as a consequence of the treatment. This affects patients' acceptance and quality of life.³⁹ The relationship between function and health-related quality of life was explored by Rogers *et al.* by using an 11 point examination.⁴⁰ Factors assessed included lip competence, speech, drooling, lip and tongue sensation, and shoulder movement. Patients who had large tumors, free tissue transfer or adjuvant radiotherapy (i.e. radiotherapy post surgery) were found to have a worse level of function. This was associated with a lower health-related quality of life. Clinical examination combined with validated questionnaires provides a useful predictor of outcome. Detailed information such as this can help patients to reach a decision in treatment choice and is a useful adjunct to informed consent.⁴¹

Information and clinical outcomes research has given valuable insight into various aspects of oncology and has helped to shape clinical practice. Both objective and patient-derived data can be used. The following are examples to illustrate the issue.

Access

Good access to facilitate tumor ablation is an essential prerequisite for a safe resection. There can be a choice between lip-split with a mandibulotomy versus a mandibular lingual releasing technique (Fig. 37.1). In a detailed study combining objective and subjective measures of outcome between these two methods, Devine *et al.*⁴ showed that there was no obvious difference in objective function or in appearance. However on subjective data the lip split mandibulotomy group reported significantly better speech, swallowing, and chewing.

Bone flaps

The choice of the most suitable reconstruction following extensive jaw resection is often between a deep circumflex iliac artery (DCIA) flap or a fibula flap. Both objective and subjective evaluation of outcome would suggest that both flaps are well tolerated with generally acceptable morbidity.⁴² However when a significant complication occurs it tends to have a very detrimental effect on health-related quality of life in both groups. The choice of either flap is based on the one that offers the most optimal reconstruction.

Mandibular resection

The issue of segmental resection or rim resection of a mandible in tumor removal has been informed by subjective outcome data.⁴³ Preoperatively patients undergoing segmental resection reported significantly more pain, chewing problems, and a lower composite UW-QOL score. Postoperatively the segment group tended to score worse at all time points, particularly in appearance, swallowing, recreation, and chewing. However, the difference between rim and segment was only seen in smaller resections without adjuvant radiotherapy. Little difference was seen between rim or segment for tumors <4 cm with radiotherapy and also between rim and segments for tumors >4 cm. Young *et al.* examined the health-related quality of life for patients undergoing



Fig. 37.1 Lip split mandibulotomy: free flap being inset following tumor ablation.

mandibular resection with immediate insertion of a reconstruction plate and subsequent staged reconstruction.⁴⁴ The staged reconstruction involved bone grafting, removal of the reconstruction plate, vestibuloplasty, implant insertion, and construction of an implant-supported prosthesis. Appearance was a key concern with segmental resections and might affect females more than males and could explain their decreased health-related quality of life.

Maxillectomy

The maxillectomy defect can be obturated using a prosthesis or reconstructed with free tissue transfer. Over time, following patient adaptation and careful clinical support, there might not be that much difference between the two approaches from the patient's perspective.⁴⁵ It is probable that primary free tissue reconstruction is associated with better health-related quality of life outcomes.⁴⁶

Neck dissection

In the management of oral and oropharyngeal cancer the selective neck dissection has an established role. This is based on the possibility of occult (i.e. hidden) lymph node metastasis not evident clinically or obvious on pretreatment imaging.⁹ From the patient's perspective a selective neck dissection causes relatively little morbidity compared to other health-related quality of life aspects⁴⁷ and is not as important as other issues such as speech, chewing or swallowing.⁴⁸ A functional neck dissection can be associated with more clinician-rated deficits than with problems reported by the patient.^{49,50} Further outcome studies are required on the role of a "wait and watch" or sentinel node biopsy policy. Sentinel node biopsy involves a dye and radioactive tracer being injected into the tumor field and traced. The first group of lymph nodes reached (sentinel nodes) are then removed, minimizing the need for a selective neck dissection.

Oral rehabilitation

Oral rehabilitation has an important role in promoting quality of life following reconstructive surgery.^{51,52} A relatively small proportion of patients require implant-retained prosthesis.⁵³ The Liverpool Oral Rehabilitation Questionnaire (LORQ) helps give more precision to the subjective evaluation of outcome.^{54,55} The factors associated with the success of implants have been assessed and the necessity of hyperbaric oxygen in an irradiated field has been brought into question.⁵²

Percutaneous endoscopic gastroscopy (PEG)

Nutritional support is an important consideration for patients undergoing surgery who already have pre-

existing weight loss and might have problems with swallowing. The benefit of percutaneous endoscopic gastroscopy (PEG) has to be weighed up against the potential morbidity and mortality. Insertion can cause peritonitis and there is the potential risk of PEG site seeding (i.e. seeding tumor cells from the primary site into the gastrostomy site during placement). Patients with long-term PEGs have been found to have a significantly worse health-related quality of life.⁵⁶ This is because those patients who require a PEG usually have advanced disease requiring radical treatment (combined surgery and adjuvant radiotherapy or chemoradiotherapy). The main problems encountered by patients, however, are not related to tube blockage, leakage or discomfort, but are due to broader aspects such as interference with family life, intimacy, and hobbies.

Postoperative radiotherapy

Choosing which patients need postoperative radiotherapy is a topic of much debate. Brown *et al.* examined 462 patients treated by primary surgery, with or without radiotherapy;¹⁵ 29% were at low risk of disease recurrence, 29% were at high risk, and 42% were at intermediate risk. For those at intermediate risk the 5-year survival was 54% for those receiving radiotherapy vs 71% for those treated with primary surgery alone. There was found to be a higher rate of locoregional recurrence in the radiotherapy group and a lower salvage rate compared to the surgery alone group. There are therefore potential disadvantages associated with the use of postoperative radiotherapy in the intermediate risk group. A randomized control trial is required to adequately evaluate the benefit of radiotherapy in the intermediate risk group. Patients needing combined treatment have a significantly poorer health-related quality of life outcome⁵⁷ and the main issues relate to the problems associated with xerostomia, swallowing, and trismus. Trismus can be commonly overlooked but can have a profound affect on a patient's chewing, diet, and overall quality of life.⁵⁸ It is more effectively evaluated as an outcome by combining measurement (35 mm cut off) and a single item question, e.g. "How much has your mouth opening decreased since treatment". Intensity modulated radiotherapy (IMRT) reduces the degree of salivary dysfunction and it is hoped it will give a better outcome from the patient's perspective in the future.

Soft palate resection

In some centers radiotherapy is the first choice of treatment for tumors at this site. Brown *et al.* showed that regardless of the size of tumor, primary surgery has a favourable outcome.⁵⁹ Speech, chewing, and swallowing were similar regardless of resection size. Speech and swallowing outcomes have been further reported by Zuydam *et al.*^{60,61} As described earlier in

the chapter if over half the soft palate has been resected, a superiorly based pharyngeal flap is recommended.³ Both objective and subjective outcome data would concur. It is believed there may be shrinkage of a free flap, especially postradiotherapy, leading to velopharyngeal incompetence, which a superiorly based pharyngeal flap helps to prevent.

Tumor resection

The surgeon performing tumor ablation aims to get an adequate margin of clearance to prevent recurrence. Where possible laser excision or primary closure (i.e. closure at the time of surgery) gives superior functional and health-related quality of life outcomes than free tissue reconstruction.⁶² This is particularly notable for the speech and swallowing outcomes for oropharyngeal and posterior one third of tongue tumors.^{60,61} In recognition of this the proportion of patients having free flaps has reduced⁶³ without any notable increase in local recurrence rates or poorer survival.

Conclusion

The evaluation of outcomes following treatment for complex benign and malignant lesions has a crucial role in clinical practice. The main outcomes are survival, complications, patient-derived quality of life, and function. Patient-derived outcomes give the patients and clinical team much better information on expected outcomes.^{64,65} The assessment is made much stronger when objective (clinician-rated) and subjective (patient-derived) data are used in combination. These data can help to shape clinical practice in each unit, aid national protocols, and inform the clinical research agenda.

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Part 6: Trauma

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Chapter 38

Assessment of the Injured Patient

Brian Bast

This chapter gives an overview based upon on the guidelines for the initial evaluation of the trauma patient by the American College of Surgeons and detailed in the Advanced Trauma Life Support courses which in recent decades have been given worldwide and attended by many oral and maxillofacial surgeons.

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Trauma is a global problem and continues to be a leading cause of death and disability in both developed and developing countries. It is the leading cause of death in persons aged 1 through 44 years in most developed countries. Trauma caused more than 300 million patients to seek hospital care worldwide in 1990. It is projected to advance to the second leading cause of death in all age groups worldwide by 2030. Motor vehicle accidents are projected to rise to the fifth leading cause of death worldwide by 2030. Violence is also projected to increase in prevalence and by 2030 will be the sixteenth leading cause of death worldwide.¹ Many trauma patients brought to the hospital will have facial injuries requiring the care of an oral and maxillofacial surgeon.²

Oral and maxillofacial surgeons will always be involved in the evaluation and management of patients who have suffered facial trauma. Facial trauma encompasses a broad spectrum of disease. We may be called to treat the minor lip laceration sustained during a sports injury and in contrast we may be called to the trauma center to evaluate a patient with multiple facial fractures sustained during a roll-over motor vehicle accident. In both of these cases the patient may have other injuries that require diagnosis

and treatment. It is critical that the oral and maxillofacial surgeon has an understanding of the initial evaluation of the trauma patient and can ensure that the appropriate work-up was completed and more serious injuries were either diagnosed or ruled out.

This chapter will review the guidelines for the initial evaluation of the trauma patient established by the American College of Surgeons and detailed in the Advanced Trauma Life Support (ATLS) courses.

Development of the ATLS concept of trauma care

An orthopedic surgeon involved in a plane crash in the 1970s recognized the inadequate trauma care provided by a rural hospital in the USA. A group of local surgeons, emergency room doctors, and nurses, working with the Lincoln Medical Foundation and the University of Nebraska, took on the task of developing a course to ensure optimal care of the injured patient. The courses were initially designed for the physician who did not routinely evaluate and treat trauma patients. The idea was to provide clinicians with a skill set that could be used in more effective trauma evaluation and resuscitation. The first ATLS course was given in 1978 and 1 year later was adopted by the American College of Surgeons as an educational program. The ATLS courses are now in their third decade and are taught in over 50 countries worldwide. The principles have been taught to over 1 million physicians since 1990.³

The concepts adopted by the ATLS course were simple and represented a significant change from the traditional medical work-up of a patient. There was less emphasis placed upon an exhaustive history and detailed physical exam. The ATLS programs were built around three core concepts which represented a dramatic change in traditional “medical” thinking. The first concept defines the ATLS approach. Treat the greatest threat to life first. The loss of an airway kills faster than the loss of intravascular volume which kills faster than an acute intracranial bleed. This prin-

ciple is simplified as the “ABCDE” approach to the trauma evaluation. The second principle is that an indicated treatment should not wait for a definitive diagnosis. And third, an extensive history is not a critical component of the initial evaluation of the injured patient.⁴

Trauma centers and trauma systems

In the USA, and in many regions around the world, trauma care is provided by trauma systems with the goal of transporting injured patients rapidly to local trauma centers for care. Care systems ensure a coordinated effort from hospital emergency medical services and trauma hospitals. They ensure an efficient triage and transport to the trauma center for acute care and rehabilitation. Many studies have reported dramatically improved outcomes when injured patients are treated at trauma centers. Multiple studies suggest that when the most severely injured patients are treated at trauma centers there is up to a 50% decrease in preventable deaths.⁵

In 1976 the American College of Surgeons Committee on Trauma developed criteria for categorizing hospitals according to the level of trauma care available. Level one trauma centers provide comprehensive trauma care, serve as a regional resource, and provide leadership in education, research, and system planning. A level one center is required to have immediate availability of trauma surgeons, anesthesiologists, physician specialists, nurses, and resuscitation equipment. Level one classification requires that a center treats at least 240 major traumas per year. Level two centers also provide comprehensive trauma care either as a supplement to a level one center in a large urban area or as a lead hospital in a less populated region. Level two centers must meet essentially the same criteria as a level one center but volume standards are not required and centers are not expected to provide leadership in teaching and research. Level three trauma centers provide prompt assessment, resuscitation, emergency surgery, and stabilization with transfer to a level one or two center as indicated. Level three centers typically serve communities that do not have immediate access to a level one or two facility. Level four centers provide advanced trauma life support prior to transfer; they are in remote areas in which no higher level of care is available.

Level one and two trauma centers carry the highest clinical demands. The required clinical capabilities include general surgery, anesthesiology, emergency medicine, neurosurgery, orthopedic surgery, oral and maxillofacial surgery, plastic surgery, hand surgery, ophthalmology, obstetrics and gynecology, thoracic surgery, critical care medicine, cardiac surgery, and microvascular surgery.⁶

Initial treatment of the trauma patient

Triage

Deaths from injury are classically described as occurring in a trimodal distribution. The first peak is within seconds to minutes of the injury and is due to severe brain or spinal cord trauma, heart or major vessel rupture. These types of injuries are not survivable. The second peak occurs from minutes to hours after injury. Deaths during this period are due to progressive intracranial bleeding (subdural/epidural hematoma), organ system damage (pneumothorax, spleen or liver laceration), pelvic fractures, or other injuries associated with significant blood loss. The third peak occurs days to weeks after the injury and is due to sepsis or multiple organ system failure. There is some debate about whether deaths from injury continue to follow the classic trimodal distribution.⁷ What remains clear is that the vast majority of preventable deaths require rapid assessment and intervention. Multiple events need to occur efficiently in the “golden hour” to enable assessment and resuscitation.

Trauma care begins outside of the hospital. Emergency medical teams are often the first to evaluate injured patients. They are trained to rapidly triage (from French *trier* = sort) the patients, so priority is given to those patients and injuries which are most serious or require urgent treatment, and to determine if the patient’s injuries warrant transport to a trauma center. Triage criteria include vital signs and level of consciousness, types of injury, and mechanism of injury. During the initial evaluation if the injured patient shows a decreased level of consciousness, high or low respiratory rate, or low systolic blood pressure they are transported to a trauma center. If the patient has obvious severe injuries (flail chest, long bone fractures, penetrating injuries, skull fractures, paralysis, pelvic fractures, or burns) they are transported to the trauma center. If the mechanism of injury would suggest severe patient injury (ejection from auto, death of co-passenger, high speed crash, prolonged extrication time, falls greater than 6m, auto pedestrian injury, or motorcycle crash) the patient is taken to the trauma center. If the injured patient has underlying factors that could complicate the management of even minor injuries (age less than 5 or greater than 55, pregnancy, immunosuppression, cardiac disease, pulmonary disease, diabetes, cirrhosis, morbid obesity, or coagulopathy) the patient is taken to a trauma center. The overriding rule is “when in doubt take the patient to the trauma center”.⁸

A number of triage tools have been developed to aid in the rapid evaluation and determination if an injured patient should be brought to a trauma center. Although sensitive, specific, and predictive of out-

come in practice, these tools may be cumbersome and are rarely used. Two of these tools have become a standard component of many triage algorithms and have been validated in the literature. The Revised Trauma Score is a physiologic scoring system which uses the first set of data obtained from a trauma patient. It consists of Glasgow Coma Scale, systolic blood pressure, and respiratory rate.⁹ The Pediatric Trauma Score is a six-component scale that includes weight, airway, systolic blood pressure, central nervous system, open wound, and skeletal trauma.¹⁰ Both of these tools provide an absolute number that can aid in the triage process.

Emergency department preparation for the arrival of a trauma patient is a vital part of the triage process. A large resuscitation area should be designated for the patient's arrival. Airway equipment, warmed intravenous crystalloid solutions, monitors, and medical personnel should be immediately available. Radiology and laboratory evaluations should also be rapidly available.

Primary survey and resuscitation

Once the trauma patient is transported to the hospital they are taken to a trauma resuscitation room for rapid assessment. A team of doctors and nurses will work together to evaluate the patient's vital functions (Fig. 38.1). The patient is undressed, monitors are placed, intravenous access is established, and supplemental oxygen delivered. The overall management consists of a rapid primary survey, resuscitation of vital functions, a detailed secondary survey, and lastly the initiation of definitive care. The primary survey identifies injuries in a systematic fashion. The idea is to identify the greatest threat to life first and resuscitate accordingly. The treating physician is constantly reminded that the most obvious injuries may not pose the greatest threat to life. This process constitutes the ABCDE of trauma care.

- **A** – Airway maintenance with cervical spine protection;
- **B** – Breathing and ventilation;
- **C** – Circulation with hemorrhage control;
- **D** – Disability: neurologic status;
- **E** – Exposure/Environmental control: completely undress patient but prevent hypothermia.

The primary survey is a fluid yet systematic process. Each system is evaluated and resuscitation initiated simultaneously. The patient's airway is evaluated and protected before moving forward to assess breathing, circulation, and disability. Re-evaluation is another vital component of the primary survey. As the examiner moves through the primary survey he will constantly return to A – Airway to confirm an intact airway or to initiate an intervention should the airway status deteriorate. In this way the primary



Fig. 38.1 Trauma resuscitation – primary survey being completed by central doctor (yellow). Trauma team placing monitors, establishing intravenous access, disrobing patient.

survey is completed multiple times during the course of a trauma evaluation. Serial examination ensures that a system deterioration will be quickly detected and addressed.

Airway

The primary survey begins with an evaluation of the patient's airway while providing cervical spine protection. In the conscious, awake, and cooperative patient with a normal mental status and no distracting injuries this evaluation is easy and rapid. If the patient is speaking and the mental status is not altered (either from injury or drugs/alcohol) the airway is generally safe. In the same patient if there is no tenderness at the posterior midline of the cervical spine, the neck has normal range of motion without pain, and there are no focal neurological deficits, the cervical spine may be cleared clinically without further work-up.

In the obtunded or uncooperative patient airway evaluation can be challenging. The initial assessment should search for current or impending obstruction. The oral cavity should be suctioned and any foreign material removed. Facial fractures and tracheal injuries should be recognized as potential sources of airway obstruction. Facial and neck soft tissue injuries and burns can also progress to airway compromise (Fig. 38.2). Cervical spine injury should be assumed until it can be definitively ruled out. Initial measures to establish an airway (chin lift, jaw thrust) should be initiated while protecting the cervical spine. Protection of the cervical spine with an immobilization device (c-collar) should be initiated and maintained. Intervention with the establishment of a definitive airway should be a priority early in the resuscitation process. Indications for intubation can be separated into several broad categories:

- **inability to ventilate** – airway obstruction, chest trauma, paralysis, respiratory fatigue;
- **inability to oxygenate** – inhalation injury, acute respiratory distress syndrome (ARDS), pulmonary edema;

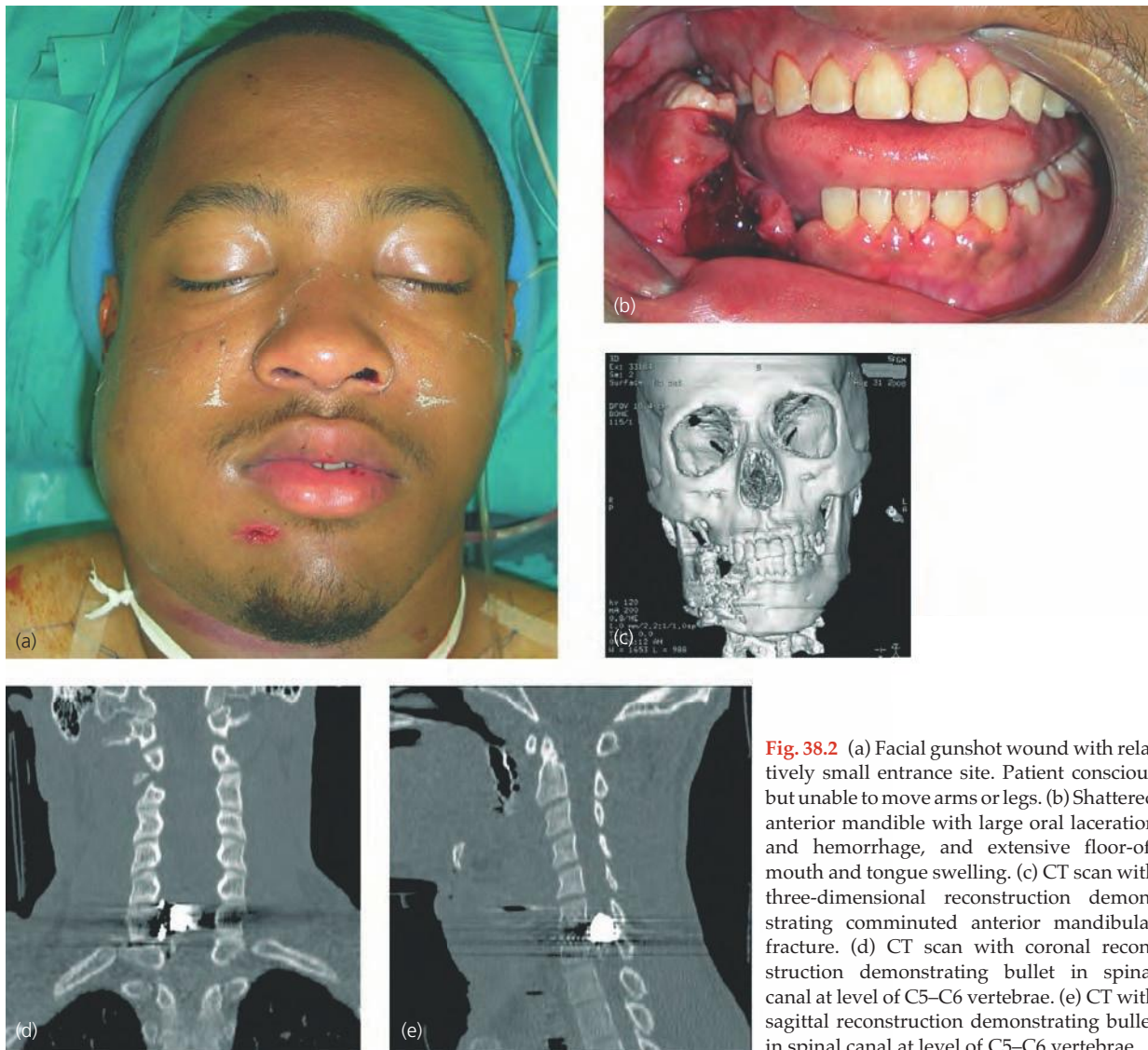


Fig. 38.2 (a) Facial gunshot wound with relatively small entrance site. Patient conscious but unable to move arms or legs. (b) Shattered anterior mandible with large oral laceration and hemorrhage, and extensive floor-of-mouth and tongue swelling. (c) CT scan with three-dimensional reconstruction demonstrating comminuted anterior mandibular fracture. (d) CT scan with coronal reconstruction demonstrating bullet in spinal canal at level of C5–C6 vertebrae. (e) CT with sagittal reconstruction demonstrating bullet in spinal canal at level of C5–C6 vertebrae.

- **airway protection** – unconsciousness, altered mental status, facial or head trauma, oral bleeding, aspiration risk (vomiting).

Once the airway has been evaluated and either cleared or definitively secured the practitioner will progress through the remainder of the primary survey. Patients that have been intubated should have a chest radiograph to confirm endotracheal tube position as soon as is reasonably possible. In addition to the anteroposterior (AP) chest film, a lateral cervical spine radiograph may be completed during the primary survey. A positive finding on the lateral film provides useful information. An absence of findings on the lateral film does not definitively rule out injury and the patient should remain in cervical spine protection until injury has been definitively ruled out. Patients at risk for cervical spine injury that cannot be cleared clinically should undergo a complete radiographic evaluation during the secondary survey. A complete plain film series would include an AP, lateral, odontoid view, and right and left oblique views. Computed tomography (CT) is recommended if plain films are

inadequate and in those patients with suspected cervical spine injury and head trauma. For a more detailed description of radiographic methods the reader is referred to Chapter 2.

Breathing

After a definitive airway is confirmed the patient's breathing is evaluated. The chest wall should be exposed to allow for a thorough inspection. Inspection will confirm appropriate chest movement with respiration. Areas of blunt trauma, penetrating injuries, open chest wounds, or flail segments (multiple rib fractures) are quickly identified. The lungs should be auscultated in all fields to confirm normal air movement. Palpation and percussion will confirm diaphragmatic excursion and may detect signs of blood or air in the pleural space.

Diminished or absent breath sounds may indicate a pneumothorax or hemothorax (separation of the visceral and parietal pleura with entry of air or blood into the pleural space). A small pneumothorax may go undetected during the primary survey and be rec-

ognized and treated during the secondary survey. A tension pneumothorax develops if, after chest wall or lung injury, a one-way valve mechanism exists that allows air to enter the pleural space without exit. With each respiratory cycle more air is drawn into the pleural space (Fig. 38.3). There is an eventual shift in the mediastinum to the contralateral side and compression of the major vessels entering the chest. With compression there is a decrease in venous return to the heart and resulting decline in cardiac output. This lesion is suspected in the patient with signs of chest trauma, absence of breath sounds on one side, hyperresonance of the chest wall, hypotension, and shift of the trachea to the contralateral side. The treatment is immediate decompression of the pleural space with a large-bore needle inserted through the second intercostal space along the mid-clavicular line, followed by the formal insertion of a thoracostomy tube.¹¹

Intubation and positive pressure ventilation can cause a relatively small pneumothorax to expand rapidly. Breathing should be repeatedly evaluated with auscultation of the chest and a chest radiograph should be obtained as soon as possible.

It is not uncommon for patients with chest trauma to have some degree of intrathoracic hemorrhage. Intrathoracic bleeding may be caused by disruption of a large central vascular structure, laceration of a systemic artery (internal mammary or intercostal), or from a lung parenchymal lesion. Enough blood can collect in the chest cavity to cause hypovolemic shock. Massive hemothorax should be suspected in trauma patients with chest trauma, diminished breath sounds, and signs of shock. Immediate thoracostomy tube placement allows for decompression of the lung parenchyma and monitoring of ongoing blood loss. A large volume (2000 ml) of blood may drain immediately and continue at rates of 100–200 ml/hour. Small lesions generally resolve spontaneously after chest tube placement and lung re-expansion. Persistent bleeding may require interventional or surgical repair.¹²

Circulation

Bleeding and hypovolemic shock account for 30–40% of trauma mortality. The volume status of a patient with multiple injuries needs to be rapidly evaluated and aggressively managed. The initial assessment begins immediately. A quick evaluation of the patient's level of consciousness, skin color, and pulses can be completed while monitors are being placed and intravenous access established. This information can help identify those patients in jeopardy of hypovolemic shock even before the blood pressure is first measured. When circulating blood volume is decreased cerebral perfusion may be critically impaired, resulting in altered levels of consciousness. Warm pink skin with rapid capillary refill is generally associated with the euvoletic patient. In contrast the patient with cold, clammy skin that appears ashen grey may be severely hypovolemic. Palpation of peripheral and central pulses can give a fast estimate of systolic blood pressure. Classically a palpable carotid pulse correlates with a systolic blood pressure of at least 60 mmHg, a palpable femoral pulse correlates with a systolic pressure of at least 60–70 mmHg, and a palpable radial pulse correlates to a systolic blood pressure of at least 80 mmHg. There has been some debate about the accuracy of these estimates.¹³ The general principle remains; full, slow, and regular peripheral pulses are generally associated with a normal circulating blood volume. A rapid, thready pulse is usually a sign of hypovolemia. Absent central pulses signify the need for immediate resuscitation.

Hemorrhage is defined as an acute loss of circulating blood volume. The average adult blood volume is approximately 7% of body weight (70 ml/kg). For a 70 kg adult the estimated blood volume is approximately 5 l. Children have estimated blood volumes of 8–9% of body weight and infants' estimated blood volumes are 9–10% of body weight. For the obese

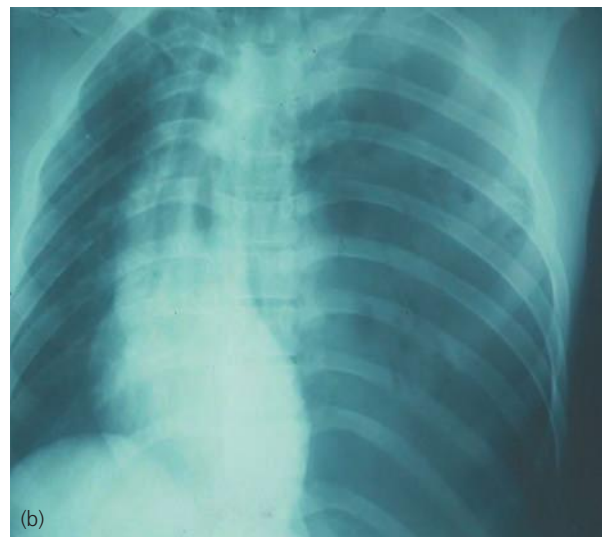


Fig. 38.3 (a) Small left pneumothorax with partial collapse of left lung (line) and air between the lung and the chest wall (pleural space). Also note multiple rib fractures on the left side. (b) Left tension pneumothorax with displacement of heart and trachea to the right side.

patient estimates of blood volume should be based upon ideal weight and not the patient's actual weight. Blood loss of 10–15% of a healthy person's blood volume can generally be tolerated without clinical sequelae. Blood donation typically takes 8–10% of a donor's blood volume.

There are multiple potential sources of bleeding in the trauma patient. External blood loss is managed during the primary survey with pressure on the wound. Large volumes of blood loss may occur with hemorrhage into the chest or abdomen. There may also be extensive bleeding associated with fractures of the pelvis or long bones.

Shock is defined as an abnormality in the circulatory system that results in inadequate tissue perfusion and oxygenation. Early signs of a collapsing circulatory system are tachycardia and peripheral vasoconstriction. As the system continues to fail, perfusion to central organs and muscle will decrease in order to preserve cerebral perfusion. Shock in the trauma patient is most likely caused by acute blood loss. Other categories of shock include cardiogenic, septic, and neurogenic.¹⁴

Classifications of hemorrhage were developed to help recognition of the early signs of hypovolemic shock and to guide volume replacement. Hypotension resulting from acute blood loss occurs relatively late. Early signs of hypovolemic shock include tachycardia and decreased peripheral perfusion. Classes of hemorrhage are defined by blood loss (volume and percentage) and the resulting physiologic responses (blood pressure, heart rate, respiratory rate, urine output, and mental status).

- Class 1 hemorrhage is less than 750 ml or less than 15% of estimated blood volume. Physiologic parameters are normal. Heart rate is less than 100 beats per minute. Blood pressure and respiratory rates are normal. Urine output is greater than 30 ml/hour. The patient may be slightly anxious but mental status is otherwise normal.
- Class 2 hemorrhage is 750–1500 ml or 15–30% of estimated blood volume. These patients will have a drop in blood pressure with a resulting tachycardia (greater than 100 beats per minute). They will also show a decline in urine output (20–30 ml/hour) and an increased respiratory rate (20–30 breaths per minute). These patients may show moderate anxiety.
- Class 3 hemorrhage is 1500–2000 ml or 30–40% of estimated blood volume. These patients are tachycardic (greater than 120 beats per minute), hypotensive, and tachypneic (30–40 breaths per minute). Urine output continues to decline (5–15 ml/hour). Confusion may be an early sign of decreased cerebral perfusion.
- Class 4 hemorrhage (greater than 2000 ml or 40% of estimated blood volume) is immediately life threatening. Symptoms include marked tachycardia (greater than 140 beats per minute), tachypnea

(greater than 35 breaths per minute), and a significant decrease in systolic blood pressure. Urine output is negligible and patients are confused or lethargic.

The diagnosis and management of shock occurs rapidly during the primary survey. Access to the vascular system is critical. Initial vascular access is obtained with two large-bore (16 gauge or larger) peripheral intravenous catheters. If peripheral access is inadequate or not readily obtainable, central venous access may be established through the jugular, subclavian, or femoral veins. With initial intravenous access, samples may be sent and blood typed and cross-matched for possible transfusion.

Resuscitation of hemorrhagic shock begins with the administration of 2 l of crystalloid solution (normal saline, lactated Ringer's solution). Further fluid resuscitation and the need to transfuse blood are based upon estimates of the volume of blood loss and the patient's response to the initial fluid bolus. If the patient had minimal blood loss (10–15% of estimated blood volume) and a rapid response to the initial fluid with a return to normal vital parameters they are not likely to require blood transfusion. If the patients had moderate blood loss (20–40% of estimated blood volume) and only a transient response to the initial fluid bolus then ongoing fluid resuscitation is anticipated. It is likely that the patient will require blood transfusion but type-specific blood may be available. In the patient with severe hemorrhage (over 40% of estimated blood volume) immediate transfusion is required. In this situation type O blood will be used until type-specific blood is available.¹⁵

Urinary catheters and arterial lines may be placed during the primary survey and both provide valuable information on the circulatory status. Urine output is a sensitive indicator of volume status and reflects renal perfusion. Urinary catheters are contraindicated in patients in whom a urethral injury is suspected. Urethral injury should be suspected with blood at the penile meatus, perineal ecchymosis, blood in the scrotum, or pelvic fracture. An arterial catheter provides immediate monitoring of the patient's blood pressure. It also provides access for repeated laboratory samples.

Disability (neurologic evaluation)

The primary survey concludes with a rapid neurologic evaluation. A decline in the patient's level of consciousness may be due to a decrease in cerebral perfusion or cerebral oxygenation, or may be due to an intracranial injury. The first response to an altered mental status is to re-evaluate airway, breathing, and circulation. A rapid neurologic assessment should include an evaluation of sensory and motor function and rectal tone. Other potential causes of an acute decline in level of consciousness are hypoglycemia and alcohol or drug intoxication. Traumatic brain

injury should remain high on the differential list until proven otherwise.

The Glasgow Coma Scale (Table 38.1) is a rapid objective clinical measure of neurologic function. The scale was initially published by two neurosurgeons

Table 38.1 Glasgow Coma Scale (GCS).

Eye opening
Spontaneous – 4
To speech – 3
To pain – 2
None – 1
Motor response
Obeys commands – 6
Localizes to pain – 5
Normal flexion (withdrawal) – 4
Abnormal flexion (decorticate) – 3
Abnormal extension (decerebrate) – 2
Flaccid – 1
Verbal response
Oriented – 5
Confused conversation – 4
Inappropriate words – 3
Incomprehensible sounds – 2
None – 1

(Graham Teasdale and Bryan Jennett) at the University of Glasgow. The scale assesses eye opening, motor response, and verbal response. Each category is scored based upon best response and the scores are tallied to determine a Coma Score. The highest score obtainable is 15 (indicating an unaltered, awake patient) and the lowest is 3 (indicating deep coma).¹⁶

Head trauma and coma can be graded based upon the Glasgow Coma Score. Scores of 14–15 are considered mild, 9–13 moderate, and 3–8 severe. The patient's Glasgow Coma Score along with mechanism of injury (blunt versus penetrating) and type of head trauma (skull fracture, intracranial lesion) are used to triage the patient's neurologic injuries.

Multiple types of injuries are possible after head trauma. Scalp lacerations and skull fractures may be identified during the primary survey (Fig. 38.4). Intracranial lesions include epidural and subdural bleeding, intracerebral bleeding, contusion, concussion, and diffuse hypoxic injury.

Skull fractures may occur along the convexity of the cranial vault (more common) or at the cranial base (Fig. 38.5). Fractures may be linear, complex, comminuted, or depressed. Linear fractures of the skull convexity are most commonly seen in the trauma patient. Open or depressed fractures may be diagnosed clinically. Closed, non-depressed fractures may require

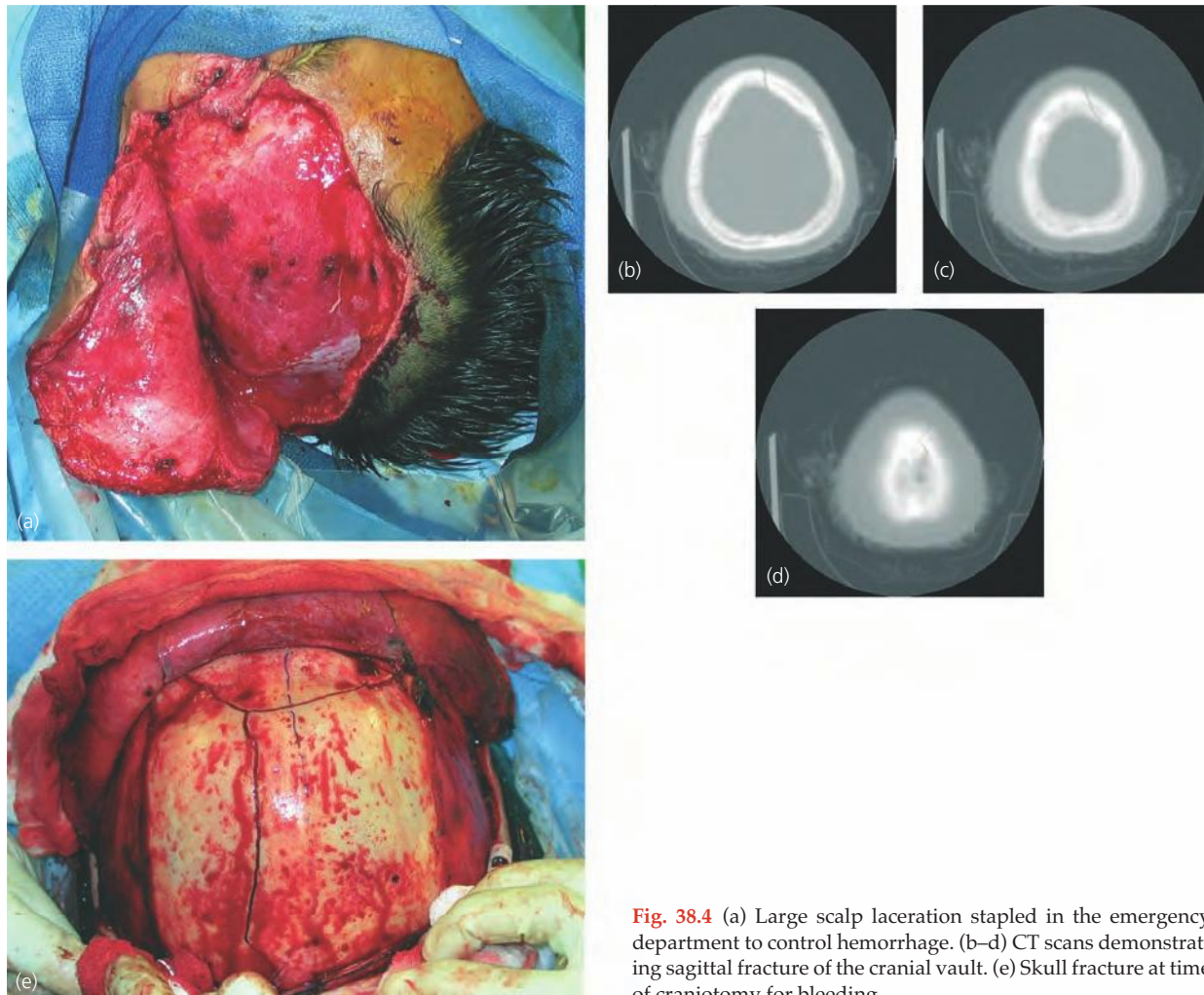


Fig. 38.4 (a) Large scalp laceration stapled in the emergency department to control hemorrhage. (b–d) CT scans demonstrating sagittal fracture of the cranial vault. (e) Skull fracture at time of craniotomy for bleeding.

CT for diagnosis. Fractures of the skull base are much less commonly seen in the trauma patient (4% of skull fractures). Skull base fractures are difficult to diagnose clinically but may be suspected in the patient with periorbital ecchymosis (raccoon eyes), mastoid ecchymosis (battle sign), cerebral spinal fluid rhinorrhea/otorrhea, or blood behind the tympanic membrane. Skull fractures increase the likelihood of a subdural or epidural hematoma. They also provide a pathway for the entry of bacteria or air (pneumocephalus).¹⁷

Hemorrhage may occur beneath the dura (subdural) or between the dura and skull (epidural); each has characteristic clinical and radiographic features. Epidural hematomas occur in up to 10% of patients with severe head trauma. They typically evolve rapidly. Many of these patients are unconscious when first seen but may have a "lucid" interval of minutes to hours before coma develops. These hematomas are located between the skull and dura and are lenticular in shape. They are most often seen in the temporal or temporoparietal region and usually result from a tear of the middle meningeal artery. The high pressure arterial bleeding of an epidural hematoma can lead to irreversible brain injury and death within hours. Rapid surgical evacuation and ligation of the damaged vessel is indicated.

Subdural hematomas are more common than epidural hematomas. They are caused by tearing of veins traversing the dura and result in blood collecting between the dura mater and the arachnoid. Blood collects more slowly in subdural hematomas but the underlying brain injury is typically much more severe than that with epidural hematomas. On imaging studies subdural hematomas appear as crescentic collections over the convexity of one or both hemispheres (Fig. 38.6). Blood tends to conform to the contour of the underlying cerebral cortex. These are more com-

monly seen in the frontotemporal region. Patients, if conscious, may present with a unilateral headache and slightly enlarged pupil. Coma, hemiparesis, and pupillary asymmetries are associated with larger hematomas. In rapidly deteriorating patients hematoma evacuation may be required. In smaller lesions no intervention may be necessary.¹⁸

The skull is a rigid container and contains brain parenchyma, fluid (interstitial and cerebrospinal), and blood (arterial and venous). The expansion of any of these compartments (e.g. edema) or the addition of a space-occupying process (e.g. hematoma) can lead to an elevation of the intracranial pressure. As intracranial pressure begins to rise above normal (15 mmHg) a progression of neurologic abnormalities will develop. Effects on the cerebral cortex will produce a decline in level of consciousness. Effects on the mid-brain will produce fixed, dilated pupils. Effects on the pons will result in loss of corneal reflex. Effects on the medulla will result in apnea, hypotension, and death.¹⁹

During the primary survey, once airway, breathing, and circulation are established and stabilized, the focus becomes preservation of cerebral function. Patients with physical findings suggestive of an intracranial process (unequal pupils, asymmetric motor exam) should undergo an immediate head CT scan. Patients with less severe head trauma who are otherwise stable but have symptoms of headache, vomiting, drug or alcohol intoxication, persistent amnesia, or seizures should also have a head CT scan while in the emergency department.²⁰

Exposure/environmental control

The primary survey concludes with complete exposure of the patient. The patient should be undressed to allow for a complete head to toe physical examination. During the resuscitation, and particularly once the patient is undressed, it is critical to protect the patient from developing hypothermia. Hypothermia develops in up to 70% of trauma patients at some point during resuscitation. Exposure, paralysis, and fluid administration all contribute to lowering the patient's core temperature. Hypothermia can produce

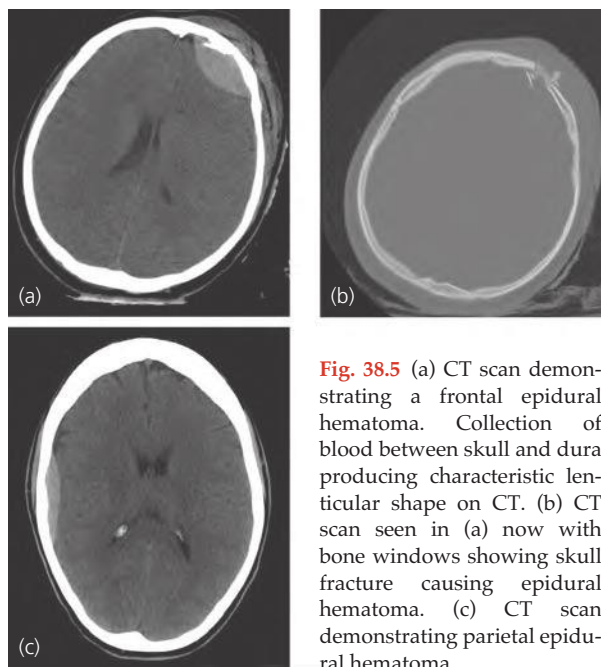


Fig. 38.5 (a) CT scan demonstrating a frontal epidural hematoma. Collection of blood between skull and dura producing characteristic lenticular shape on CT. (b) CT scan seen in (a) now with bone windows showing skull fracture causing epidural hematoma. (c) CT scan demonstrating parietal epidural hematoma.

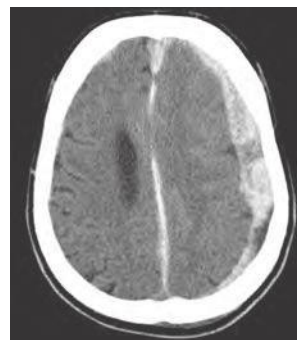


Fig. 38.6 CT scan demonstrating a subdural hematoma. Blood between the dura and brain with irregular borders and causing an obvious mass effect on the brain tissue with loss of ventricle and shift of midline structures.



Fig. 38.7 (a, b) Plain radiographs demonstrating radial and ulnar fractures mid-diaphysis. (c, d) Plain radiographs demonstrating comminuted, displaced distal femur fracture.

a relative coagulopathy. It alters platelet function, the coagulation cascade, and the fibrinolytic system. A drop in the core temperature of just a few degrees is enough to produce a marked decrease in clotting ability. Hypothermia also produces a dramatic increase in oxygen consumption. Studies have shown that a decrease in the core temperature of 0.3°C produces a 7% increase in oxygen consumption and a decrease of 1.2°C produces a 92% increase in oxygen consumption. Shivering has been shown to increase oxygen consumption by 400%. Hypothermia can produce negative inotropic changes in the heart and respiratory depression, and exacerbate hyperglycemia by decreasing insulin production and creating end-organ insulin resistance. Heaters, forced air warming devices, and administering warmed fluids should be started early during the evaluation of trauma patients.²¹

Secondary survey

The secondary survey is a complete history and head to toe physical examination of the patient. This is not initiated until the primary survey is complete and the patient is relatively stable. The secondary survey is an incredibly important part of the overall trauma evaluation. The reported incidence of missed injuries in the trauma patient ranges from 8–60%. Studies have shown that practitioners are most likely to miss injuries in the most critically injured patients.²² A large proportion of missed injuries involve orthopedic injuries, including fractures in the upper and lower extremities. Some centers now advocate a tertiary survey to be completed while the patient is in hospital as a failsafe method of identifying potentially missed injuries.

The secondary survey begins with a more detailed patient history. Details of the current injury are



Fig. 38.8 Plain radiograph demonstrating pelvic fracture with wide diastasis of the pubis with widening of the sacroiliac joints.

obtained from the patient, family, or other witnesses. A full medical history including medical conditions, medications, drug allergies, and tobacco, alcohol, and substance abuse are also obtained.

The physical examination begins with a re-evaluation of the patient's vital signs. If the patient's vital signs are worsening or if there is a deterioration of any system evaluated during the primary survey, the secondary survey is halted and resuscitation is continued.

A brief neurological examination is completed and should include a recalculation of the Glasgow Coma Score. Cranial nerves are evaluated with particular attention on the pupillary exam. Motor and sensory examinations of the upper and lower extremities are completed bilaterally. Deep tendon reflexes are also evaluated in upper and lower extremities. Focal abnormalities in the exam should alert the examiner to potential brain or spine injuries.

Next a full maxillofacial examination is completed. The scalp, facial skeleton, eyes, ears, nose, and oral cavity are evaluated. Visual acuity is assessed and fundoscopic and otoscopic exams are completed. The neck is evaluated for cervical spine trauma, penetrating injuries, and evidence of carotid injury.

Penetrating neck injuries are often characterized by the anatomic zone of the neck involved. Zone one is bound by the thoracic inlet inferiorly and the cricoid cartilage superiorly. Structures at greatest risk in this zone are the subclavian vessels, brachiocephalic veins, common carotid arteries, aortic arch, jugular veins, trachea, esophagus, lung apices, cervical spine, spinal cord, and cervical nerve roots. Zone two is bound by the cricoid cartilage inferiorly and the inferior border of the mandible superiorly. Important structures in this region include the carotid and vertebral arteries, jugular veins, pharynx, larynx, trachea, esophagus, and cervical spine and spinal cord. Zone three is from the inferior border of the mandible to the base of the skull. Penetrating injuries superficial to the platysma muscle may require little more than a simple closure. Penetrating injuries deep to the platysma may cause acutely life-threatening injuries or may harbor occult injuries that can progress to life-threatening over time if not diagnosed. Potential work-up could include neck exploration, angiography, CT, esophagoscopy, and bronchoscopy.²³

The chest is evaluated next and should undergo inspection, palpation, percussion, and auscultation. The chest wall should be evaluated front and back for signs of trauma. The ribs, clavicles, sternum, and thoracic and lumbar spine should be palpated. Areas of pain on palpation should be evaluated for fracture. Percussion and auscultation will confirm normal air flow.

Next the abdomen is evaluated. The abdomen should be inspected for signs of trauma including seat belt marks, ecchymosis, and penetrating injuries. Palpation of a soft non-tender abdomen in a neurologically intact patient without other distracting injuries and without intoxication may be followed without further work-up. An abnormal (tense or tender) or unreliable (unconscious, intoxicated or altered) exam should be followed by CT scans with both intravenous and intragastric contrast or by an ultrasound evaluation. An unstable patient with a high clinical suspicion for an abdominal injury may proceed straight to the operating room for an exploratory laparotomy.

The FAST exam (focused assessment with sonography in trauma) is used to identify free fluid (usually blood) in the peritoneal, pericardial, and pleural spaces. FAST scans attempt to identify free fluid in four regions: right upper quadrant (abdomen), left upper quadrant (abdomen), behind the bladder in the pelvis, and in the pericardial space. It is not as accurate as CT at diagnosing abdominal injuries. It is most helpful in evaluating the abdomen and pericardial

space in the unstable patient or in those who could be observed without CT. It remains an accurate evaluation that can be completed rapidly and repeated, and does not require a radiologist or technician. The exam is portable and can be completed bedside in the emergency room or hospital ward. The ultrasonography provides limited information about solid organ injury, the retroperitoneum or the diaphragm.²⁴ It has also been shown to have decreasing accuracy in patients with pelvic fractures.²⁵

The physical examination should continue with an examination of the extremities, pelvis, and genitourinary system. Obvious deformities of the long bones warrant radiographic and orthopedic evaluation. Ecchymosis and edema should also alert the examiner to possible underlying fractures. Peripheral pulses should be evaluated. Abnormalities in peripheral pulses should be evaluated for vascular injury. The pelvis is examined for any deformity and palpated for instability and pain. Pelvic fractures should be suspected when there is ecchymosis over the iliac wings, pubis, labia, or scrotum. The anterior posterior pelvis film is a useful initial screen for pelvic fractures and can be completed in the emergency department.

The perineum should be examined for contusions, lacerations, and urethral bleeding. A rectal examination should be completed to assess rectal tone, prostate position, rectal blood, and the integrity of the rectal wall. Female patients should also undergo a vaginal examination looking for blood and vaginal wall injuries. Female patients should also have a pregnancy test completed.

Serial and repeated examination and evaluation are hallmarks of the trauma evaluation. It is well documented that an initial secondary survey is inadequate in many critically injured patients. The concept of a tertiary examination developed in the early 1990s. The idea was that the trauma patient should be re-evaluated early in the course of hospitalization with the intention of looking for missed injuries. A complete and thorough head to toe physical examination is completed. All radiographic studies should be reviewed by a radiologist.

Conclusion

In recent decades there have been many advances in trauma care. Patients are resuscitated rapidly and are now surviving injuries that would have been fatal in the past. Oral and maxillofacial surgeons undergo education and training and oral and maxillofacial surgery residents now participate in Advanced Trauma Life Support courses. Oral and maxillofacial surgeons will always be involved with the early evaluation and treatment of patients with facial trauma. Having an understanding of the complexity of trauma care and the details of an appropriate trauma evaluation will improve patient outcomes.

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Chapter 39

Traumatic Dental Injuries

Lars Andersson and Jens O. Andreasen

Traumatic dental injuries (TDI) are the most common of the oral injuries. The prognosis for some TDI depends very much on correct early emergency management at the place of accident and at the clinic. This chapter presents how to diagnose, classify, and treat TDI with emphasis on the emergency phase. An overview of pulpal and periodontal healing and complications after TDI is also given.

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somatic injuries, are most frequently seen at ages 15–25 years and occur throughout life (Fig 39.1).^{1,3}

In a study of patients presenting with traumatic oral injuries in Sweden, traumatic dental injuries (TDI) were the most common oral injury (92%), followed by orofacial soft tissue injuries (28%), whereas fractures of the maxillofacial bones were relatively rare and seen in only 6% of such patients.¹ Most studies report an annual incidence of TDI at 1–2% of the population.^{2–4} The prevalence is high and every fourth boy and every fifth girl have already sustained dental injuries at the age of 14.⁵ A study in the UK showed that one in five children has sustained a TDI to the permanent teeth before leaving school.⁶ A comprehensive national survey in the USA showed that one in four adults had evidence of TDI.⁷ Due to the high number of injured individuals and often complex treatment, TDI are associated with high costs for society and the individual.^{8–10}

The prognosis of treatment for some dental injuries, especially avulsed teeth, depends very much on early and correct management at the place of the accident and during emergency treatment at the clinic.^{2,11–16}

Examination and diagnosis of dental injuries

History taking is important with regard to when, where, and how the injury occurred, as well as the patient's general health and possible medication. Be alert to other injuries and symptoms indicating more serious injuries. See Chapter 38 regarding assessment of the injured patient. It is also of value to ask what has been done before the patient reached the clinic, such as any treatment carried out elsewhere and how avulsed teeth have been stored.

A brief summary is given here of the important issues for emergency examination and for TDI. For a more detailed description the reader is referred to recent textbooks and manuals on the subject.^{2,16} As for oral and maxillofacial injuries, it is important to

Epidemiology of traumatic dental injuries and relation to oral and somatic injuries

Oral injuries (dental, soft tissue, and bone) caused by trauma occur frequently. They are most common during the first 12 years of life and are rare after the age of 30.^{1–3} In contrast, other injuries to the body,

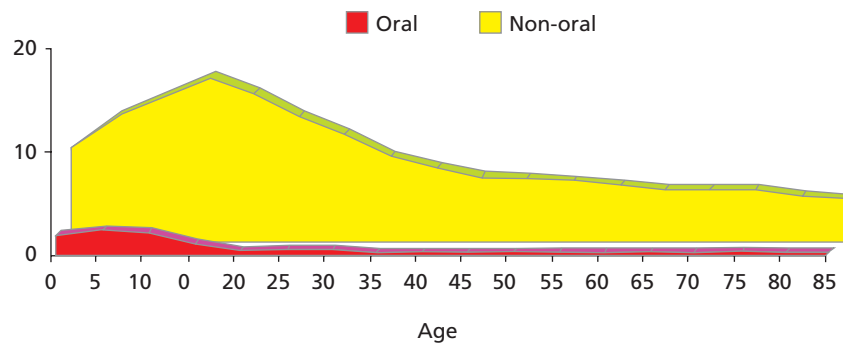


Fig. 39.1 Incidence per 100 inhabitants per year for oral and non-oral injuries. From Andreasen *et al.* 2007.²

examine and treat TDI in a systematic way. Examine from the outside towards the inside and treat from the inside towards the outside is a helpful principle for a systematic approach for examining oral injuries. Suturing of lips should ideally be carried out after intraoral emergency examination and treatment has been performed, otherwise it may be difficult to enter the oral cavity once edema has started to develop.

The possibility of inhaling or swallowing teeth at the time of injury should always be considered when teeth, parts of the teeth or prosthetic appliances are missing. This is especially important in the unconscious patient; if there is reason to suspect inhalation or swallowing of a tooth or dental appliance, it is important that radiographs of the chest and the abdomen be considered. Inhalation of foreign bodies is normally associated with symptoms such as coughing but may also occur in a conscious patient without producing symptoms.

Examination of crowns of teeth should be carried out in order to look for presence and extent of fractures and pulp exposures. Crown–root fractures in the molar and premolar regions should be expected when there has been an indirect trauma such as a blow to the chin. Crown–root fractures in one quadrant are very often accompanied by similar fractures on the same side of the opposing jaw. For this reason it is therefore necessary to examine occlusal fissures of all molars and premolars to detect possible fractures (Fig. 39.2).



Fig. 39.2 Induced dental trauma. (a) Impact was transferred via the chin to the dental arches. (b) Crown–root fractures were inflicted in both right premolars by the forceful occlusion. From Andreasen *et al.* 2007.²

The fracture surface should be carefully examined for the extent of fracture in dentin and pulp exposures (Fig. 39.3). When pulp is exposed, the size and location should be recorded. In some cases, the dentin layer may be so thin that the outline of the pulp can be seen through the dentin wall. One should take care

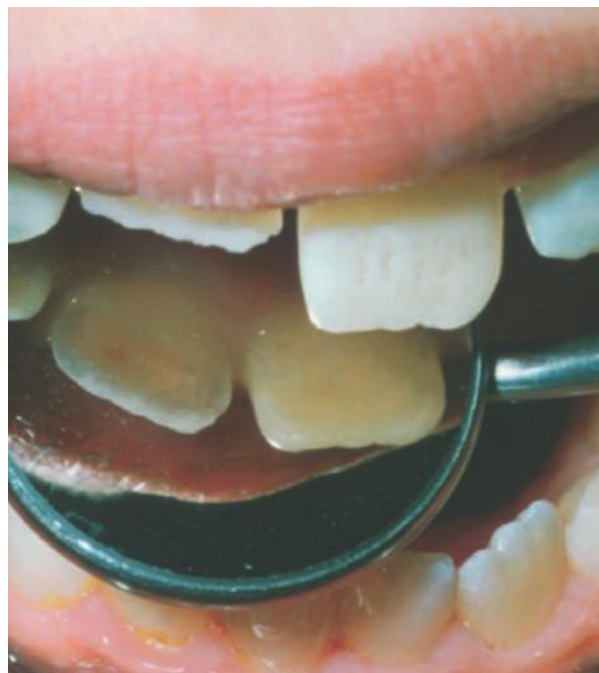


Fig. 39.3 Right central incisor with crown fracture of dentin showing pulpal exposure.

not to perforate the thin dentinal layer during the examination.

Displacement and avulsion of teeth are usually evident by visual examination; however, minor abnormalities can often be difficult to detect. In such cases, it is helpful to examine the occlusion as well as radiographs taken at various angulations. In cases of tooth luxation, the direction of the dislocation as well as extent should be recorded. It is important to remember that, apart from displacement and interference with occlusion, laterally luxated and intruded teeth present very few clinical symptoms (Fig. 39.4). Moreover, these teeth are normally firmly locked in their displaced position and do not usually demonstrate tenderness to percussion. While radiographs can be of assistance, diagnosis is confirmed by the percussion tone, when tapping on the tooth with the handle of an instrument. A high, bony percussion tone is registered compared to the normal sound of uninjured teeth. When a Le Fort I fracture is present a change in percussion sound to a cracked-like sound can be noticed.

In the primary dentition, it is of utmost importance to diagnose the direction of dislocation of the apex of a displaced primary tooth, since this tooth is located in close relationship to the permanent successors and can impinge on the development of the permanent



Fig. 39.4 Intruded incisors. The teeth are impacted and locked in the bone and show no signs of tenderness to percussion. A high, bony percussion sound is registered.

successor (Fig. 39.5). This diagnosis is often best done by palpating the alveolar process from a vestibular approach.

The patient may also react with pain to percussion of a tooth which is indicative of damage to the periodontal ligament. As with all examination techniques used at the time of injury, the percussion test should be started on a non-injured tooth to ensure a reliable patient response. In smaller children, the use of a fingertip can be a gentler diagnostic tool.

Patients can usually detect and guide the examiner to the place where there has been a change in the occlusion. Abnormalities in occlusion can indicate fractures of the jaw or alveolar process or a lateral luxation (Figs 39.6–39.8). All teeth should be tested for abnormal mobility, both horizontally and axially. It should be remembered that erupting teeth and primary teeth undergoing physiologic root resorption always exhibit some mobility. The typical sign of alveolar fracture is movement of adjacent teeth when the mobility of a single tooth is tested (Fig. 39.6). Palpation of the alveolar process is important for detection of fractures with dislocations.

When mobility of a tooth is detected it is not possible to discriminate between luxation injuries and root fractures without radiographic examination (Figs 39.9 and 39.10). The site of the root fracture will have an influence on the degree of mobility.

Pulp testing following traumatic injuries is a controversial issue. These procedures require cooperation and a relaxed patient, in order to avoid false reactions. However, this is often not possible during initial treatment of injured patients, especially children. The interpretation of pulpal sensibility tests performed immediately after traumatic injuries is complicated by the fact that sensitivity responses can be temporarily or permanently decreased, especially after luxation injuries. However, repeated testing has shown that normal reactions can return after a few weeks or months. Moreover, teeth which have been loosened can elicit pain responses merely from pressure of the pulp testing instrument. It is therefore

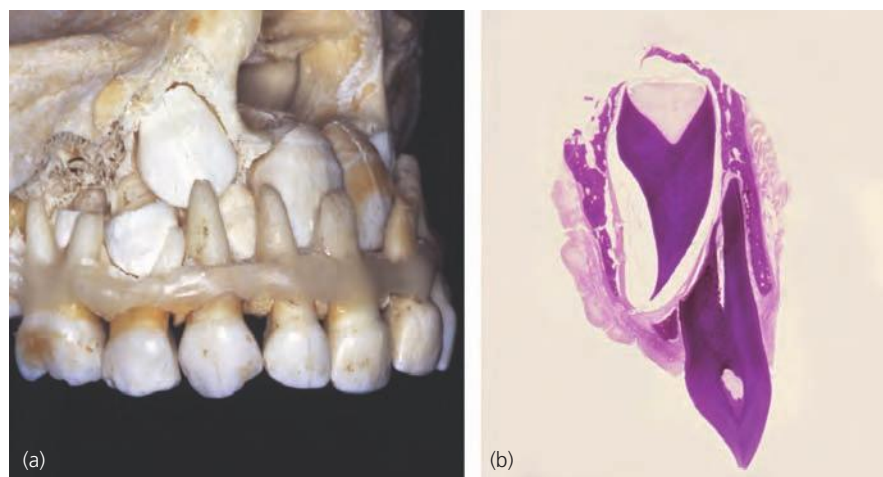


Fig. 39.5 (a) Anatomic relationship between primary incisor and its permanent successor in a human skull. (b) Histologic relationship between primary incisor and its permanent successor. From Andreasen *et al.* 2007.²



Fig. 39.6 A step in the mandibular incisor level indicating mandibular body fracture.



Fig. 39.7 A rift in the palatal mucosa and a step in the premolar occlusal level indicating a fracture of the maxillary alveolar process.



Fig. 39.8 Primary contact in premolar region indicating an alveolar process fracture in the maxilla.



Fig. 39.9 Central incisors with root fractures.



Fig. 39.10 Radiograph showing fractures of the roots 11 and 21.

important to reposition and immobilize such teeth, e.g. root-fractured or extruded incisors, prior to pulp testing. Another important factor to consider when testing the pulp is the stage of eruption; teeth under root development sometimes show an unreliable response. Teeth undergoing orthodontic movement display higher excitation thresholds. If local anesthetics are to be administered for various treatment procedures, pulp testing should be performed prior to doing this. Pulpal sensibility testing is difficult in the emergency situation but it is helpful, as such tests have a strong predictive value for later pulp healing complications. For this reason it is important to carry out a sensibility test within the first week after trauma to establish a point of reference for evaluating pulpal status at later follow-up examinations.

Radiographic examination of TDI

This is an overview of suitable examinations for patients who have been subjected to dentoalveolar trauma. For more details of radiographic examination the reader is referred to Chapter 2 in this book.

Dental films

All injured teeth should be examined radiographically to diagnose injuries and to reveal the stage of root formation which is important for the choice of treatment method. The ideal method is the use of different angulations for each traumatized tooth, using a standardized projection technique. Thus, a traumatized anterior region of the maxilla is covered by one occlusal film, projected steeply superiorly, and three periapical exposures perpendicular to the long axes of the teeth, where the central beam is directed between the central incisors and for each lateral incisor. This procedure ensures diagnosis of even minor dislocations or root fractures. It is important to bear in mind that a steep occlusal exposure is of special value in the diagnosis of root fractures and lateral luxations with oral displacement of the crown.

Most root fractures are disclosed by radiographic examination provided that they are steeply superiorly projected. A widening of the periodontal space is seen in lateral and extrusive luxations, whereas intruded teeth often demonstrate a lack of periodontal space.

Extraoral radiographs may be of value for determining the direction of dislocation of intruded primary incisors. Dislocated tooth fragments within a lip laceration can be demonstrated radiographically by the use of an ordinary film placed between the dental arches and the lips (Fig. 39.11). A short exposure time is advocated for this situation.

Panoramic technique

This method gives an excellent overview of the dentoalveolar region. It is especially useful in cases where a jaw fracture is suspected.



Fig. 39.11 Radiograph of a lacerated lip showing coronal fragment of a fractured incisor.

Conventional computed tomography scanning

This is a very useful method in the diagnosis of maxillofacial fractures; however, the resolution is not optimal and radiation exposure is too high to make it useful for dental trauma diagnosis.

Micro computed tomography scanning, cone-beam computed tomography

Conventional dental and panoramic images have limited diagnostic ability due to their two-dimensional character. Resolution in computed tomography (CT) scans is not high enough to be useful for diagnosis in dental trauma. Cone-beam CT has high enough resolution to be a valuable tool for diagnosis of different dental injuries (Fig. 39.12). For more information regarding cone-beam CT see Chapter 2.

Classification and clinical findings

TDI can be classified in many ways but the following system based on World Health Organization classification, modified by Andreasen,^{2,17} is well suited to the clinical situation; following this system facilitates treatment.

Injuries to the hard dental tissues and the pulp

A schematic overview of hard tissue injuries is presented in Fig. 39.13.



Fig. 39.12 Cone-beam CT picture of a central incisor showing a lateral luxation with fracture of the labial bone plate. From Andreasen *et al.* 2007.²

Crown infraction

This is incomplete fracture of the enamel without loss of tooth substance (Fig. 39.13a mesial). Various patterns of craze lines can be detected depending on the location and direction of trauma, e.g. vertical, horizontal, or oblique lines.

Uncomplicated crown fracture

This is a fracture with loss of tooth substance (Fig. 39.13a distal, Fig. 39.13b). It involves enamel or enamel and dentin without pulp exposure. The exposed dentin usually gives rise to symptoms such as sensitivity to hot and cold stimuli and mastication.

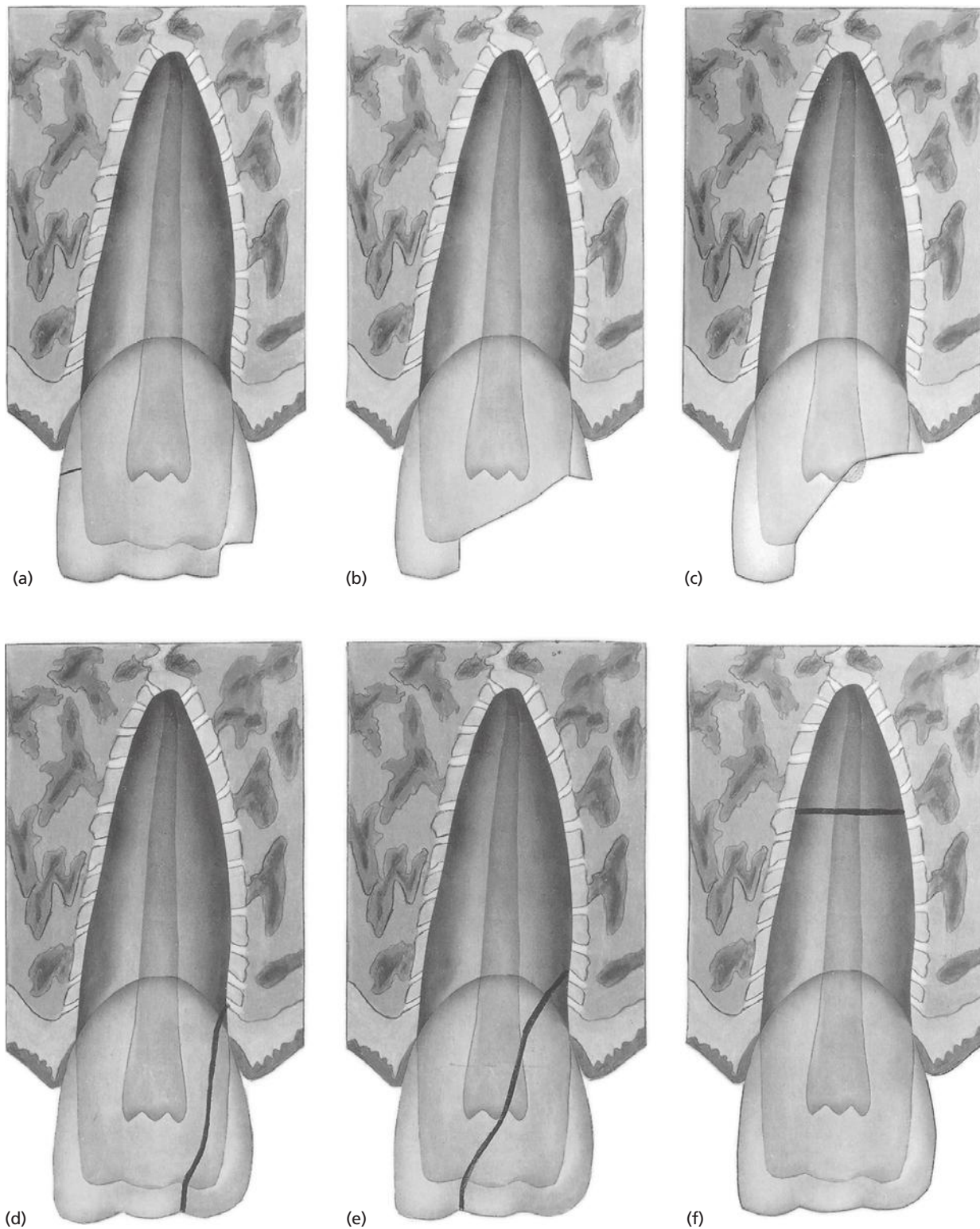


Fig. 39.13 Injuries to the hard dental tissues and pulp. (a) Crown infraction and uncomplicated fracture without involvement of dentin. (b) Uncomplicated crown fracture with involvement of dentin. (c) Complicated crown fracture. (d) Uncomplicated crown-root fracture. (e) Complicated crown-root fracture. (f) Root fracture. From Andreasen *et al.* 2007.²

Complicated crown fracture

This is a fracture with loss of enamel and dentin with pulp exposure (Fig. 39.13c). The exposed pulp often gives rise to pain and sensitivity.

Uncomplicated crown–root fracture

This is a fracture of enamel, dentin, and root structure without pulp exposure (Fig. 39.13d).

Complicated crown–root fracture

This is a fracture of enamel, dentin, and root structure with pulp exposure (Fig. 39.13e). Vitality testing is usually positive. Even with pulp exposure, symptoms are usually few and limited to pain upon heat and cold exposure when touching the fragment or biting on the fragment.

Root fracture

This is a horizontal root fracture comprising dentin, cementum, and pulp (Fig. 39.13f). The fracture is located within the alveolus. The coronal fragment may be mobile and displaced. The tooth might be slightly extruded and tender to percussion.

Injuries to the periodontal tissues

A schematic overview of injuries to the periodontal tissues is presented in Fig. 39.14.

Concussion

This is an injury to the tooth-supporting structure without abnormal loosening or displacement of the tooth (Fig 39.14a). The tooth has not been displaced and does not have increased mobility. It is tender to touch or tapping.

Subluxation

Subluxation is injury to the tooth-supporting structure with abnormal loosening, but without displacement of the tooth (Fig 39.14b). The tooth is tender to touch or tapping and has increased mobility. There might be bleeding from the gingival sulcus.

Extrusive luxation (extrusion)

This is partial displacement of the tooth out of its socket (Fig 39.14c). The tooth appears elongated and is excessively mobile. There is bleeding from the gingival sulcus.

Lateral luxation

Displacement of the tooth occurs in a direction other than axially (Fig. 39.14d). The tooth is displaced in its

socket, most commonly with the crown in a retroclined position. The tooth is most often immobile due to its locked position in the socket and there is a high ankylotic percussion tone.

Intrusive luxation (intrusion)

The tooth has been displaced axially into the alveolar bone through the alveolar socket (Fig 39.14e). The crown appears shortened. There is usually bleeding from the gingiva. Percussion tone is high and metallic similar to that of an ankylosed tooth. The percussion tone can be used to distinguish an intruded tooth from a partially erupted or unerupted tooth.

Avulsion (exarticulation, total luxation)

This is complete displacement of the tooth out of its socket (Figs 39.14f and 39.15; see also Fig. 39.20). There is bleeding from the socket which is empty.

Treatment

The following treatment guidelines focus on what should be carried out in the emergency situation for permanent teeth.^{18,19} For more detailed information regarding treatment after the emergency situation see textbooks and articles^{2,3,13,20–25} and International Association of Dental Traumatology (IADT) guidelines,^{18,19} which are regularly updated on the Internet (www.iadt-dentaltrauma.org).

Infraction

No treatment is necessary. In cases of multiple craze lines, they might later be sealed with an unfilled resin to prevent future discoloration.

Crown fracture**Uncomplicated crown fracture**

- If the fracture is confined to enamel; a slight grinding or smoothing of sharp edges may be sufficient.
- With more extensive loss of enamel, a later composite restoration is necessary as recontouring is not sufficient.
- Fracture comprising dentin and enamel but the fragment is not found: the exposed dentin should be covered with glass ionomer as an emergency treatment. It is possible to make a permanent composite restoration using a bonding agent in the emergency situation, or this treatment can be performed at a later stage.
- Fracture comprising dentin and enamel and the fragment is found: the fragment can immediately or later be bonded to the tooth (Fig. 39.16). If it is decided to perform the bonding later it is impor-

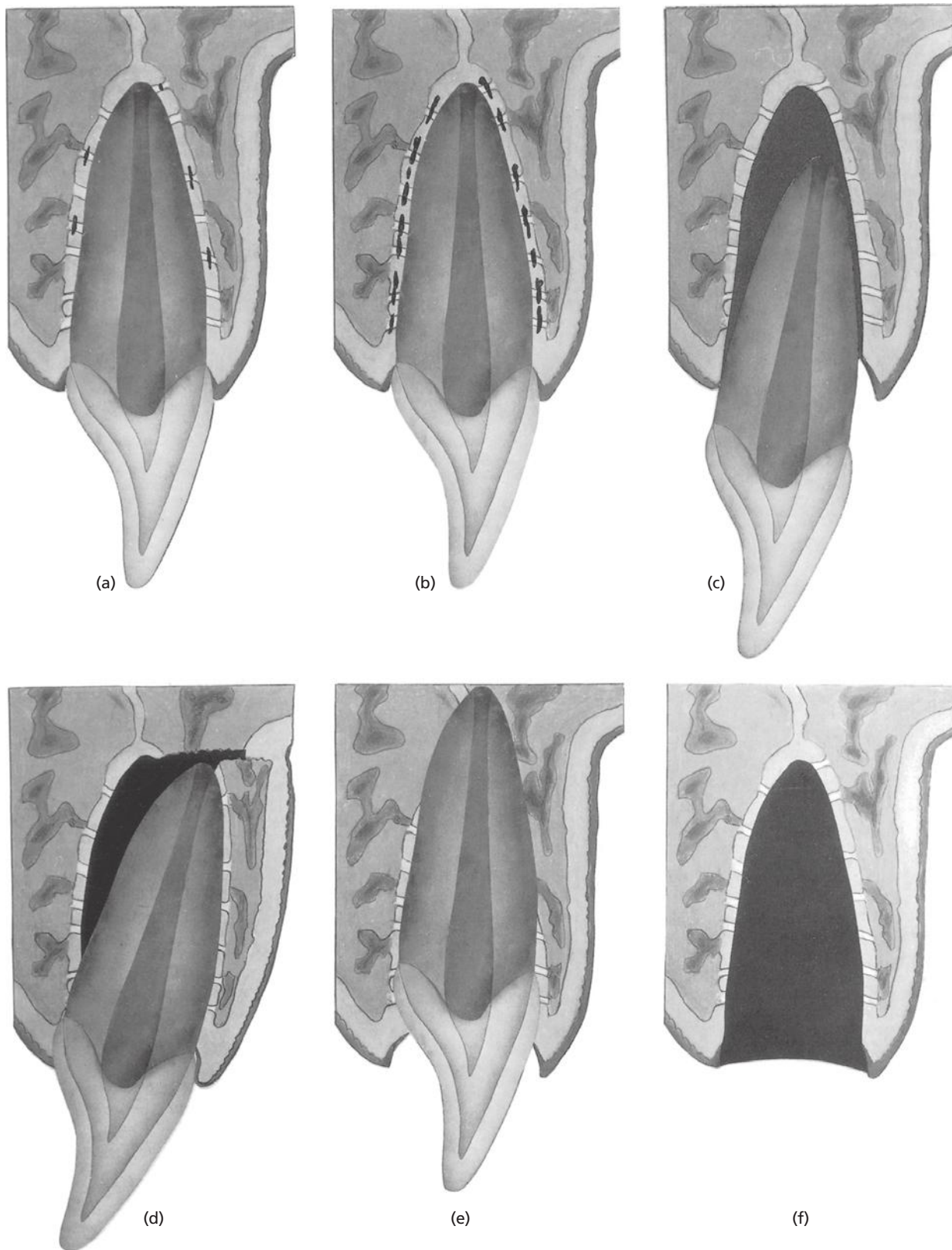


Fig. 39.14 Injuries to the periodontal tissues. (a) Concussion. (b) Subluxation. (c) Extrusive luxation. (d) Lateral luxation. (e) Intrusive luxation. (f) Avulsion. From Andreasen *et al.* 2007.²

tant to cover the exposed dentin with a thin layer of, for example, glass ionomer cement. Save the fragment by keeping it moist in saline.



Fig. 39.15 Avulsed right central and lateral incisor.

Complicated crown fracture

Exposed vital pulp

Open root apex

In young patients with immature, still developing roots it is very important to preserve pulp vitality by pulpotomy; this technique-sensitive procedure can be performed later, when optimal facilities are available. In the emergency phase pulp capping with calcium hydroxide can be performed.

Pulpotomy is performed by removal of 1–2 mm of the exposed pulp tissue below the exposure site. The pulp is removed using a sterile round diamond bur in a high-speed hand piece then irrigated with sterile saline. After hemostasis, $\text{Ca}(\text{OH})_2$ or white mineral trioxide aggregate (MTA) can be applied. A thin layer of glass ionomer is placed to achieve a tight seal. Composite restoration can be performed either in the emergency phase or later. If the crown fragment is available, bonding of the crown fragment to the tooth can be performed.



Fig. 39.16 (a) Crown fractured right central incisor. (b) Tooth fragment found. (c) Fragment bonded back to its original position using a dentin-bonding technique.

Closed root apex

In young patients when crown substance loss is not too extensive, pulp capping or pulpotomy can be performed. If there is severe loss of crown substance or if too much time has elapsed between the injury and treatment, pulp extirpation is carried out and $\text{Ca}(\text{OH})_2$ placed to save the tooth in the emergency phase. Root canal treatment is carried out later.

Necrotic pulp**Open root apex**

An apexification procedure is performed using $\text{Ca}(\text{OH})_2$ paste or a root-end closure procedure is performed using MTA. Follow-up is important.

Closed root apex

Canal debridement is carried out and $\text{Ca}(\text{OH})_2$ is applied; root canal treatment is completed within 2 weeks.

Crown-root fracture**Uncomplicated crown-root fracture**

Removal of the coronal fragment for careful inspection of the fracture level is necessary in order to take a final decision on whether restoration is possible for the remaining fragment. This can be done in the emergency phase. An alternative in the emergency phase, is to stabilize the coronal fragment to the tooth by bonding. The definitive treatment depends on the extent of the fracture.

To restore the tooth, gingivectomy, ostectomy, or orthodontic extrusion may later be required. Surgical extrusion is not recommended because of risk of devitalizing the pulp. If post space is required, elective root canal treatment should be carried out.

Complicated crown-root fracture

Removal of the coronal fragment for inspection of the fracture level is necessary. If there is a deep fracture or longitudinal fracture making the tooth non-restorable, extraction of the tooth has to be performed.

Pulp treatment**Closed root apex**

Pulp extirpation and $\text{Ca}(\text{OH})_2$ application is preferred in the emergency phase in teeth with completed root development. Orthodontic or surgical extrusion may later be required to enable restoration of the tooth.

Open root apex

Regardless of the size of the exposure a superficial pulp amputation and temporary filling is performed (see crown fracture). To restore the tooth later, gingivectomy, ostectomy, orthodontic extrusion, or surgical extrusion can be performed.

Root fracture

The displaced coronal fragment may be repositioned (Fig. 39.17). A radiograph should be taken to check for correct repositioning. The tooth is splinted for 4 weeks. If the fracture is close to the cervical third then the tooth should be stabilized for longer (up to 4 months). Root canal treatment is not performed in the emergency phase of the treatment, because pulp healing is possible, even in cases with discoloration of the coronal fragment. Signs of pulp necrosis are watched for at follow-up visits. If pulp necrosis develops then root canal treatment of the coronal segment is performed, as the apical fragment normally contains vital pulp tissue.

Concussion

No treatment is needed. However, the injury should be documented in the records for follow-up purposes or later insurance claims.

Subluxation

Splinting is generally not necessary but a flexible splint to stabilize the tooth for patient comfort can be used for up to 2 weeks.

Lateral luxation

The tooth is repositioned manually with a finger in the vestibule or with forceps to disengage it from its bony lock and gently reposition it into its original location (Fig. 39.18). The tooth is stabilized for 4 weeks using a flexible splint. The pulpal condition is monitored in the weeks after trauma. If the pulp becomes necrotic, root canal treatment is indicated to prevent root resorption. In immature, developing teeth, revascularization can be confirmed radiographically by evidence of continued root formation and possibly by positive sensibility testing.

Extrusive luxation (extrusion)

Extruded teeth should be manually repositioned in the tooth socket and stabilized for 2 weeks using a flexible splint.

Intrusive luxation (intrusion)**Teeth with incomplete root formation**

Spontaneous re-eruption is allowed to take place. If no movement is noted within 3 weeks, rapid orthodontic repositioning is recommended.

Teeth with completed root formation

The tooth should ideally be repositioned orthodontically as soon as possible (Fig. 39.19). Surgical reposi-

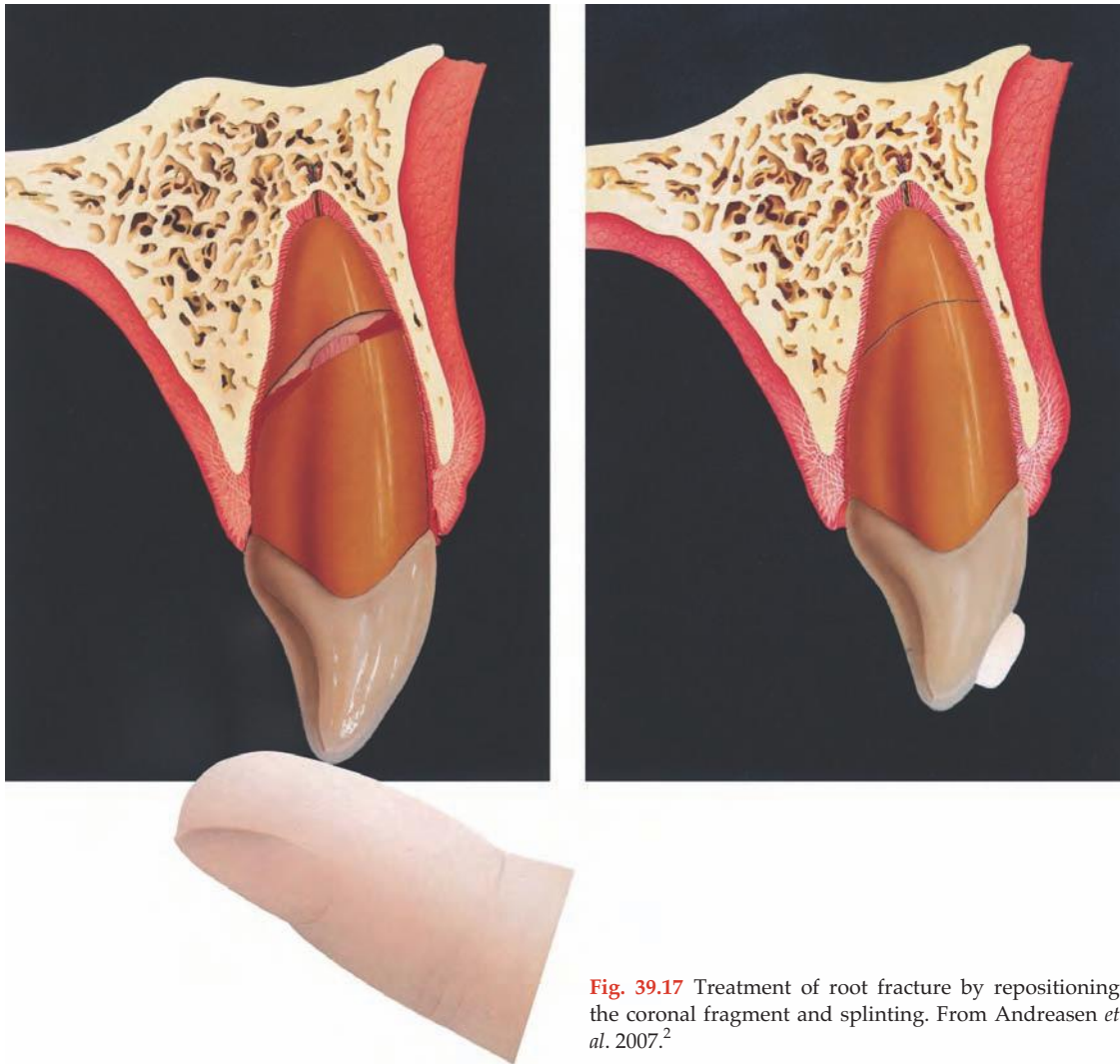


Fig. 39.17 Treatment of root fracture by repositioning the coronal fragment and splinting. From Andreasen *et al.* 2007.²

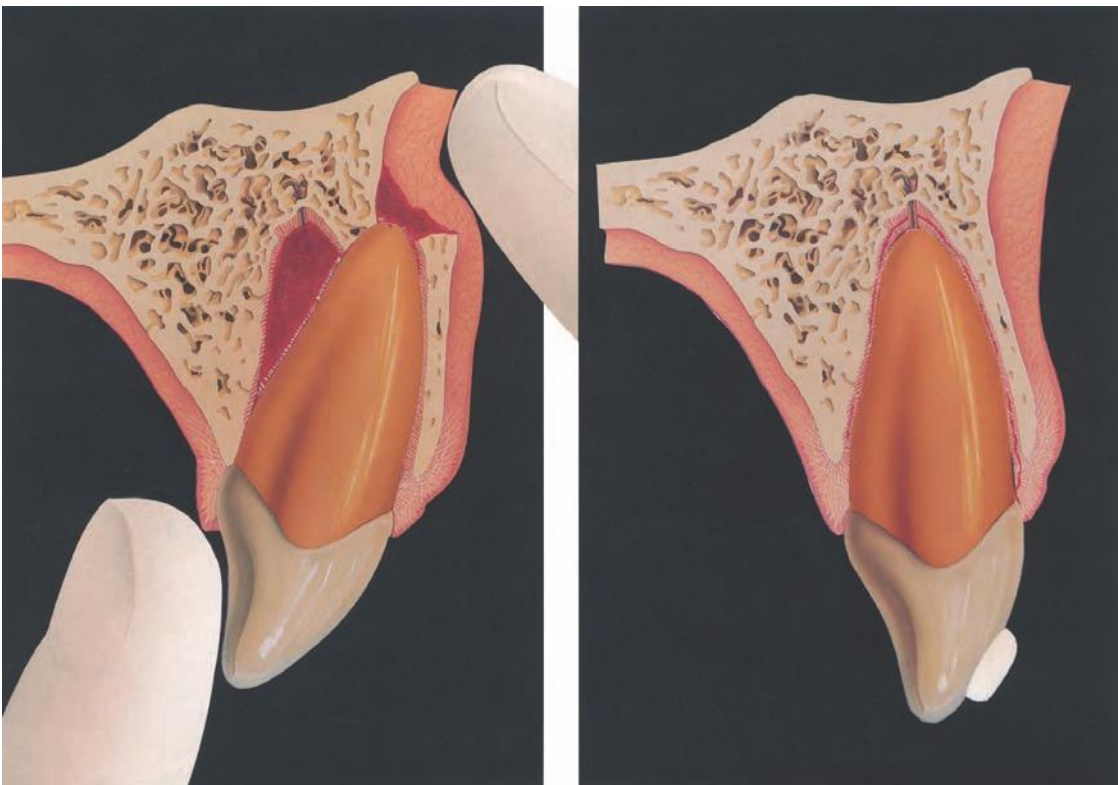


Fig. 39.18 Treatment principle for lateral luxation: repositioning and splinting. From Andreasen *et al.* 2007.²



Fig. 39.19 (a) Intruded left central incisor. (b) Orthodontic repositioning. (c) Final result after 2 months.

tioning in the emergency phase is an alternative treatment but has been associated with periodontal attachment loss. The pulp will remain necrotic in intruded teeth with completed root formation, and root canal treatment using a temporary filling with Ca(OH)_2 is recommended within the first weeks.

Avulsion

See Fig. 39.20.

Advice immediately after the accident

An avulsed permanent tooth is a critical injury since the prognosis depends very much on what is done immediately after the injury. Instructions may have to be given by telephone to the patient or other people at the place of accident, e.g. parents, teachers, etc. The following instructions should be given:

1. Make sure it is a permanent tooth. (Primary teeth should not be replanted because of the risk of injury to the underlying permanent tooth.)
2. Keep the patient calm.
3. Find the tooth and pick it up by the crown (the white part) and avoid touching the root.
4. If the tooth is dirty, wash it briefly (10 seconds) under cool running water and reposition it. Try to encourage the patient/parent to replant the tooth. Hold it in position.
5. If this is not possible, place the tooth in a suitable storage medium, e.g. a glass of milk or in saline.

The tooth can also be transported in the mouth, keeping it between the molars and the inside of the cheek. Avoid storage in water.

6. Seek emergency dental treatment immediately.

At the clinic

A patient may come to the clinic with the tooth already replanted or with the tooth outside the mouth stored in a medium. Treatment must be related to the status of the root development (closed or open apex) because there is a chance for revascularization of the pulp in teeth with open apices.^{26–29} Another factor of importance is the status of the periodontal ligament cells. If the tooth has been immediately replanted or replanted within the first hour or stored in a suitable storage medium, such as milk, saliva, or a special storage medium there is a chance for healing.^{26–29} However if the tooth has been stored dry for more than 1 hour the periodontal ligament cells will be necrotic, healing with normal periodontal ligament cannot be expected, and the tooth will be subjected to later root resorption.^{11–14} Such teeth should still, in the majority of cases, be replanted in the emergency phase and final decisions on more definitive treatment should be taken at a later stage when all factors can be taken into consideration, such as tooth development, Angle class, congenitally missing teeth, growth status of the patient, etc. Such comprehensive planning will require a multidisciplinary approach together with other specialists, e.g. endodontists, orthodontists, prosthodontists, and pediatric dentists.

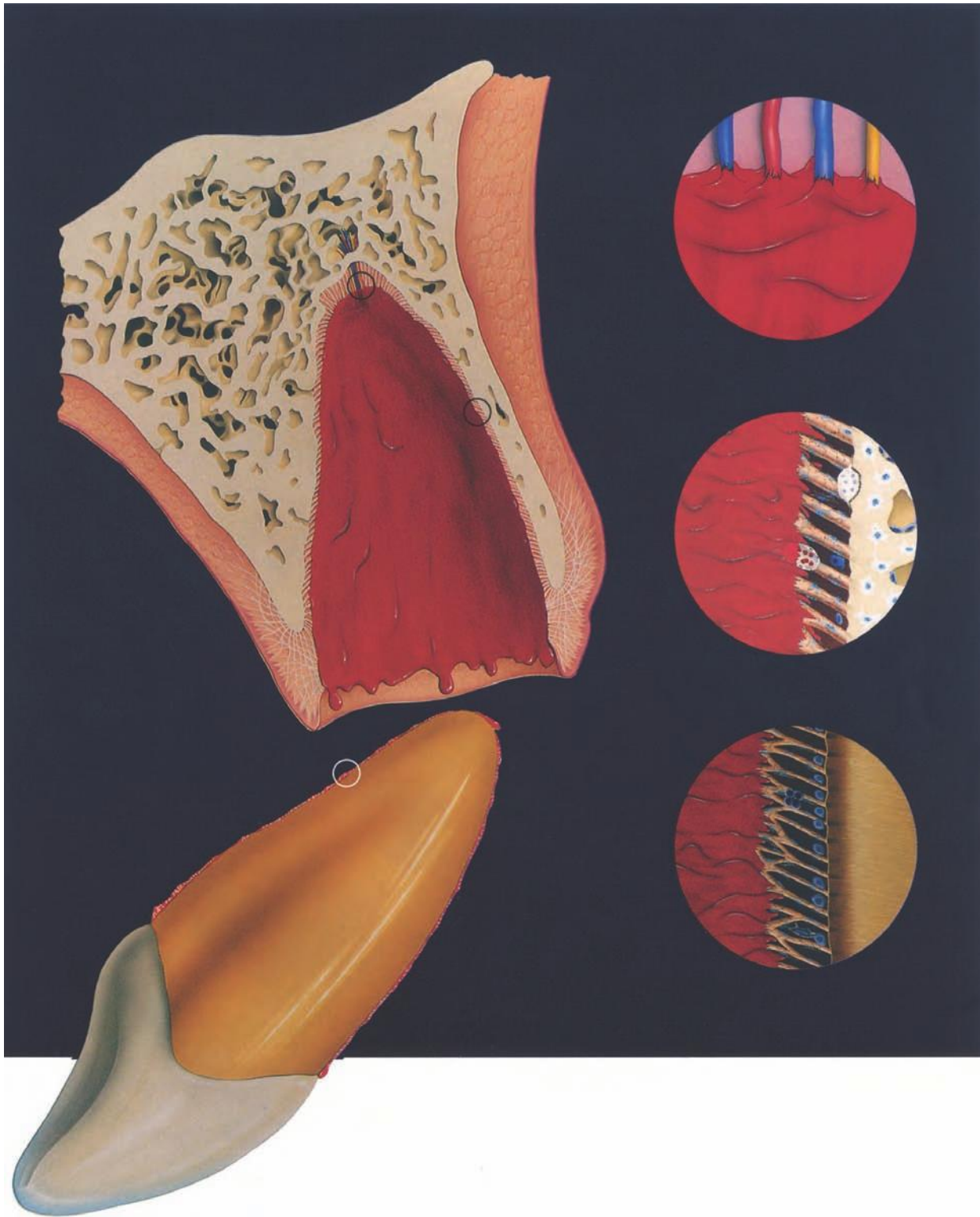


Fig. 39.20 Mechanism of avulsion. Frontal impacts lead to avulsion with subsequent damage to both the pulp and periodontal ligament (PDL). The vascular supply of the pulp is interrupted, the PDL attaching to the alveolar socket and the root surface is damaged. The extraoral time and environment determine the fate of the PDL and pulp after replantation. From Andreasen *et al.* 2007.²

In the emergency phase the following treatment should be carried out.

Avulsed permanent tooth with closed apex

The tooth has been replanted prior to the patient arriving at the dental office or clinic:

1. Do not extract the tooth.
2. Clean the area with water spray, saline, or chlorhexidine.
3. Suture gingival lacerations if present.
4. Verify normal position of the replanted tooth both clinically and radiographically.
5. Apply a flexible splint for up to 2 weeks.
6. Administer systemic antibiotics.
7. Evaluate the need for a tetanus booster.

8. Initiate root canal treatment 7–10 days after replantation and before splint removal by placing calcium hydroxide as an intracanal medicament until permanent filling of the root canal.

The tooth has been kept in special storage media, milk, saline, or saliva, and the extraoral dry time is less than 60 minutes:

1. If contaminated, clean the root surface and apical foramen with a stream of saline and place the tooth in saline. Remove the coagulum from the socket with a stream of saline before replantation.
2. Examine the alveolar socket. If there is a fracture of the socket wall, reposition it with a suitable instrument. Replant the tooth slowly with slight digital pressure. Suture gingival lacerations.
3. Verify normal position of the replanted tooth both clinically and radiographically.
4. Apply a flexible splint for up to 2 weeks.
5. Administer systemic antibiotics.
6. Evaluate the need for a tetanus booster.
7. Initiate root canal treatment 7–10 days after replantation and before splint removal by placing calcium hydroxide as an intracanal medicament until permanent filling of the root canal.

If the extraoral dry time is longer than 60 minutes, delayed replantation is carried out. The aim of delayed replantation is not to achieve normal periodontal healing. The expected final outcome is ankylosis and resorption of the root. However the retained root will prevent the alveolar process from atrophying. Fluoride has been reported to delay the rate of replacement resorption of the retained root.

The technique for delayed replantation is:

1. Remove attached necrotic soft tissue or the root with gauze. Root canal treatment can be done on the tooth prior to replantation through the open apex or during the first weeks after replantation.
2. Remove the coagulum from the socket with a stream of saline.
3. Examine the alveolar socket. If there is a fracture of the socket wall, reposition it with a suitable instrument.
4. Immerse the tooth in a 2% sodium fluoride solution for 20 minutes.
5. Replant the tooth slowly with slight digital pressure. Suture gingival lacerations.
6. Verify normal position of the replanted tooth clinically and radiographically.
7. Splint the tooth for 4 weeks.
8. Administer systemic antibiotics.
9. Evaluate the need for a tetanus booster.

During follow-up, ankylosis and replacement resorption are expected in the first year. If there is infraposition of the tooth crown of more than 1 mm, it is recommended to perform decoronation to preserve

the contour of the alveolar ridge. This procedure should be reserved for patients with remaining vertical growth.^{30–33}

Avulsed permanent tooth with open apex

The tooth has already been replanted prior to the patient arriving in the dental office or clinic:

1. Clean the area with water spray, saline, or chlorhexidine.
2. Do not extract the tooth. Suture gingival lacerations if present.
3. Verify normal position of the replanted tooth both clinically and radiographically.
4. Apply a flexible splint for up to 2 weeks.
5. Administer systemic antibiotics.
6. Evaluate the need for a tetanus booster.
7. If revascularization of the tooth pulp does not occur within the first months, root canal treatment may be recommended.

The tooth has been kept in special storage media, milk, saline, or saliva, and the extraoral dry time is less than 60 minutes:

1. If contaminated, clean the root surface and apical foramen with a stream of saline. Topical antibiotics can be considered.
2. Remove the coagulum from the socket with a stream of saline and then replant the tooth.
3. If there is a fracture of the socket wall, reposition it with a suitable instrument. Replant the tooth slowly with slight digital pressure.
4. Suture gingival lacerations, especially in the cervical area.
5. Verify normal position of the replanted tooth clinically and radiographically.
6. Apply a flexible splint for up to 2 weeks.
7. Administer systemic antibiotics.
8. Evaluate the need for a tetanus booster.
9. If revascularization of the tooth pulp does not occur within the first months, root canal treatment may be recommended.

If the extraoral dry time is longer than 60 minutes, delayed replantation is carried out. The aim of delayed replantation is not to achieve normal periodontal healing. The final outcome is ankylosis and resorption of the root. However the retained root will prevent the alveolar process from undergoing severe atrophy. Fluoride has been reported to delay the rate of replacement resorption of the retained root.

The technique for delayed replantation is:

1. Remove attached necrotic soft tissue with gauze. Root canal treatment can be done on the tooth prior to replantation through the open apex or the first weeks after replantation.
2. Remove the coagulum from the socket with a stream of saline.

3. Examine the alveolar socket. If there is a fracture of the socket wall, reposition it with a suitable instrument.
4. Immerse the tooth in a 2% sodium fluoride solution for 20 minutes.
5. Replant the tooth slowly with slight digital pressure. Suture gingival lacerations.
6. Verify normal position of the replanted tooth clinically and radiographically.
7. Splint the tooth for 4 weeks.
8. Administer systemic antibiotics.
9. Evaluate the need for a tetanus booster.

During follow-up, ankylosis and replacement resorption are expected in the first year. If there is infraposition of the tooth crown of more than 1 mm and the patient is still in a growth active period, it is recommended to perform a decoronation procedure to preserve the contour of the alveolar ridge.^{30–33}

Splinting of TDI

After emergency treatment of TDI where teeth and root fragments have been repositioned, there is a need for splinting to stabilize the injured tooth in the initial healing period. However splinting may also have adverse effects on healing, such as increased risk for ankylosis and osseous replacement resorption or compromised revascularization of the pulp if splinting is not done well. Bars used for maxillomandibular fixation in jaw fracture treatment are not suitable for splinting of luxated or replanted teeth

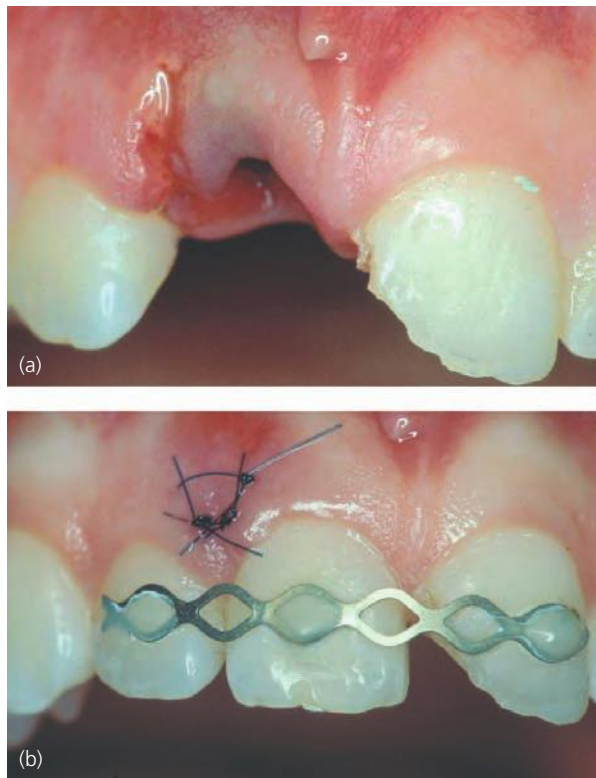


Fig. 39.21 Titanium trauma splint. (Courtesy of Dr von Arx, Berne, Switzerland.)

because there is a risk for extrusion of teeth due to the fact that the preformed bar does not have any occlusal curve built in. Hence, the bar will extrude the luxated teeth.

Since the discovery of adhesive techniques, dental splints have been made by an acid-etched bonding technique which fulfills most requirements of a modern splint. An optimal splint should enable some micromovements of the injured tooth, allowing it to move a little bit during the healing phase. This can be achieved either by the splint being flexible, by combining the bonding material with a wire to allow for flexibility, or by not stretching out the splint too far.

The bonding material can be combined with a steel wire, twist flex wire, nylon fishing line, fiberglass or titanium splint. Some examples of splints can be seen in Figs 39.21–39.23. The splinting period should ideally be related to the type of TDI. The periods shown in Table 39.1 have been recommended by an expert group of IADT.^{18,19,34}

TDI in the primary dentition

Similar diagnosis and classification principles are applied to primary teeth. However the treatment of primary teeth is often modified and consideration must always be given to the underlying permanent tooth germs. For this reason primary teeth must never be replanted. Moreover, a primary tooth that has been dislocated in the direction of the permanent underlying tooth germ should be extracted, while dislocation away from the underlying tooth germ may allow the primary tooth to be repositioned and

Table 39.1 Splinting periods for various TDI requiring fixation.

TDI	Splinting period
Subluxation	(2 weeks)
Extrusive luxation	2 weeks
Avulsion	2 weeks
Lateral luxation	4 weeks
Root fracture (middle third)	4 weeks
Root fracture (cervical third)	4 months

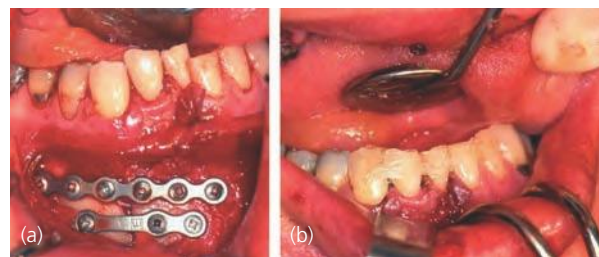


Fig. 39.22 Patient with mandibular body fracture and luxated incisors. (a) The mandibular fracture has been splinted by plate and screw osteosynthesis. (b) A separate fiberglass splint has been applied to the luxated and repositioned teeth. The use of separate splints will enable optimized splinting time and type.

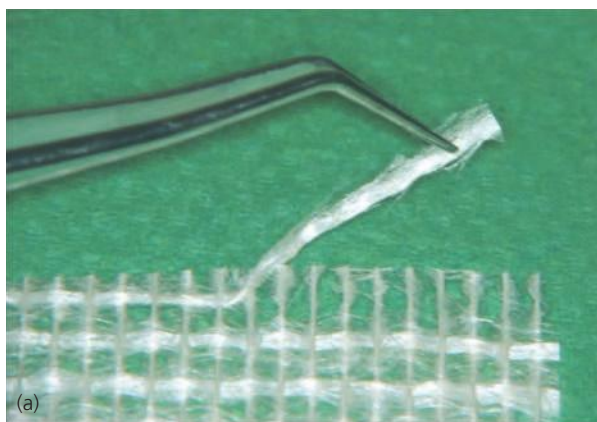


Fig. 39.23 Splinting using fiberglass and resin. (a) A string of fiberglass. (b) The fiberglass is bonded to the acid-etched enamel of the luxated tooth and the adjacent teeth. (c) Finished splint.



kept. A primary tooth should not be kept if it poses a risk for the permanent tooth because of infection. Other modifying factors when treating traumatized primary teeth are maturity of the child, caries, tooth development, occlusion, and time for exfoliation. For more detailed information regarding treatment of primary teeth after trauma see textbooks and manuals^{2,16} or recent guidelines for primary teeth.³⁵

Complications

Pulpal complications

The pulpal complications that commonly occur after dental trauma can be categorized as pulp canal obliteration (PCO), pulp necrosis, and internal root resorption. The incidence of such complications varies among traumatized teeth, depending on the type of the trauma and how it was managed.

Pulp canal obliteration

Partial or complete obliteration of the pulp canal is characterized by deposition of hard tissue within the root canal space and yellow discoloration of the clinical crown. This can be recognized clinically as early as 3 months after the injury. PCO is seen commonly after luxation injuries and horizontal root fractures in young adults. On clinical examination the tooth appears darker in hue than the adjacent teeth and exhibits a dark yellow color due to decrease in trans-

lucency from a greater thickness of dentin under the enamel.

Approximately 3.8–24% of traumatized teeth develop varying degrees of PCO. Studies indicate that the yearly incidence of pulpal necrosis occurring in these teeth is <1%. A tooth with PCO does not need root canal treatment unless there is evidence of periradicular radiolucency or the tooth becomes symptomatic. If the pulp tissue becomes necrotic and a periradicular radiolucency develops, non-surgical root canal treatment has been shown to be successful in 80% of cases.

Pulp necrosis

This is a common sequel of traumatic injuries; the incidence increases with the severity of the injury and in cases where the roots are fully formed. Pulp necrosis also depends on the type of injury. If pulp necrosis is detected then root canal treatment should be initiated immediately to prevent inflammatory root resorption.

Internal root resorption

This process of root resorption is usually detected radiographically. The radiographic appearance is typically a radiolucent lesion occurring in the middle of the root with loss of the outline of the pulp canal space. The presence of vital pulp tissue is a prerequisite for this to occur and usually it is asymptomatic. If detected early then root canal therapy should be initi-

ated and the prognosis can be excellent. If the resorptive defect is large, rendering the tooth compromised, then the risk of root fracture is increased. Follow-up radiographs are an essential means of detection and management.

Periodontal complications

When luxation injuries occur, the periodontal membrane is torn and damaged against the bone walls. This membrane can also be injured as its cells become exposed to drying or to a non-physiological environment. Provided infection does not intervene, healing of small injured areas may take place spontaneously. Two complications that may occur are inflammatory root resorption and dentoalveolar ankylosis.

Infection-related (inflammatory root) resorption

If the pulp is infected in a tooth that has been exposed to injuries to the root cementum and periodontal membrane then serious complications may occur. Inflammation on the root surface may be maintained by the necrotic infected pulp through osteoclastic exposure of dentinal tubules. If the necrotic pulp is not removed the tooth will be quickly resorbed. Infection-related resorption can be detected on radiographs as radiolucent root resorption cavities. Infection-related root resorption can be treated by debridement of the canal space and application of $\text{Ca}(\text{OH})_2$ dressing. Because some dental injuries have an extremely high risk of Infection-related root resorption, the pulp should always be extirpated



Fig. 39.24 Radiograph of an ankylosed incisor. Root resorption with osseous replacement is seen.

when treating such dental injuries; in other injuries revascularization of the pulp and healing may occur.

Dentoalveolar ankylosis

If large areas of the periodontal membrane have been injured, either by trauma itself or as a result of Infection-related root resorption, the remaining periodontal membrane cells cannot produce a new periodontal membrane. Instead, bone will grow into these defective areas and the tooth will be united with the alveolar bone, resulting in dentoalveolar ankylosis. This is a serious condition for the tooth in that the tooth will be replaced by bone and will be eventually lost. Radiographically bone is seen replacing the tooth substance (Fig. 39.24). This process is called replacement resorption or osseous replacement. In adults this replacement resorption will proceed slowly and the tooth may stay in place for several years. However in young individuals, where growth is not yet completed, the replacement is much faster and furthermore, dentoalveolar ankylosis will interfere with the growth of the alveolar process and the tooth will be infrapositioned (Fig. 39.25). It is therefore important to diagnose dentoalveolar ankylosis early. A high metallic percussion sound can be heard when a percussion test of the tooth is per-



Fig. 39.25 Infraposition of left central incisor as a result of ankylosis after trauma. The tooth had a high percussion sound.



Fig. 39.26 Decoronation of ankylosed incisor. The root is left in the bone for osseous replacement, keeping the width of the alveolar process.

formed. If ankylosis is diagnosed in a growing patient decoronation may be necessary and a multidisciplinary approach is recommended for treatment planning and coordination with the patient's growth status and dental development.^{30–33}

Summary

Oral and maxillofacial surgeons will regularly see many trauma patients. TDI occur frequently and are often seen simultaneously with other oral and maxillofacial injuries. The prognosis for some TDI is highly dependent on prompt and correct emergency management. Hence the oral and maxillofacial surgeon should have knowledge to manage dental injuries in the emergency phase. This chapter has presented the basic knowledge needed for such management.

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Chapter 40

Midfacial Fractures

Petr Schütz and Lars Andersson

The maxillofacial region is important, with vital functions such as breathing, eating, talking, smelling, and vision. Moreover, it is also an esthetically important region. Injuries to the maxillofacial region may, therefore, have serious consequences for the individual's quality of life.

Earlier used methods, of conservative treatment of maxillofacial fractures with suspension wires and intermaxillary fixation, have been replaced by methods aiming at exposing fracture lines, allowing precise alignment and application of osteosynthesis hardware and enabling a faster return to function. The aim of this chapter is to give an overview of modern treatment principles in the management of fractures of the midface by describing surgical approaches, techniques, and strategies for fracture treatment. A section on biodegradable osteosynthesis material is also presented.

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Midfacial skeleton as a three-dimensional structure

The midface skeleton (Fig. 40.1) is a complex structure composed of multiple, mostly paired, bones intimately linked by sutures. It is connected to and supported by bones of the neurocranium, namely frontal bone, sphenoid bone, and temporal bone. In individuals with preserved dentition, further support is provided by mandible via contact of the dental arches.

The midface can be viewed as a labyrinth of air-containing cavities, with the exception of the orbits, surrounded by thin bony lamellae with rims of thick bone (Fig. 40.2). These thicker rims constitute a system of pillars and struts resembling the framework of a building, and the position and stability of each is interrelated.¹ The midface buttress system comprises

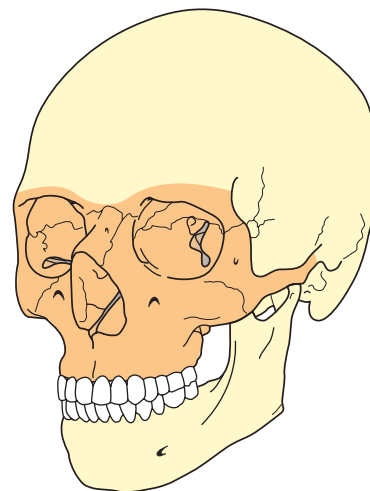


Fig. 40.1 Midfacial area highlighted.



Fig. 40.2 The midface as a system of cavities: O, orbits; E, ethmoidal sinuses; M, maxillary sinuses; N, nasal cavities.

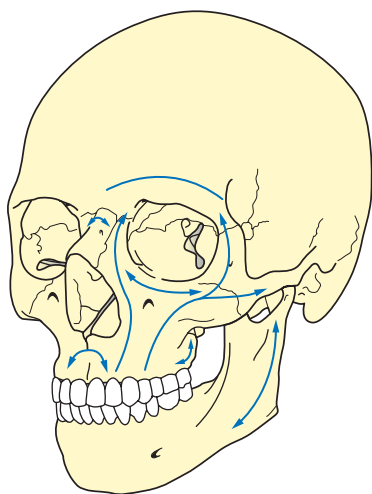


Fig. 40.3 Projection of masticatory forces through the vertical pillars of the midface.

vertical, horizontal, and sagittal components, of which the sagittal component is the weakest.²

The well defined, paired **vertical buttresses** (Fig. 40.3) are: (1) nasomaxillary (made up of the frontal process of the maxilla); (2) zygomatico-maxillary (made up of the zygomatico-alveolar crest, body and frontal process of the zygoma); and (3) pterygomaxillary (made up of the pterygomaxillary junction and the pterygoid process of the sphenoid bone). These buttresses developed as an adaptation to masticatory forces that are transmitted by them to the skull base. The **horizontal buttresses** include the superior and inferior orbital rims, maxillary alveolus and palate, serrated edges of the greater wings of the sphenoid bone and the zygomatic arches.³ The **sagittal buttresses** are represented by zygomatic arches, while the central part of the midface lacks a strong sagittal buttress (Fig. 40.3).²

The main objective of fracture repair of the midface is the reconstruction of these buttresses using osteosynthetic devices. These buttresses are robust enough bony structures to allow secure insertion of screws. The exceptions are the pterygomaxillary buttresses and the sphenoid greater wings, which are not surgically accessible.

The ratio between the small bone volume of the midface and its large surface area provides an excellent blood supply despite the absence of large nutritive vessels. Consequently, fractures of the midface heal and remodel fast and well and are resistant to infection. On the other hand this unique healing capacity can also complicate management of fractures if there is a delay in treatment.

Classification

There are several systems for classification of midface fractures. Fractures can comprise single or multiple fracture lines in the same bone or have fracture lines that communicate with each other, so-called comminuted fractures. Fractures can also be of the greenstick type which is often seen in young children where the bone is not so highly mineralized.

Fractures can also be classified as closed, when there is no communication with the outer environment through lacerations in the skin, oral mucosa, and gingiva, or through a gingival pocket. An open fracture is defined as a fracture where there is such a communication through a laceration or when the fracture is engaging a dentate area. A fracture can also be complicated if there is a considerable injury or defect in the overlying soft tissue.

Fractures can also be classified in relation to the first or secondary place of energy release. A direct fracture occurs where the first impact takes place, e.g. a blow to the cheek resulting in a zygomatico-maxillary fracture, while an indirect fracture happens in the region of the zygomatic arch and possibly also the skull base.

Orbital fractures are often classified as blow in or blow out, depending on the way the orbital walls are fractured. When most of the maxillofacial bones are engaged in a trauma the term panfacial fracture is used. From a clinical and treatment point of view, classification using the above terminology in combination with a classification related to the anatomic site of the midface fracture is most often used.

René Le Fort's (French surgeon, 1869–1951) experimentally based categorization of midfacial fracture patterns is the most popular one.⁴ This simple classification system distinguishes three fracture patterns (Fig. 40.4):

- Le Fort I (also known as Guérin fracture,⁵ after Alphonse Guérin, French surgeon, 1816–1895), separates the whole complex of alveolar and palatal processes of the maxilla, horizontal plates of the

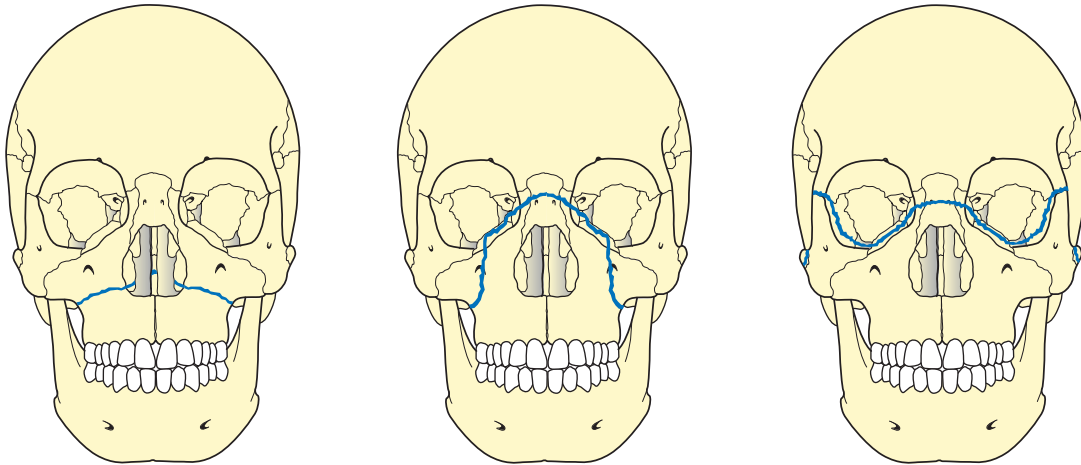


Fig. 40.4 Le Fort fracture patterns.

palatal bones and lower parts of the pterygoid plates just above the pterygo-maxillary junction;

- Le Fort II (also known as "pyramidal" fracture) separates the whole maxilla with part of the nasal bones and the lower part of the pterygoid plates;
- Le Fort III separates both zygomatico-maxillary complexes plus the nasal bones, palatal bones and most of the pterygoid plates, from the rest of the cranium.

However, in clinical practice Le Fort types of fractures are very rarely encountered in pure forms as described above. In most instances the fracture lines of particular types combine in quite unpredictable, often

asymmetric patterns (Fig. 40.5). The reason for this variability rests with the many different trauma mechanisms and high energies involved, which were not predicted and were perhaps unimaginable in the time when René Le Fort performed his experiments.

There are other, very common and historically recognized fracture patterns that do not fit into the Le Fort classification. Prominent among them is a fracture of the zygomatic bone, first reported by Joseph Duverney (French anatomist, 1648–1730).⁶ This is currently described as zygomatico-maxillary complex (ZMC) fracture,⁷ or zygomatico-orbital fracture,⁸ because the zygomatic bone is almost never involved alone, as it is in intimate contact with the temporal, frontal, sphenoid, and maxillary bones (Fig. 40.6). An isolated fracture of the zygomatic arch is a separate clinical entity.

Nasal bone fractures are given little attention in maxillofacial literature, perhaps because they are mostly isolated and managed by ear, nose, and throat (ENT) surgeons. Despite that, they are the most frequent facial fractures in general.^{9–12}

Another clinically useful concept is the one of naso-orbito-ethmoid (NOE) fracture.¹³ It reflects the

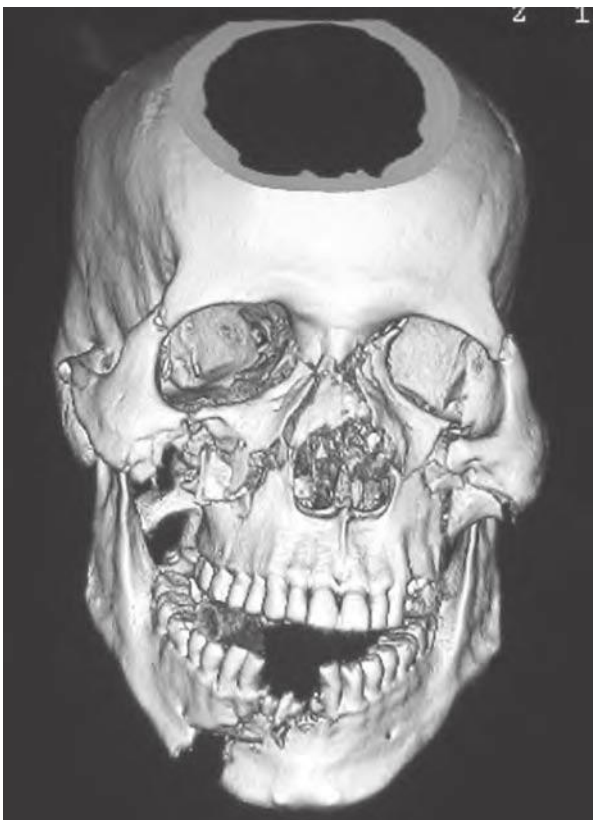


Fig. 40.5 3D CT of panfacial fracture with patterns of all three Le Fort fractures recognizable.

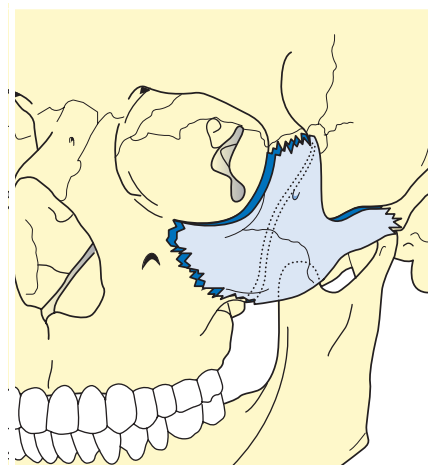


Fig. 40.6 Schematic drawing of zygomatico-maxillary complex fracture.

fact that high-energy injuries to the nasal area break and displace bony structures involving the nasal bones, and also the frontal processes of the maxilla, the lacrimal bones and the ethmoid bone (Fig. 40.7). A very similar term is frontonasal fracture, which describes the concomitant involvement of the nasal part of the frontal bone, and especially of the fronto-nasal ostium.¹⁴

The diagnosis of the sagittal palatal fracture is important (Fig. 40.8), although it always occurs in combination with other fractures, often of Le Fort type.¹⁵

The term orbital fracture¹⁶ can mean any fracture involving either the orbital rim or the orbital wall. In clinical practice it is usually understood to be a fracture of the orbital walls.

It is obvious that above-mentioned terms and definitions are useful in the description of topographically limited injuries, but when it comes to complex midfacial fractures, a list of separate diagnoses is usually developed. These diagnoses often overlap when adjacent areas of the midface are affected. Moreover, complex fracture patterns can be described in more than one way. This makes communication between clinicians difficult and can have insurance, billing and medico-legal repercussions. On the other hand to describe each fracture line separately would be too intricate, laborious, and perplexing. To overcome this

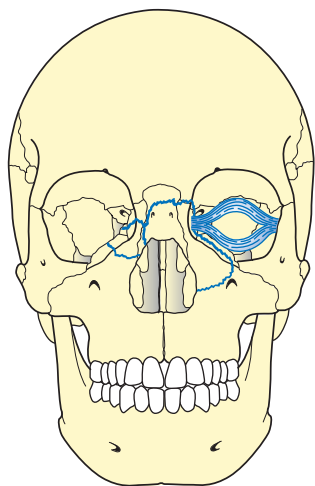


Fig. 40.7 Schematic drawing of naso-orbito-ethmoidal fracture.

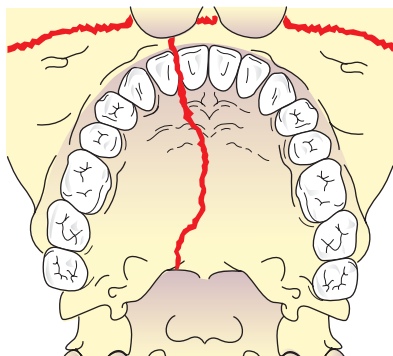


Fig. 40.8 Schematic drawing of parasagittal fracture of the hard palate.

problem, there have been many attempts to develop comprehensive descriptive and scoring systems which are mentioned here briefly for completeness.^{17–21} The system of Cooter and David¹⁷ is alpha-numerical coding of skull fractures, coding fracture sites alphabetically and their severity numerically. A similar elaborate system, more suitable for research than for clinical use, providing detailed analysis of fracture pattern and severity, was developed by Buitrago-Téllez *et al.*¹⁸ The system devised by Guerrissi¹⁹ aims at quantification of severity of maxillofacial injuries for the purpose of evaluation of esthetic and functional impact as well as identification of patients with potentially life-threatening injuries. This system unfortunately brings together bone and soft tissue injuries, which makes interpretation difficult. Donat *et al.*²⁰ base their classification on the effect on skeletal supportive beams and buttresses. Follmar *et al.*²¹ offer a hierarchical system integrating classical anatomic nomenclature and established fracture patterns, avoiding descriptive duplicity.

From a clinical point of view it is practical to use the classification system in Table 40.1.

Epidemiology

The peak incidence of maxillofacial fractures is in the age range of 20–30 years.^{22–30} The incidence is higher in males.^{22–31} The etiological factors vary with societies and geographic location in the world.^{22–51} In developing countries road traffic accident is the main cause of maxillofacial injuries; in more developed countries there has been a decrease in the proportion of road traffic accidents as etiologic factors, especially in the past three decades, due to a more developed and safer traffic environment and the use of seat belts.^{23–25,27–31,37} Many studies have reported that a life style with high alcohol consumption is a major contributing factor.^{22–25,27,30,40,44,47,48,53–60} In contrast there are other societies in the world where alcohol consumption does not play a major role in the etiology of facial fractures because it is forbidden due to religious and cultural reasons.^{25,26,29,34,35,39,41}

Table 40.1 Classification of midface fractures with regards to anatomic location and clinical relevance.

Frontal bone
Zygomatic arch
Zygomatico-maxillary complex
Orbit
Nasal
Naso-orbito-ethmoidal fractures
Le Fort I, Le Fort II, Le Fort III
Maxillary sinus walls
Maxillary alveolar process
Palate

Maxillofacial injuries due to sports are frequently seen; the etiology varies as different sports are played in different geographic locations.^{23,61–70} The use of helmets and mouth guards can prevent some injuries. In developing countries, fall injuries are more common due to lack of safety measures.^{23,26,28,49,56}

Throughout history there have been maxillofacial injuries caused by weapons. The head and neck region is affected in 16% of all war injuries.⁷¹ Weapons have developed from low velocity to high velocity, and in recent years blast injuries from landmines and roadside bombs have become more common.⁷² In some societies, where there is easy access to weapons, gunshot injuries occur frequently during peace time, and maxillofacial injuries are sustained due to gunshot injuries caused by crimes and suicide attempts.⁷³

Assessment of patients with midface injuries

Assessment of the injured maxillofacial patient is presented in detail in Chapter 38. It is important to take a thorough history. Since many patients are unconscious there are often only reports from eyewitnesses. Such reports are not always reliable.

Patients with midface fractures are often examined and treated with a multidisciplinary approach. Responsibilities and routines vary with traditions worldwide. In recent decades the oral and maxillofacial surgeon has taken an increasing responsibility for midface injuries and today is often the leader of the team. Ophthalmologists are consulted for eyeball trauma, blindness, and problems regarding vision, ENT specialists have to be consulted for vertigo and hearing problems related to trauma, and neurosurgeons must be consulted when there is cerebrospinal fluid leakage and when intracranial bleeding or air is suspected. Plastic surgeons are consulted when there are large soft tissue defects.

Open or closed reduction

In contrast to the mandible where some fractures can still be treated non-surgically with intermaxillary fixation in a cooperative patient, the majority of midface fractures are treated surgically. In the past, suspension wiring techniques, aimed at stabilizing the fractured bone to a more superiorly located stable non-fractured bone, were used. However, it was difficult to achieve and maintain proper reduction with these techniques.

In recent decades bone plates and screws have been developed. These are mostly made of titanium. The principle is to expose, reduce, and stabilize the fractures by rigid fixation using the plates and screws. Treatment should ideally be carried out in the emergency phase within the first 24 hours, and if these conditions cannot be met, treatment should be

planned within the first week. Longer delay may result in difficulties in repositioning the fragments.

Surgical approaches to midfacial skeleton

The aim of surgical approaches to midfacial fractures is to enable wide subperiosteal exposure of all fracture lines, precise alignment, and application of osteosynthesis hardware. This should be achieved with minimal additional morbidity and minimal permanent consequences.⁷⁴ Special considerations are given to cosmetic effect of resulting scars, preservation of function of branches of facial and trigeminal nerves, and avoidance of major vessels during surgery. These goals are best met by an intraoral approach.

While the whole mandible can, in principle, be approached surgically from intraoral incisions, sometimes with assistance of an endoscopic technique when operating on condyles, the intraoral approach offers exposure of midfacial skeleton limited to the anterior and inferior parts of the maxilla and palate. Everything above the level of the infraorbital rims must be accessed through the skin or trans-conjunctival incisions.

Intraoral incisions

Intraoral incisions in management of midfacial fractures are usually limited to the upper vestibule and alveolar process. The palatal mucosa must be incised only very rarely, to enable plating of a sagittal palatal fracture or to facilitate the suture of an associated laceration.

Disinfection of the mucosal surface before incision is not necessary. However in some trauma patients, oral hygiene is compromised and the mouth should be copiously irrigated with saline and mechanically cleaned with moist gauze if necessary. Infiltration of the submucosa with local anaesthetic with vasoconstrictor will reduce the initial bleeding and make dissection fast and comfortable.

The upper vestibular incision starts at the level of the first molar about 1 cm above the attached gingiva and proceeds anteriorly, sloping slightly caudally, so that it leaves a cuff of about 5 mm of loose mucosa at the upper labial frenum. An incision placed too high posteriorly will violate the attachment of the buccinator muscle and cause the buccal fat to enter into the operation field. The incision is usually accomplished in two steps, firstly cutting mucosa and then periosteum at the base of the alveolar process. Dissection then proceeds subperiosteally. It can reach the inferior orbital margin and the body of the zygomatic bone. The periosteum can be undermined over the maxillary tuber up to the pterygomaxillary suture. The infraorbital neurovascular bundle should always be identified and protected (Fig. 40.9). The margin of the

piriform aperture should be identified medially and the nasal mucosa protected.

In the presence of gingival and mucosal lacerations, these should be incorporated into the incision if feasible. This means that sometimes a marginal mucoperiosteal flap will be created to avoid ischemia of lacerated superiorly pedicled flaps.

Closure of a buccal sulcus incision is usually accomplished in one layer with either continuous or interrupted mattress sutures. Sufficiently deep bites of tissue should be taken to prevent dehiscence. In the case of bilateral incision (horseshoe-shaped incision), consideration should be given to using the nasal alar base cinch suture and V-Y plasty of upper lip mucosa to prevent widening of the nose and shortening of the upper lip.

Displaced fractures of the hard palate are sometimes accompanied by laceration of the mucoperiosteum (Figs 40.10 and 40.11). Some undermining of margins and extension may be required to enable application of a fixation device. Suturing can be, however, difficult, especially if mucosal margins are crushed or defective. In such a situation, parallel releasing incisions and undermining, creating bipediced flaps, can help, as in cleft palate surgery.

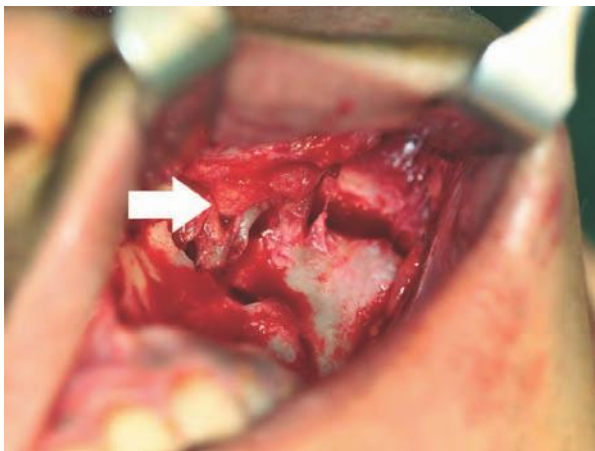


Fig. 40.9 Fractured zygomatico-maxillary complex exposed from upper vestibular incision. White arrow indicates the infra-orbital nerve.



Fig. 40.10 Parasagittal displaced fracture of the maxilla with laceration of hard palate mucosa.

Midfacial degloving by intraoral incision

Midfacial degloving is an extension of a horseshoe-shaped intraoral incision achieved by releasing the skeletal attachment of the nose and retraction of its soft tissue envelope cranially, so that the inferior and medial orbital margins and nasal bones are exposed. This approach is seldom used in management of acute trauma, but can be advantageously used in secondary reconstruction of the naso-orbito-ethmoidal (NOE) and frontonasal area.⁷⁵ In these cases it can replace the much more time-consuming coronal incision.

Periorbital incisions

The sheer number of periorbital incisions in use shows that none of them is applicable in all clinical situations. Some of them are fraught with a high percentage of cosmetically unfavorable outcomes and are currently criticized or even have already been abandoned. Among widely discarded approaches are prominent infraorbital and lateral eyebrow incisions. The subciliary incision may possibly also be abandoned in the future. The discussion in the literature currently concentrates on the following controversies: sub tarsal incision⁷⁶ versus transconjunctival incision⁷⁷ and transconjunctival incision with⁷⁸ or without⁷⁹ lateral canthotomy. To give the reader a full understanding of the topic, all incisions will be described, even those the authors do not use any more. Detailed anatomy of this extremely complex region is to be found elsewhere.⁸⁰

Infraorbital incision

The infraorbital incision is located over or slightly below the inferior orbital margin, preferably in a skin crease. The incision should be sloped caudally in medio-lateral direction and horizontal extension should be avoided to prevent violation of lymphatic



Fig. 40.11 Laceration sutured after reduction of fracture with help of para-alveolar releasing incision and undermining of resultant bipediced flap.

drainage resulting in long-lasting lymphatic edema. After incising skin, blunt dissection proceeds directly through fibers of the orbicularis oculi muscle to the orbital margin above the infraorbital foramen and the attachment of the mimic muscles. The periosteum is incised 2 mm below the orbital margin and dissected with a sharp periosteal elevator. If orbital floor is to be explored, dissection continues behind the orbital margin in close contact with bone, avoiding perforation of periosteum (Figs 40.12 and 40.13).

Closure is done in two layers: the periosteum is reattached by 4/0 resorbable suture and the skin by running intradermal 5/0 monofilament suture.

The advantages of this approach are technical ease, wide exposure, and no risk of ectropium or scleral show. The disadvantage is a prominent scar.

Subtarsal incision

The subtarsal incision is located just under the inferior margin of the lower tarsal plate that is at least 5 mm below the lid margin, usually in the first skin crease. The incision is either directly deepened through the orbicularis muscle, or stepped caudally. Dissection proceeds above the orbital septum and parallel with

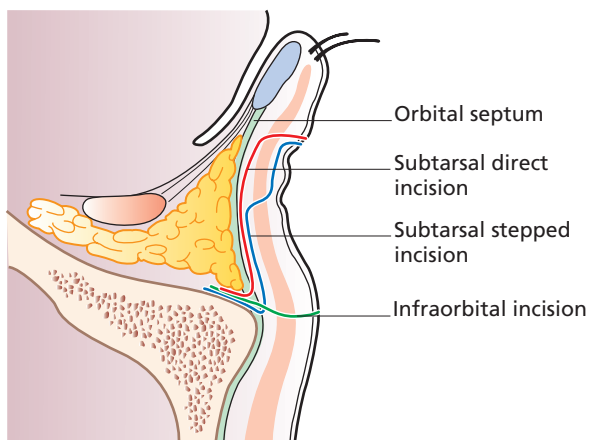


Fig. 40.12 Subtarsal and infraorbital incisions and dissecting paths.



Fig. 40.13 Fracture of the inferior orbital rim exposed through infraorbital incision.

it caudally, towards the inferior orbital margin. The rest of the procedure is identical to infraorbital incision (Fig. 40.14).

This incision provides very good cosmetic results and has a low risk of ectropium or scleral show. Currently it seems to be the preferred cutaneous lower lid incision (Figs 40.15 and 40.16).

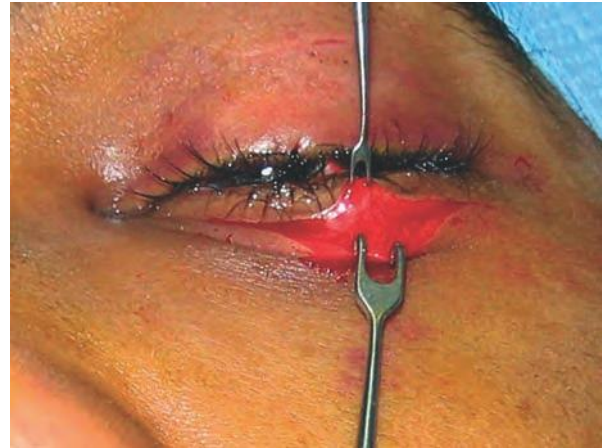


Fig. 40.14 Subtarsal incision: incision in skin crease of lower lid.



Fig. 40.15 Subtarsal incision: exposure of fracture of inferior orbital rim.



Fig. 40.16 Subtarsal incision: appearance of scar camouflaged in skin crease 6 months after operation.

Subciliary incision

The incision is done 2mm below the eyelashes and commences at the level of the inferior lacrimal punctum. It proceeds laterally along the whole width of the eyelid and can be extended up to the lateral orbital margin in a skin crease. After incising the skin, it is possible to cut directly through the orbicularis oculi muscle and proceed with dissection in the same manner as in the subtarsal incision. Another option is to dissect about 2mm caudally between the skin and the muscle and divide the muscle after that in a stepwise manner. The third option, now largely discarded, is to dissect subcutaneously to the orbital margin and cut muscle only there (Figs 40.17 and 40.18). This method of dissection is most prone to complications. The rest of the dissection is again identical to the previous approaches.

Subciliary incision results in a nearly imperceptible scar, however it is technically much more difficult and, unfortunately, it is fraught with a high incidence of complications. Up to 40% of patients develop scleral show or even ectropium.⁷⁸

Transconjunctival incision

The lower eyelid is retracted by a Desmarres lid retractor or 5/0 silk stay sutures through the tarsal

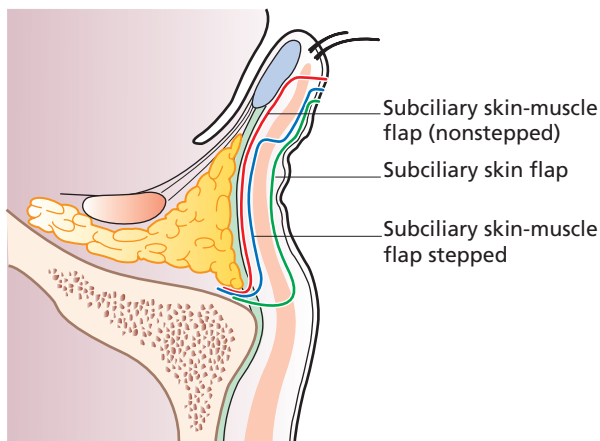


Fig. 40.17 Schematic drawing of three different variants of subciliary incision.



Fig. 40.18 Fracture of the inferior orbital rim exposed through subciliary incision.

plate. Some authors recommend use of a corneal shield to prevent corneal abrasions, but in our opinion it is not necessary. The cornea is usually satisfactorily protected by the upper eyelid, or can be covered by a conjunctival flap suspended by a suture. It is important to check the size of the pupil frequently if dissection reaches deep into the orbit close to the optic foramen and nerve. Use of a corneal shield makes this uncomfortable, so a corneal shield may not be used. The conjunctival incision is made between the lower margin of the tarsal plate and the conjunctival fornix. It is then deepened through the lower lid retractor and capsulo-palpebral fascia. The orbital margin is reached by spreading the prolapsing orbital fat with fine dissecting scissors. The periosteum is incised just behind the inferior orbital margin so that the orbital septum remains intact.

Closure consists of just suturing the periosteum; suturing the conjunctiva is not necessary. It is a good idea to mark the periosteal margins by a suture at the beginning of the orbital dissection to identify them easily during closure. The above-described procedure is known as retroseptal transconjunctival approach and is also widely used in cosmetic eyelid surgery.⁸¹

Alternatively a preseptal approach has been described, which avoids the orbital fat, but is technically more demanding and has a higher complication rate than the retroseptal procedure.⁸² The incision is made just under the inferior margin of the tarsal plate and dissection proceeds between the orbital septum and the preseptal part of the orbicularis muscle to the orbital rim, where the periosteum is incised below the orbital septum attachment (Fig. 40.19).

The transconjunctival incision without lateral canthotomy provides somewhat limited access, especially in young individuals with tight lids (Fig. 40.20). On the other hand it is cosmetically perfect since it leaves no visible scar.

Lateral canthotomy

The lateral canthotomy commences with about a 5mm long skin incision running horizontally from

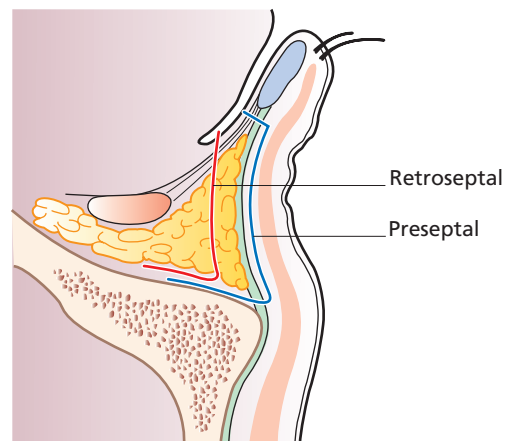


Fig. 40.19 Schematic drawing of transconjunctival incision with preseptal and retroseptal dissection paths.

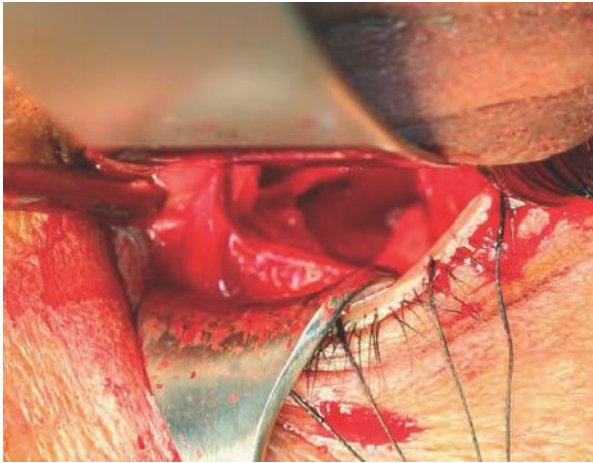


Fig. 40.20 Exposure of the orbital floor through transconjunctival retroseptal approach.

the lateral eyelid commissure. The incision should not extend beyond the lateral orbital margin. The skin is undermined with fine scissors caudally and the lower limb of the lateral palpebral ligament is identified as a tough whitish strip. The lower lateral margin of the eyelid is pulled medially using Adson forceps and the stretched lower limb of the ligament is divided with scissors. This enables free retraction of the lower eyelid and dissection can continue with a transconjunctival incision as described above. Combination of a lateral canthotomy and a retroseptal conjunctival incision makes orbital floor dissection easy and clear. It provides a kind of bird's eye view of the orbital floor and the infero-medial wall (Fig. 40.21).

With extension of the skin incision to about 1 cm and division of both limbs of the lateral palpebral

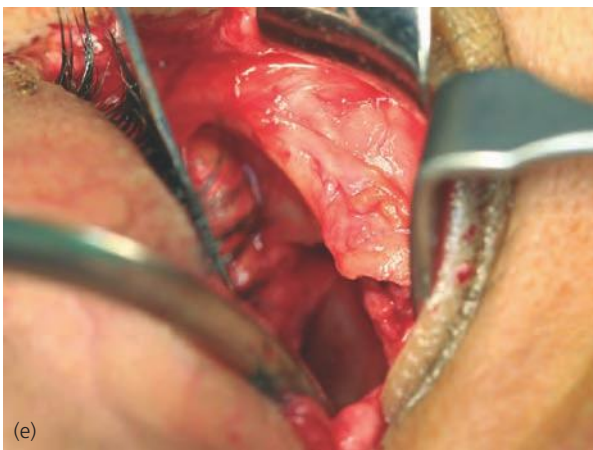


Fig. 40.21 Lateral canthotomy. (a) Division of inferior limb of lateral canthal ligament. (b) Incision of conjunctiva. (c) Dissection through capsulo-palpebral fascia and lower lid retractor. (d) Incision of periosteum of inferior orbital margin. (e) Fracture of inferior orbital rim and orbital floor exposed. (Courtesy of Dr Sabriyah Al-Saleh, Ophthalmology Department, Al-Adan Hospital, Kuwait.)

ligament, the periosteum can be stripped from the lateral orbital margin up to above the zygomatico-frontal suture. This will enable reduction and fixation of fractures in this area from the same approach as fractures of the lower orbital rim and floor (Fig. 40.22).⁸³

In closure of this approach, meticulous reattachment of the lateral canthus is of utmost importance. Failure to reconstruct the lateral palpebral ligament properly will result in rounding of the lateral palpebral angle and shortening of the palpebral fissure. The upper lateral margin of the inferior tarsal plate must be engaged by non-resorbable 5/0 monofilament suture and fastened tightly to the periosteum on the inner side of the lateral orbital margin at the

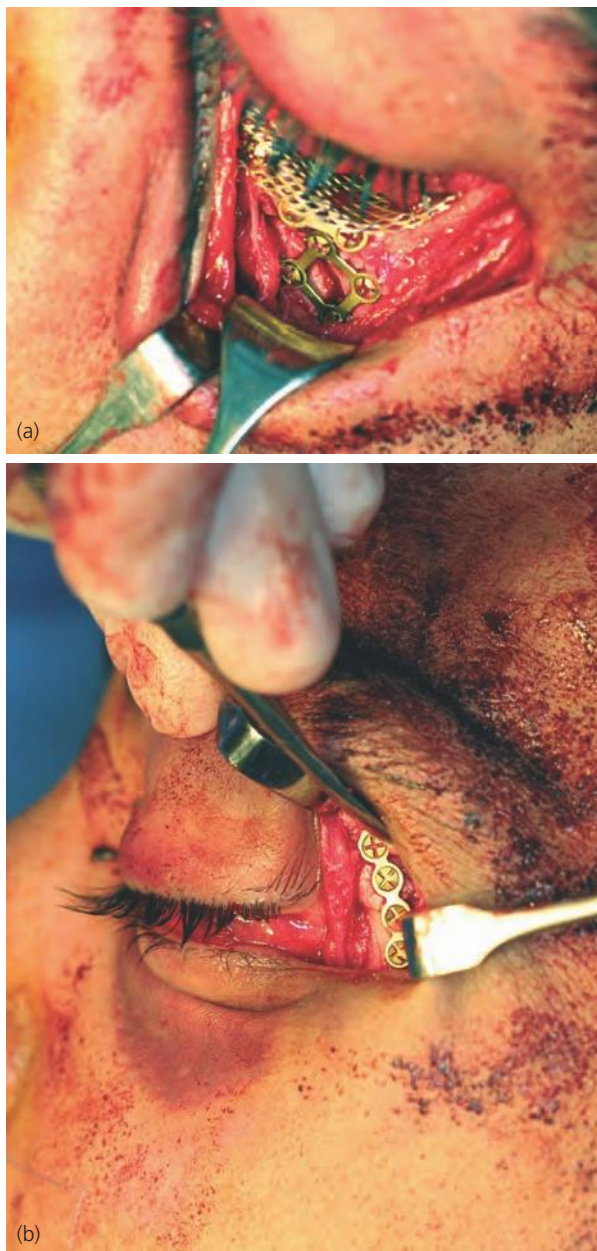


Fig. 40.22 Lateral canthotomy used for simultaneous approach to inferior and lateral orbital rim. (a) Inferior orbital rim plated and orbital floor reconstructed with titanium mesh. (b) Lateral orbital rim plated from the same approach. (Courtesy of Dr Sabriyah Al-Saleh, Ophthalmology Department, Al-Adan Hospital, Kuwait.)

level of Whitnall's tubercle. If the periosteum here was stripped or damaged by the dissection, the suture can be fixed to a drill hole, screw or miniplate. It is always better to overcorrect the suspension of the ligament in a cranial direction than to undercorrect it, because some loosening of suspension usually occurs in the postoperative period. If also the upper limb of the ligament was cut, it must be resuspended as well. The skin is closed with 6/0 monofilament sutures, with particular care to close the lid commissure precisely.

A disadvantage of lateral canthotomy is an external scar, which, however, is short and usually well camouflaged in periorbital wrinkles (Fig. 40.23). Imprecise resuspension of the lateral palpebral ligament can also result in a cosmetic problem.

Lateral eyebrow incision

This is an easy way to expose the fronto-zygomatic suture and apply osteosynthesis hardware. The incision, about 2 cm long, is made in the lateral eyebrow and can be directly deepened down to periosteum. The incision line should be kept straight without curving it alongside the supero-lateral orbital margin. Shaving of eyebrow hair is not necessary; actually it should be avoided because regrowth of the eyebrow is often irregular. If needed, the incision can be elongated medially and stop short of the supraorbital foramen. Unfortunately the eyebrow thins laterally in many individuals and the scar can be quite prominent in such circumstances.

Upper blepharoplasty incision

This approach takes advantage of the extreme laxity of the skin and subcutaneous tissue of the upper eyelid. The incision is made in a skin crease of the upper eyelid, just penetrating the orbicularis muscle and avoiding damage to the deeper attachment of the levator aponeurosis and the orbital septum. A musculocutaneous flap is then undermined and pulled cranio-laterally so that the periosteum of the orbital margin comes into view. This is then incised and



Fig. 40.23 Appearance of lateral canthotomy scar 1 month after operation.

retracted to expose the zygomatico-frontal area of the orbital margin. Closure is done in two layers: the periosteum is closed with 4/0 Vicryl, and the skin is sutured with 6/0 monofilament. The incision regularly gives excellent esthetic results (Fig. 40.24).

Coronal incision

In principle, the coronal incision follows, and extends below, the course of the coronal suture of the neurocranium, which is the suture between the frontal bone and the parietal bones. The incision always reaches across the temporal area to the superior attachment of the external ear and often continues caudally as far as the attachment of an earlobe. Sometimes the term bicoronal incision is used to denote the full extent of it, as opposed to a hemicoronal incision that is employed unilaterally.

A fully developed coronal flap provides access to the frontal bone, zygomatic arches, bodies of the zygomatic bones, medial, superior and lateral orbital margins and much of the corresponding orbital walls, as well as the nasal bones. By preauricular extension it is possible to address the temporomandibular joint and the upper neck of the condylar process of the mandible. It also allows harvesting of calvarial bone grafts.

Most authors state that there is no need to shave the hair, or that a 2 cm strip of shaven skin is suffi-

cient.⁸⁴ In the authors' opinion, this is valid for female patients with long hair, which can be divided by a comb and braided. Females are also understandably more distressed by the prospect of hair shaving than males. In consenting male patients, the authors see no harm in a complete hair shave, which makes suturing of the flap much more comfortable and subsequent wound care easier and more hygienic (Fig. 40.25).



Fig. 40.24 (a) Upper blepharoplasty incision approach to zygomatico-frontal component of ZMC fracture. (b) Favorable appearance of scar 1 month after operation.



Fig. 40.25 Skin preparation for coronal incision. (a) Combing and braiding of hair in a female patient. (b) Shaving of 2 cm strip of skin in a female patient. (c) Full head shave in a male patient; zig-zag pattern incision line is marked with surgical pen.

After proper skin disinfection and draping the planned line of incision is marked with a surgical pen. The incision line runs from ear to ear across the top of the head either straight or in a zig-zag or sinuous fashion.⁸⁵ There is always some hair loss in the incision line and the scar is much less prominent if it is not straight, especially in a patient with a short hair-cut. The placement of the incision line should take into consideration future balding patterns in men, and anterior migration of the scar due to growth of the neurocranium in young children.⁸⁶ There is no advantage in placing the incision more ventrally, because the extent of exposure is given by the caudal extent of the incision: the lowest points define the axis around which the flap will rotate.

The three superficial layers of the scalp make up one functional unit.⁸⁷ Vascularization is very rich and, due to the presence of fibrous septa, the vessels gape and bleed profusely when cut. After marking the incision the sub-galeal layer is infiltrated with saline or diluted local anesthetic with vasoconstrictor (e.g. adrenalin 1:200 000). This reduces the initial bleeding and makes establishment of the proper dissection level easier. The incision starts on the top of the head and progresses step by step latero-caudally to both sides, while arresting bleeding after each step (Fig. 40.26). The incision penetrates through the skin, subcutaneous tissue, and galea aponeurotica, and stops just above the pericranium. The fourth layer of loose areolar tissue is the level of flap development. Dissection inside this level is initially facilitated by undermining the incision line with a spreading hemostat. Hemostasis is mainly achieved by compression of wound margins by Raney clips, Tessier scalp clamps or a running interlocking silk suture. A use of electrocautery should be minimized and only bipolar coagulation should be employed to protect hair follicles.

After the whole length of the incision has been developed to proper depth, the scalp is pulled forward with a pair of cat paw retractors and the flap is dissected by reverse cutting with a large blade. This progresses to the point where the base of the flap dissected so far reaches a 45° angle with the zygomatic arches. The trans-sectional arrangement of soft tissues in the temporal area is more complicated.⁸⁷ The frontal branches of the facial nerve leave the parotid gland and cross close to the periosteum of the zygomatic arch into the temporo-parietal fascia, 15–28 mm ventral to the external acoustic meatus.⁸⁸ To protect them, further dissection in the temporal areas must continue under the deep temporalis fascia. The temporalis fascia is incised over the root of the zygoma and the incision progresses firstly through the external leaflet of fascia, just above the temporalis muscle fibers, alongside the base of the developing flap, to the superior temporal line. From here right and left incisions in the temporalis fascia are connected by incising the pericranium between them. The forward dissection of the coronal flap continues in the sub-

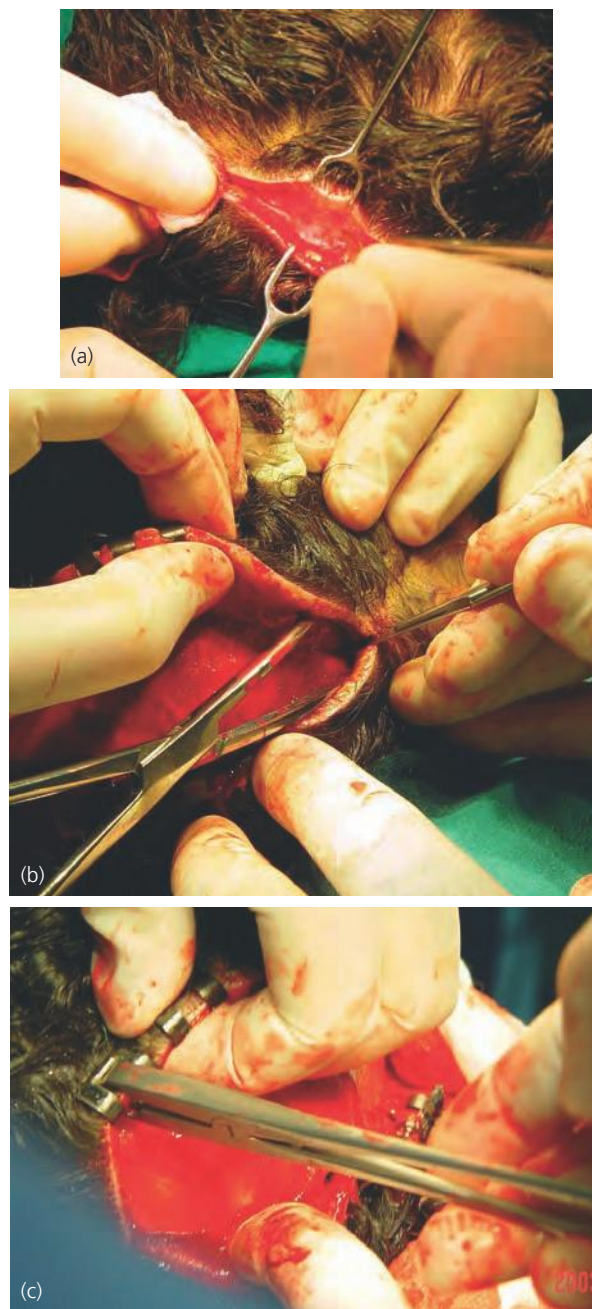


Fig. 40.26 Coronal incision. (a) Cutting through skin, subcutaneous layer and galea aponeurotica. (b) Dissection in sub-galeal layer. (c) Hemostasis using Raney's clips.

pericranial level, then subfascial level over the temporalis muscles. The connection between the periosteum and temporalis fascia at the superior temporal line is firmly adherent to the underlying bone and requires sharp dissection, which is best done by cutting diathermy (Fig. 40.27).

At this point it is necessary to consider if a pericranial flap will be needed for anterior cranial fossa repair. If this is the case, its design must be incorporated into the periosteal dissection instead of cutting the periosteum straight across the frontal bone (Fig. 40.28).

When the dissection reaches the orbital margins, careful attention is paid to identification and freeing of the supraorbital neurovascular bundles. This is

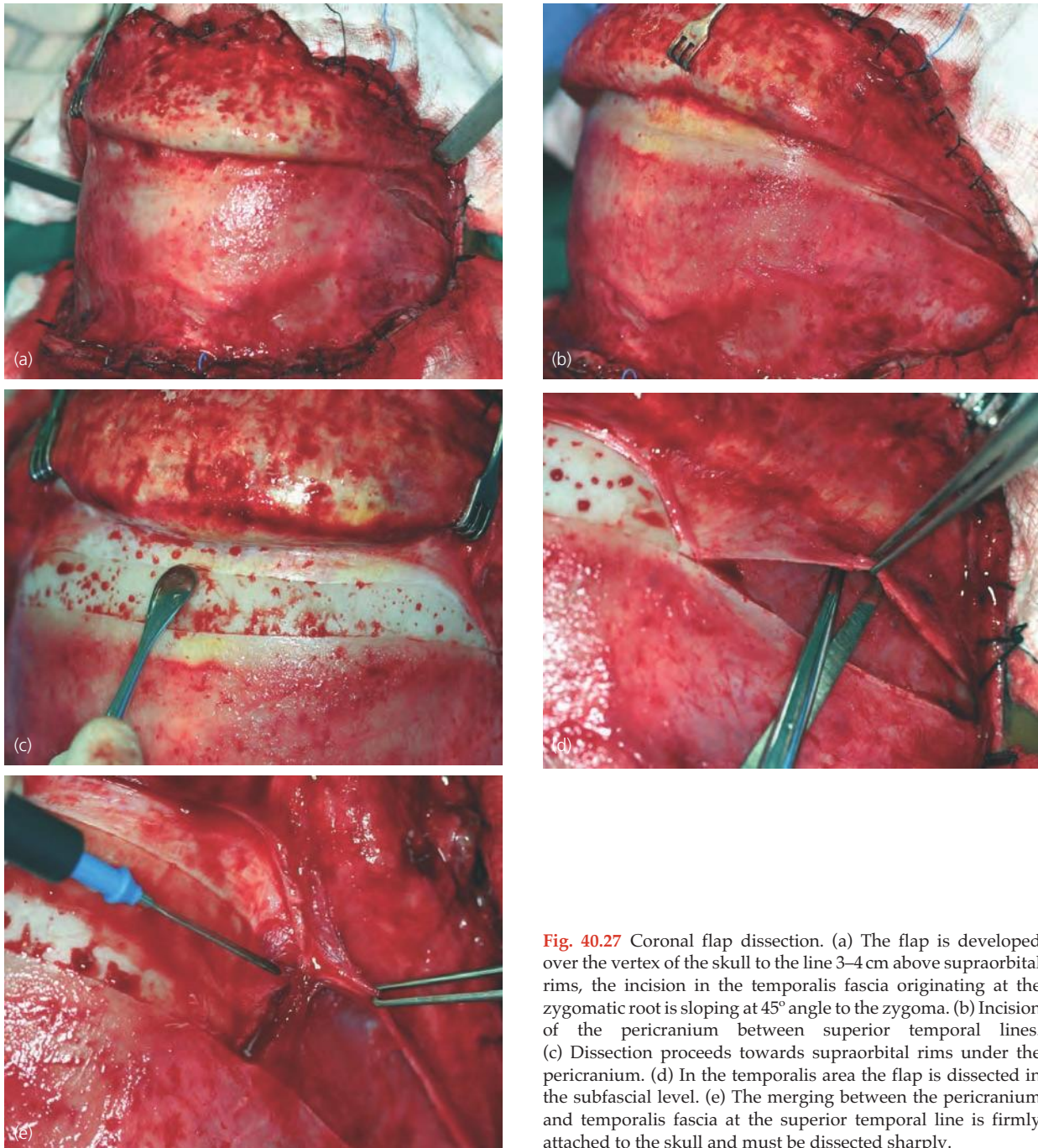


Fig. 40.27 Coronal flap dissection. (a) The flap is developed over the vertex of the skull to the line 3–4 cm above supraorbital rims, the incision in the temporalis fascia originating at the zygomatic root is sloping at 45° angle to the zygoma. (b) Incision of the pericranium between superior temporal lines. (c) Dissection proceeds towards supraorbital rims under the pericranium. (d) In the temporalis area the flap is dissected in the subfascial level. (e) The merging between the pericranium and temporalis fascia at the superior temporal line is firmly attached to the skull and must be dissected sharply.

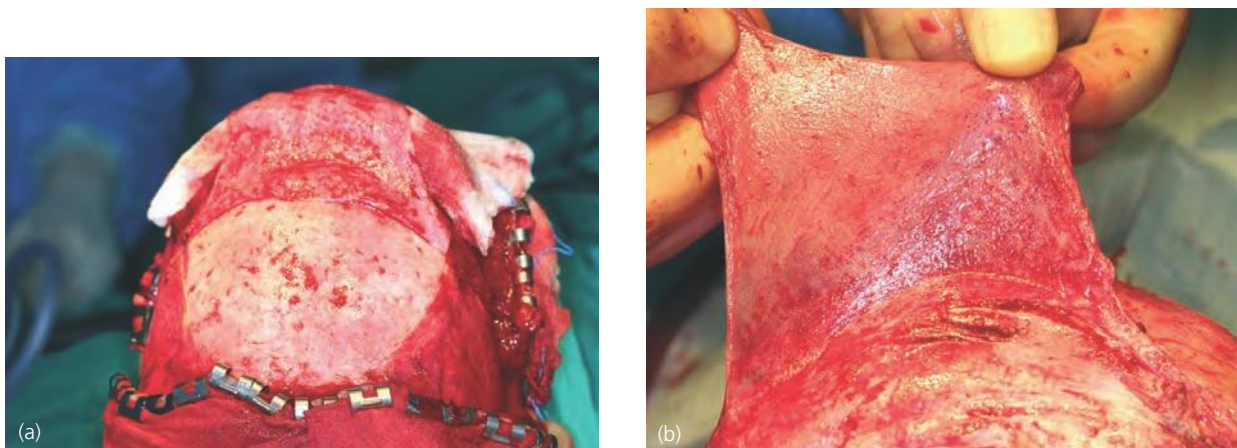


Fig. 40.28 (a) Pericranial anteriorly based flap can be raised over the vertex of the skull. (b) The flap can be used to support the dura mater repair and isolate it further from the paranasal sinuses.

easy if only supraorbital notches are present. If the bundles pass through supraorbital foramina, these must be converted into notches by resecting the foramina's inferior margins with a fine chisel. The periosteum must be subsequently elevated beyond the orbital margin and inside the orbital cavity to allow free retraction of the flap. If more exposure of the nasal skeleton and medial orbital walls is required, the periosteum above the nasal root can be incised vertically (Fig. 40.29).

Closure starts with suturing of the temporalis fascia. This should be very meticulous to prevent facial

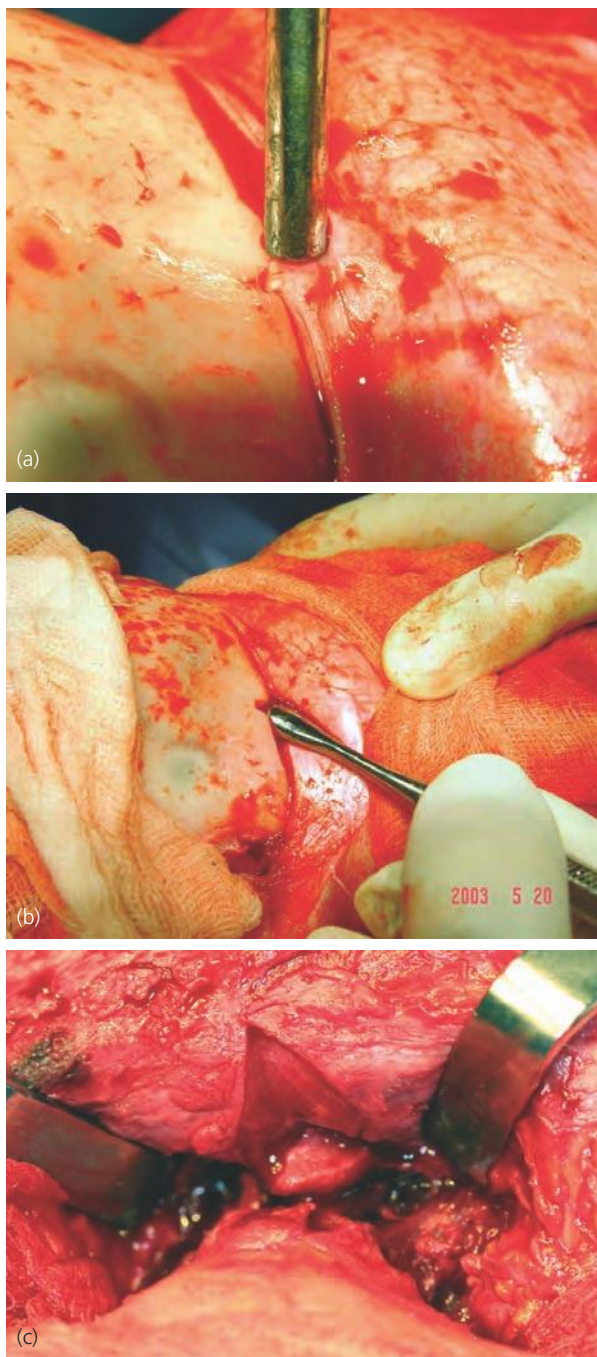


Fig. 40.29 (a) Supraorbital neurovascular bundle is encountered at the supraorbital rim. (b) It can be freed from the supraorbital foramen after resection of its inferior margin. (c) Vertical incision of periosteum over the nasal root will facilitate exposure of the nasal bones.

sagging. If the body of the zygomatic bone was widely denuded, the zygomatic periosteum should be supported by suspension sutures applied to hardware or purposely created drill-holes. Next suction drains are introduced under the galea aponeurotica and the coronal incision is closed in two layers: galea and subcutaneous tissue by widely inserted reversed vertical sutures of slow-absorbing Vicryl, the skin by surgical staples. When haemostatic devices like Raney clips are removed from flap margins before closure, some bleeding usually resumes, and this is controlled by bipolar electrocautery.

Limited transcutaneous approaches

Limited transcutaneous approaches use short incisions or stab incisions to insert surgical instruments to manipulate displaced fragments of bone and elevate them to their proper position, where they are supposed to interlock with stable contiguous bone margins. These approaches do not allow visual control of reduction or insertion of osteosynthetic hardware. They are almost exclusively used to reduce zygomatico-maxillary complex fractures.⁷ Sometimes, they are employed in combination with other approaches that allow application of osteosynthesis hardware.

Transcutaneous bone hook or screw

A stab incision is made over the inferior margin of the body of the zygoma on a line dropped from lateral canthus. A sharp bone hook is inserted with gentle pressure until its tip comes into contact with bone. It is then slipped under the bone margin lifting the handle of the instrument at the same time until firm purchase is felt by the surgeon (Fig. 40.30).

Alternatively, a T-bar screw (Carroll-Girard screw), an instrument resembling a corkscrew, is inserted into a drill-hole in the body of the zygoma and used as a handle to manipulate it. A pediatric narrow-flanged nasal speculum is used to protect soft tissues during drilling and screw insertion.

The skin is closed by a single 6/0 monofilament suture.

Gillies approach

A short oblique incision is made in the temporal area above the middle of the zygomatic arch. The incision is deepened to the temporalis fascia and the margins are somewhat undermined. The fascia is incised, taking care not to damage underlying muscle. A suitable instrument, like a Rowe elevator or a strong periosteal elevator, is inserted under the fascia and advanced towards and under the displaced bone fragment (Fig. 40.31).

The wound is closed in two layers: fascia with strong Vicryl, and skin with monofilament or staples.



Fig. 40.30 Transcutaneous bone hook technique. (a) The stab incision is made after palpation of the lower margin of the body of the zygomatic bone. (b) Tip of bone hook is inserted through the incision and brought into contact with the inferior margin of the zygomatic bone. (c) By lifting the handle of the instrument the tip of the hook is driven under the body of the zygomatic bone. (d) The fracture is reduced by traction while palpating the zygomatic arch and orbital margin.



Fig. 40.31 Gillies technique: Rowe's elevator is inserted through a short temporal incision subfascially under the zygoma.

Use of traumatic wounds

Traumatic wounds can sometimes be used for open reduction and internal fixation of midfacial fractures. This applies especially to rare cases of assaults with sharp weapons, which result in long, deep, cut wounds with regular margins and fractures located immediately under the wounds (Fig. 40.32). Lacerations, on the other hand, often do not penetrate all soft tissue layers and can be quite distant from

underlying fractures. Careful judgment is necessary to decide if such a wound can be deepened or extended enough to allow for uncompromised access to the fracture without functional or esthetic detriment.

Because most midfacial fractures are not definitely treated at the time of admission, it is desirable to consider whether facial wounds should be immediately formally repaired or only approximated.

Endoscopic approaches

There is a general trend in many surgical disciplines towards less invasive treatment, avoiding different unfavorable consequences and patient discomfort related to traditional open surgical procedures. Maxillofacial surgery is no exception and endoscopic procedures have become a well established part of therapeutic options in this field. The main advantages of endoscopic approaches in the maxillofacial area are limited incisions and consequent minimal scarring, less damage to vascular circulation, reduced blood loss, avoiding the risk of facial nerve damage, and improvement of intraoperative visual control in areas of limited exposure. In maxillofacial traumatology the main focus has been on endoscopically assisted reduction and fixation of mandibular condylar

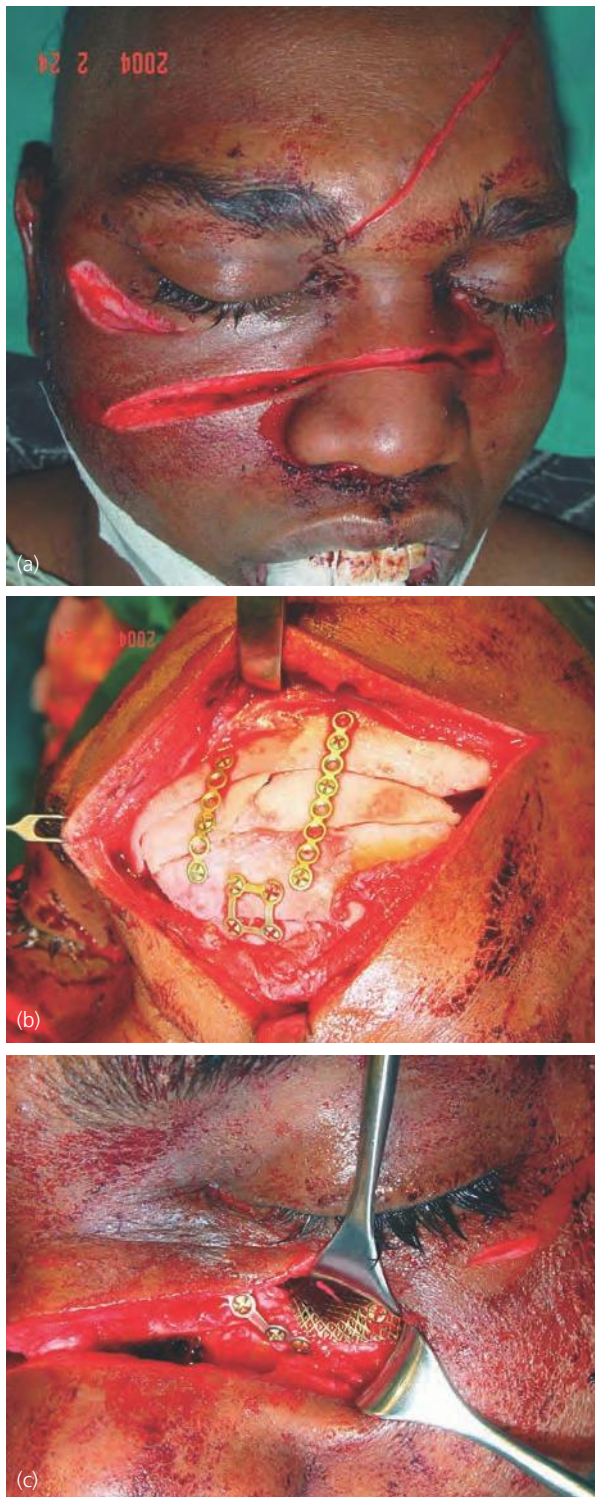


Fig. 40.32 (a) Several cut wounds of the face caused by assault with kitchen cleaver. (b) Cut wound of the forehead used to reduce underlying fracture of frontal bone. (c) Repair of inferior orbital rim and floor from another cut wound in the same patient.

fractures which is covered in Chapter 43. In management of midfacial trauma there has recently been development of treatment of orbit, frontal sinus, and zygomatic arch fractures.⁸⁹⁻⁹¹

Endoscopy augments, rather than replaces, general principles of adequate skeletal exposure, accurate fracture reduction, and appropriate internal fixation.⁹¹ The disadvantages of endoscopic fracture repair are

the need for special instruments, more complicated hardware manipulation in limited space, and consequently prolonged operation time.⁹²

Treatment at the site of fracture

Fractures of the maxillary alveolar process

Fractures of the alveolar process are often seen in combination with dental injuries. They are frequently accompanied by changes in the occlusion of the teeth. Hematoma in the vestibulum is often seen. Typically when a tooth in a fractured alveolar process segment is moved, the adjacent teeth in the segment move with it. Bi-manual manipulation will often disclose mobility of a bone fragment. Palpation with a finger placed in the vestibulum over the alveolar process may disclose a step in the bony contour. Fracture of the alveolar socket wall is often seen with lateral luxation of teeth and comminution of the alveolar socket is seen with intrusion of teeth. Radiographic examination will reveal fracture lines involving the periodontal space but extending into the bone beyond the periodontal space.

Dislocated fractures of the alveolar process without involvement of teeth are treated by repositioning and stabilization with osteosynthesis plates. When the alveolar socket is involved by comminution of the alveolar socket or fracture of the alveolar socket wall it is also very important to treat the dental luxation properly. Luxated teeth should be repositioned and splinted. The dental splint is usually also sufficient to treat the alveolar process fracture. Intruded teeth should be left for spontaneous eruption depending on the stage of root formation, and orthodontic traction may be preferred to surgical repositioning for reasons of periodontal healing. For details on treatment of dental trauma see Chapter 39.

Fractures of the maxillary sinus walls

Isolated fractures of maxillary sinus walls result mostly from direct impact, which is always somewhat mitigated by a thick layer of cheek soft tissue. The anterior wall is usually affected. The fracture can have the character of a mere fissure, or can be comminuted and displaced to various degrees. In extreme cases of comminuted fractures the completely detached fragments are found free in the maxillary sinus. Fracture lines sometimes reach the ostium of the infraorbital canal and the infraorbital nerve can be affected. The posterior wall of the maxillary sinus can be broken by the indirect mechanism of transmission of a deforming force through elastic bone. Fracture of the piriform aperture with intranasal dislocation of the fragment can lead to nostril obstruction.

Symptoms

Fracture is usually accompanied by edema and hematoma of the cheek. Anaesthesia of the innervation area of the infraorbital nerve can be present. Intraorally submucosal hematoma may be observed in the upper vestibule. Before development of edema it may be possible to palpate crepitus over the anterior maxillary wall during intraoral examination. There can be bleeding from the nose if blood escapes from the maxillary sinus through the natural ostium of the canalis semilunaris or if the lateral nasal wall and mucosa are injured as well.

Radiographic examination

Plain radiographs, such as as occipitomenal view or orthopantomogram, are not usually suitable for diagnosing fracture lines in thin sinus walls, but they can raise suspicion by detecting the presence of sinus hematoma. On the other hand, axial computed tomography (CT) scans with three-dimensional (3D) or sagittal reconstructions demonstrate even fine undisplaced fracture lines well.

Treatment

Undisplaced or even moderately displaced fractures do not require treatment, because they will not result in observable deformity. Fractures indicated for operation are those with free fragments or with prolapse of soft tissues into the maxillary sinus, and fractures with nasal obstruction.

The fracture is approached by a vestibular incision, the prolapsed soft tissues are reduced, free fragments are removed, and the hematoma is evacuated. Only non-vital mucosal lining is removed from the sinus; no nasal fenestration is done. Fragments big enough to hold screws are repositioned and fixed by micro/miniplates. If this cannot be achieved and the resulting defect would lead to recurrence of soft tissue prolapse, the defect is reconstructed using titanium mesh (Fig. 40.33).

Le Fort I fracture (Guérin fracture, suborbital maxillary fracture)

This fracture line runs more or less in a horizontal direction and separates the lower part of the maxilla, the horizontal plates of the palatal bones, and the inferior one third of the pterygoid processes of the sphenoid bone from the rest of the cranium. The nasal septum is also fractured or dislocated from its maxillary attachment. There can be interfragments along the fracture line. The main fragment is usually dislocated dorsally and its anterior part is also dislocated cranially, where it can become wedged, leading to anterior open bite and/or reverse overjet. Alternatively the whole segment can become loose and drooping with gravity leading to elongation of the middle facial third.

Symptoms

Aside from above-mentioned occlusal disturbance, the upper dental arch can be mobile; however it is sometimes wedged in a pathologic position and cannot easily be moved. In cases without wedging, the maxilla can descend under gravity on mouth opening. Steps can be palpated intraorally on the anterior maxillary wall before edema obscures them. Hematomas in the upper vestibule and epistaxis are frequent.

Radiographic examination

Orthopantomogram can detect Le Fort I fracture, especially if the pyriform aperture, zygomatico-alveolar crest and pterygomaxillary suture are carefully examined. CT scanning (coronal slices and 3D reformatting) offers superior imaging (Fig. 40.34).

Treatment

The purpose of treatment is to re-establish proper positioning of the maxilla with the upper dental arch and to achieve preinjury occlusion, as well as anterior projection of upper lip and anterior facial height.

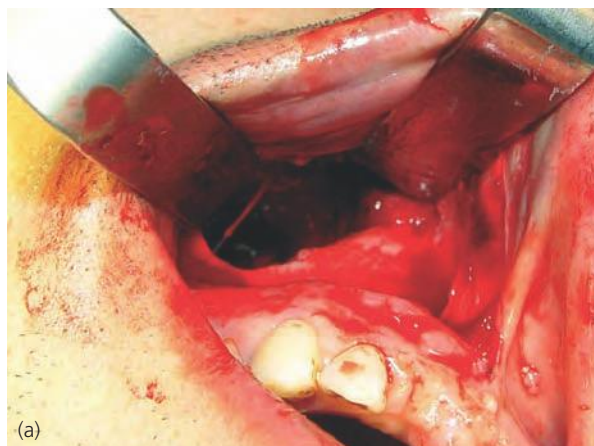


Fig. 40.33 (a) Comminuted fracture of anterior maxillary wall: defect after debridement of small fragments. (b) Maxillary sinus wall reconstructed with titanium mesh.

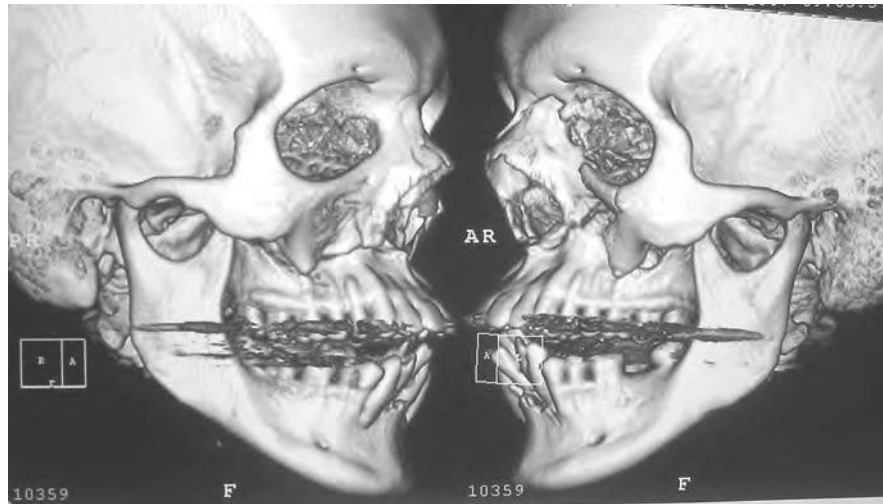


Fig. 40.34 Le Fort I fracture: 3D CT reconstruction. Note posterior displacement of the central fragment.

Historically, these fractures were treated by closed reduction and maxillo-mandibular fixation supported by internal wire suspensions or external fixators.⁹³ This mode of treatment usually restored occlusion, but the vertical position of the maxilla was compromised, leading to shortening of the middle facial third. This problem had already been recognized in the preminiplate period, and jig-saw puzzle reconstruction with interfragmentary wiring and bone grafting if necessary was recommended.^{1,13}

Today the leading tenet is reconstruction of the vertical maxillary buttresses. Two of them (paranasal and zygomatico-alveolar) are surgically accessible, so that four supporting struts can be repaired. This will

re-establish proper maxillary position in all three dimensions.

The surgery commences with application of arch bars on both dental arches. Then the maxilla is exposed by bilateral upper vestibular incision. Under visual control the maxilla is mobilized by wire traction on an arch bar, bone hook, or Rowe-Killey desimpaction forceps if necessary. Maxillo-mandibular fixation is secured in correct occlusion (mandibular fractures being repaired first) and the whole complex is rotated cranially with the mandibular condyles properly seated. In non-comminuted cases this maneuver will result in precise reduction of the buttresses that can be plated (Fig. 40.35). In comminuted

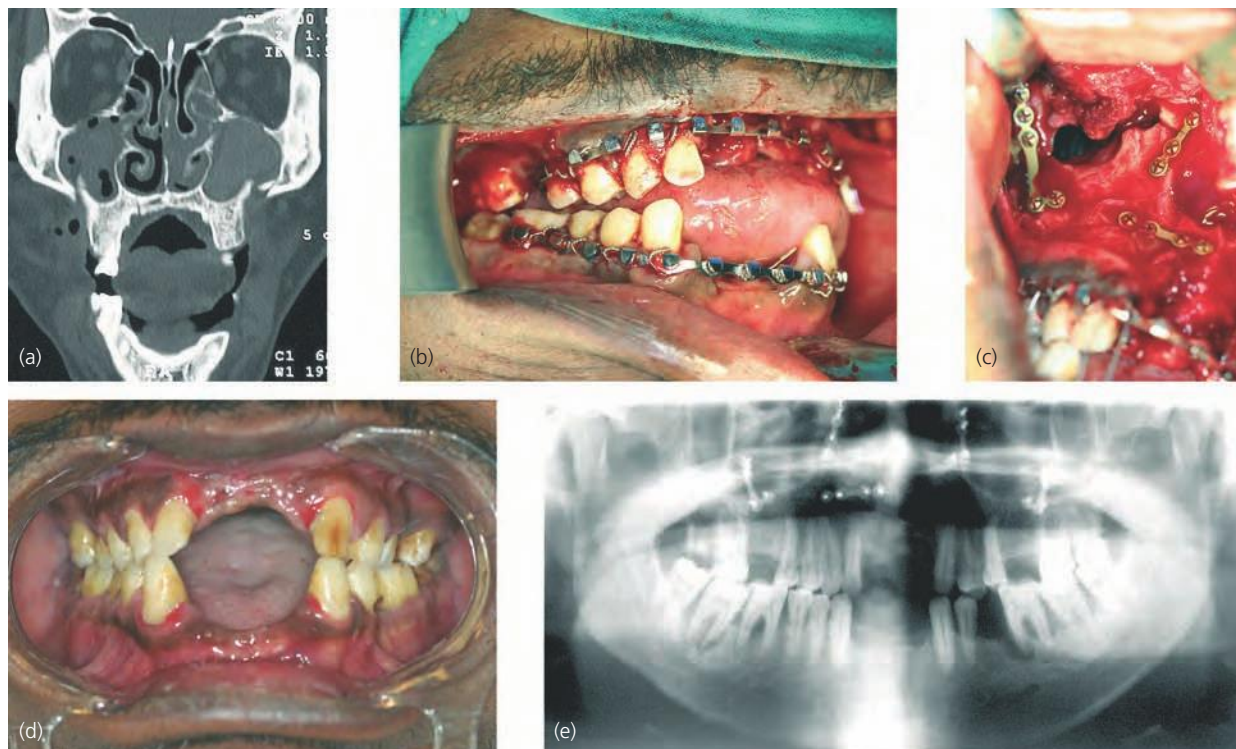


Fig. 40.35 Le Fort I fracture. (a) Coronal CT scan; note superior displacement of the fragment with comminution of fracture lines and linear fracture of hard palate. (b) Anterior open bite in the same patient. (c) Osteosynthesis of zygomatico-alveolar, paranasal, and sagittal fracture lines with titanium miniplates. (d) Normal occlusion after treatment. (e) Postoperative panoramic radiograph.

cases the buttresses should be reassembled or grafted.⁹⁴ If a small defect up to 5 mm exists, it can be bridged by miniplate. The need for bone grafting is extremely rare.

Fractures without displacement or with minimal displacement can heal spontaneously,⁹³ but must be supervised. The precondition is a non-comminuted nature of the fracture, because otherwise the facial height could be decreased by gradual impaction of a large fragment by function of masticatory muscles.

Fractures of the hard palate

Fractures of the hard palate are infrequent. They are found in less than 10% of patients with midfacial fractures.⁹⁵ They practically never occur in isolation and are usually part of alveolar process fractures or more complex midfacial fractures of the Le Fort type.⁹⁶ The rare exceptions are isolated fractures of the posterior margin of the palatal bone in young children due to falls on foreign bodies held in the mouth at the time of the accident.

Fracture patterns

The most often encountered fracture of hard palate is a component of alveolar process fracture. It begins interdentally, encircles an alveolar segment and either returns to the alveolar crest (in the incisor–canine area) or runs towards the posterior palatal margin (in the premolar–molar area).

A sagittal palatal fracture splits the hard palate and anterior alveolar process in the midline. It is found in children and adolescents, whose sagittal palatal sutures have not yet ossified.⁹⁷ A parasagittal palatal fracture is usually found in adults with a fully ossified midpalatal suture. It can start anywhere between the canines, either from the interdental septum or through the alveolar socket. In such cases, the involved tooth is usually luxated or avulsed. The fracture line travels posteriorly just next to the palatal crest and can reach the posterior margin of the palatal bone or deviate towards the maxillary tuberosity. Both sagittal and parasagittal fractures naturally involve the pyriform aperture. Some authors also recognize a variant of this fracture pattern: the para-alveolar palatal fracture, which runs further from the midline, close to the palatal base of the alveolar process.⁹⁵

A transverse palatal fracture runs in a coronal plane and divides the hard palate into anterior and posterior parts. It is rarely diagnosed, because it is mostly undisplaced. A complex palatal fracture combines the above-described fracture lines in an unpredictable fashion and is found only in the most severe midfacial injuries.

Symptoms

It must be remembered that palatal fractures, with the exception of alveolar process fractures, are almost

invariably associated with complex midfacial fractures, mostly of the Le Fort type.⁹⁶ Among other symptoms of midfacial fractures, suspicion should be raised by soft palate ecchymosis, hard palate mucosa laceration, luxated or missing incisor and split laceration of the upper lip and/or nasal floor. Fractures can be displaced to various extents, which can result in traumatic diastema and lateral crossbite. Fresh fractures show pathologic mobility.

Radiographic examination

A panoramic radiograph together with occlusal intra-oral film can confirm the diagnosis, however these investigations are often unfeasible in severely injured patients. The examination of choice is coronal and axial CT scan.

Treatment

Displaced palatal fractures greatly complicate treatment of complex midfacial injuries. They increase the potential for fracture malalignment, especially in cases of concurrent mandibular body–condyle fractures. In such instance it is very hard to establish the correct width of the dental arches.

In the presence of an intact mandibular dental arch, this can be used as a matrix for upper arch reduction. The fragments are then fixed by arch bar and circumdental wiring. This may be enough for isolated alveolar fractures, but not for sagittal or parasagittal fractures combined with a Le Fort type fracture. Such fractures have a tendency for rotation of fragments outwards and posterior splaying of the upper dental arch.

Proper reduction and fixation of the maxillary parts into full contact and proper axial alignment of teeth is easier in cases where the fracture line is widened only in the anterior part. In these cases the arch bar fixation can be supplemented by miniplate osteosynthesis at the base of the alveolar process just under the pyriform aperture (Fig. 40.36). It is much harder to achieve reduction of posteriorly widened fractures. Digital pressure is usually insufficient and a special instrument (reduction forceps) is sometimes needed to achieve approximation of the fragments (Fig. 40.37). These should be then secured by miniplates, usually inserted through the palatal mucosa laceration, which can be extended and undermined for this purpose (Fig. 40.38). Vertical fracture lines on the anterior maxillary surface are also plated, so that the whole caudal maxillary segment below Le Fort I line is restored to one unit. The treatment then continues with reconstruction of the vertical maxillary buttresses.⁹⁸

Fractures of nasal bones and related structures

Nasal fractures are not only among the most common facial fractures, but also the most frequent fractures

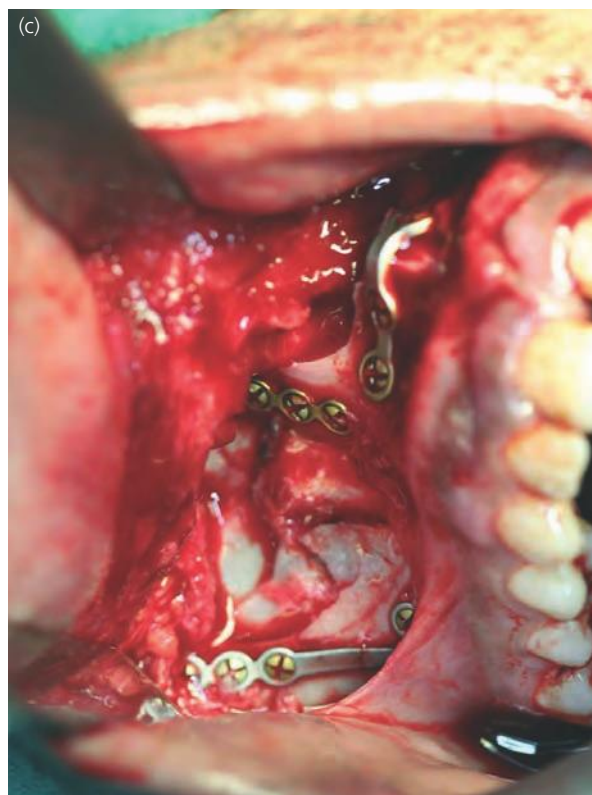
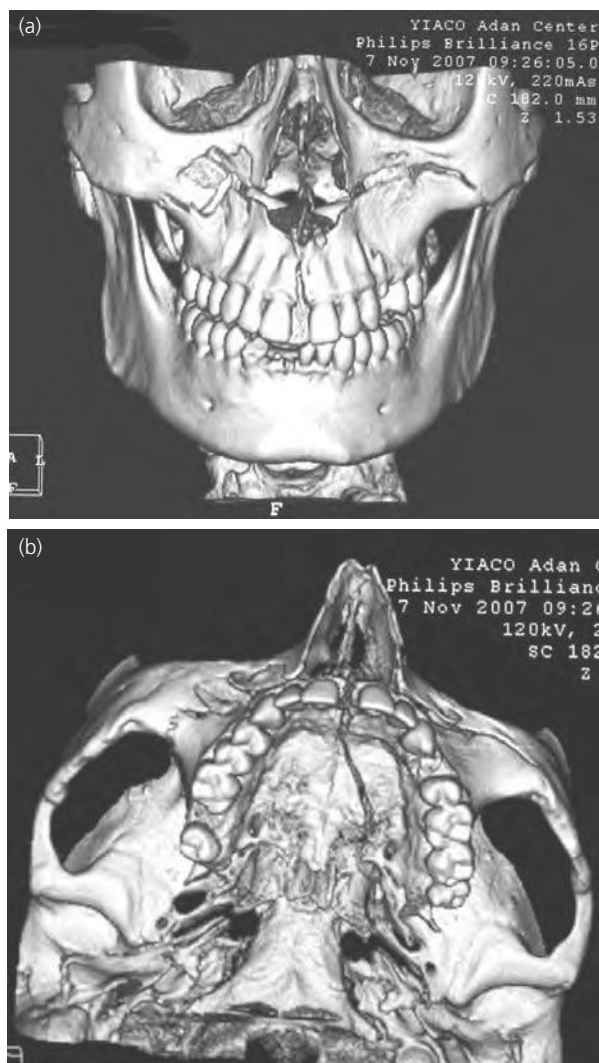


Fig. 40.36 Le Fort I fracture with sagittal split of the palate. (a) 3D CT reconstruction. (b) The fracture line deviates from the midline behind the central incisors and the alveolar process is displaced laterally. (c) Osteosynthesis of zygomatico-alveolar and paranasal pillars with 1.7 mm miniplates; the sagittal fracture is fixed with a 2.0 mm miniplate.



Fig. 40.37 Hayton-Williams clamp can be used to compress laterally splayed maxillary halves.

in general.^{9–11,99} Their incidence is often underestimated, because many of them are not diagnosed at the time of injury. This omission can lead to chronic nasal obstruction, septal deviation, and external deformity requiring secondary corrective procedures.

Less than perfect results of nasal fractures treatment were reported in retrospective studies.^{100,101} It was also noted that approximately 30% of patients with a nasal fracture have pre-existing nasal deformities.¹⁰¹ Preinjury facial photographs should be obtained whenever possible. Photography of the injury is an important part of the documentation,

because it can later give the patient an appreciation of the extent of deformity before surgical intervention.

Fractures of nasal bones

Symptoms

Nasal fracture should be suspected with any midface trauma. The initial main symptoms include epistaxis, nasal obstruction, and external deformity. This is usually obscured by edema developing within a few hours after injury. Earlier nasal obstruction and fracture should be excluded by careful history so as not to confuse present injury with pre-existing deformities. Crepitance or mobility of broken segments on palpation is diagnostic of fracture. The tip of the nose should be pressed postero-superiorly to evaluate the degree of loss of cartilaginous support. Skin and mucosal lacerations, ecchymosis, and hematomas may be also present. More severe fractures are accompanied by eyelid edema, subcutaneous emphysema, periorbital ecchymosis, conjunctival chemosis, and subconjunctival hemorrhage.

A very important part of evaluation is careful intranasal inspection. Intranasal examination should include topical decongestion of the nasal mucosa. It

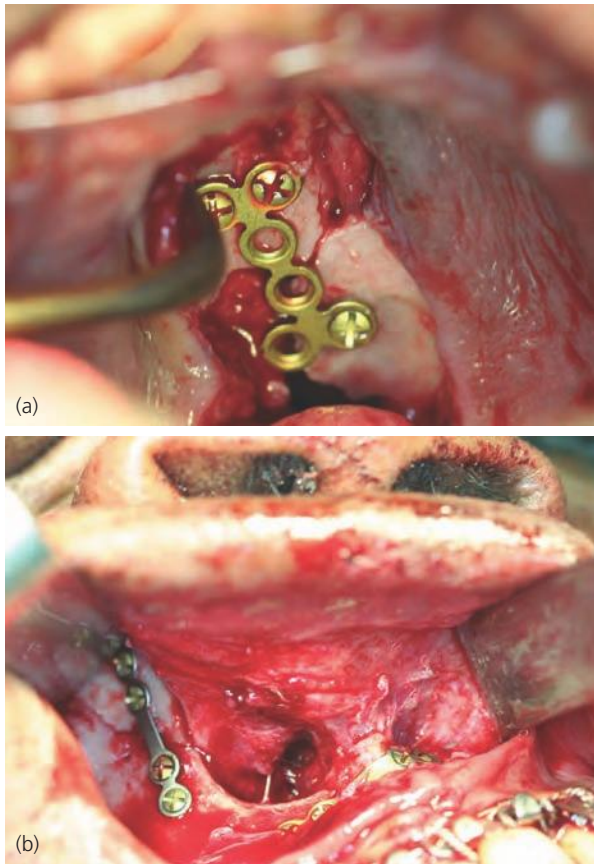


Fig. 40.38 (a) After reduction of a parasagittal fracture by compression the palatal laceration is used to insert a miniplate. (b) Properly transversally reduced arch is then fixed to upper maxillary part.

should be especially focused on checking for septal dislocation and submucous hematoma.

Radiographic examination

Routine radiographs are presently considered to be financially inefficient and unnecessary.^{101,102} Plain nasal films fail to reveal nearly 50% of clinically evident fractures. Old fracture lines cannot be differentiated from current minimally displaced fractures. Axial and coronal CT scans should be ordered instead, if there is any suspicion of more complicated fracture pattern, like NOE fracture (Fig. 40.39).

Treatment

Unless the patient presents before significant distorting edema has developed, most authors advocate delaying the procedure 3–7 days to allow for recession of the edema. Closed reduction may still be attempted in the first 3 weeks. After 3 weeks the fracture has usually healed and it cannot be easily reduced. In such case corrective rhinoplasty should be delayed 6 months or more to allow for maturation of the scar tissue.¹⁰³

The nose is extremely well sensitively innervated and perfect anesthesia is essential for achievement of a satisfactory result. Most nasal fractures can be managed under local anesthesia and no differences in outcome were found between patients treated under

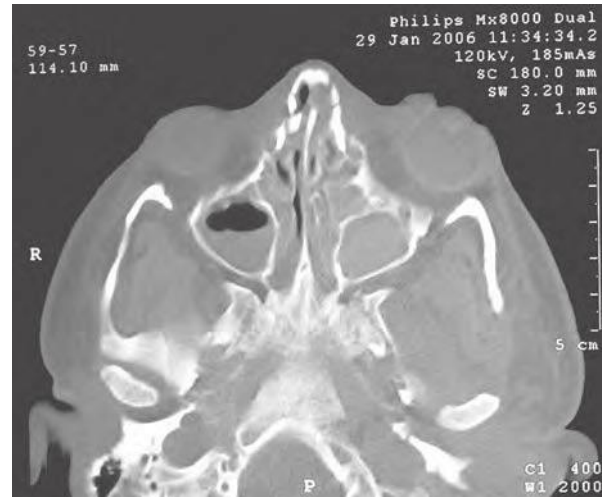


Fig. 40.39 Comminuted naso-ethmoid fracture; axial CT scan.

general and local anesthesia.¹⁰⁴ Intravenous sedation is a great help in such an instance. However, if any doubt exists about the straightforwardness of the procedure or the level of a patient's cooperation, general anesthesia should be employed. Even if the reduction is done under general anesthesia, the nose is anesthetized using 4% cocaine with 1:100 000 epinephrine-soaked pledgets. This shrinks the nasal mucosa sufficiently to allow adequate intranasal examination and instrumentation.

Closed reduction should always be attempted in patients seen within the first 2–3 weeks post injury, even if they will need subsequent rhinoplasty. Necessary instruments include Asch and Walsham forceps, blunt Boies nasal elevator, intranasal specula, good suction, and a head light. Nasal fractures are reduced by upward and outward pressure with an elevator placed under the nasal bone about 1 cm below the nasofrontal angle. The reduction is controlled and helped by external digital pressure and molding. The Asch or Walsham forceps may be used by inserting one blade in each nostril or by placing one blade in the nose under the nasal bone and the other on the overlying skin. Incomplete greenstick fractures of the septum and nasal bones can account for later deviations and should be completed prior to reduction.

The bony and cartilaginous structures of the nose are intimately connected with the nasal septum. A common mistake in reduction of nasal fractures is to reduce the bony fracture without attention to the septum. Septal fractures are actually present in an overwhelming majority of cases.¹⁰⁵ Reducing the nasal bone fragments first often reduces the septum simultaneously; if not, the Asch forceps is used to gently elevate the nasal pyramid while the displaced septum is manipulated into its proper place.

If the nasal bones are unstable or comminuted, they must be supported with intranasal packing. The authors usually use expandable foam packs with embedded small airway to enhance patient comfort.

The reduced septum may be also stabilized with silastic intranasal splints secured by a mattress suture, or by subperiosteally placed biodegradable membrane.¹⁰⁶ The reduced nose is supported externally by a splint (Fig. 40.40). The packing is removed after 3 days and the splint after 10 days.

If the fracture cannot be managed satisfactorily by closed reduction, the operation should be immediately converted to an open reduction to avert or minimize the need for secondary revision.⁹⁹ Open reduction, as well as septoplasty and rhinoplasty for delayed repair, should be performed only by an experienced nasal surgeon.

If the nasal fracture is a part of a more complex midface fracture pattern, it can best be treated using a coronal flap (Fig. 40.41). After complete circumorbital dissection and release of the supraorbital nerves, the whole bone skeleton of the nose can be exposed, sometimes with the help of vertical periosteal incision over the nasofrontal angle to release tension. The fragments may be elevated into correct position with intranasal instruments and fixed with miniplates. This approach also offers the possibility of primary bone grafting. Another option, employed mostly for delayed treatment or secondary procedures, is midfacial degloving.⁷⁵

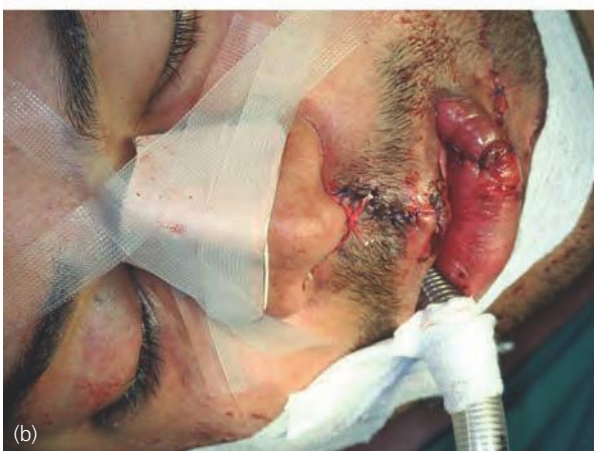
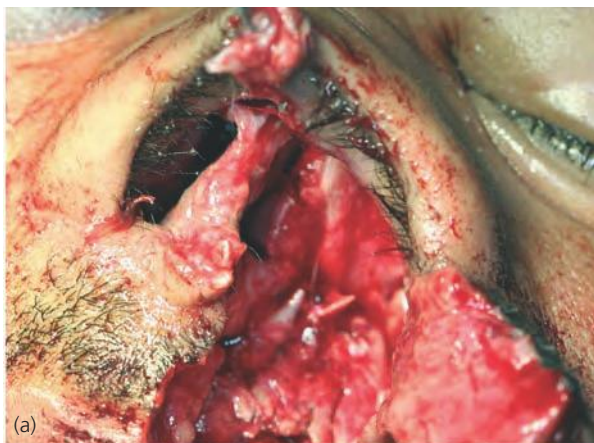


Fig. 40.40 (a) Laceration of upper lip and nasal sill accompanying nasal fracture. (b) After suturing of soft tissues, the nose is reduced, packed, and supported by an external splint.

Naso-orbito-ethmoidal fractures

Fractures of the nose sometimes do not remain limited to the nasal bones, but extend into adjacent bony structures: the frontal processes of the maxilla, the lacrimal bone, the ethmoidal bone or even the nasal process of the frontal bone. The nasal bones and the frontal processes of the maxilla form a relatively sturdy unit compared to underlying fine, paper thin, bony labyrinth of the lacrimal bones, lamina papyracea, ethmoidal cells, and cribriform and perpendicular plate of the ethmoid. These deeper structures easily yield and telescope on to themselves, thus allowing dorsal displacement of the robust outer frame. Line of such fracture, denominated as NOE fracture, separates the nasal bones from their frontal articulation, continues latero-caudally across the frontal process of the maxilla into the orbit, and runs through the lamina papyracea or lacrimal bone, and further through the orbital process of the maxilla and the inferior orbital margin back to the lateral wall of the nose (Fig. 40.42). Such fracture may be more or less comminuted. With dorsal displacement of the external framework communication between the nose and the frontal sinus can be disrupted. Important



Fig. 40.41 Repair of a nasal fracture through a coronal flap approach.

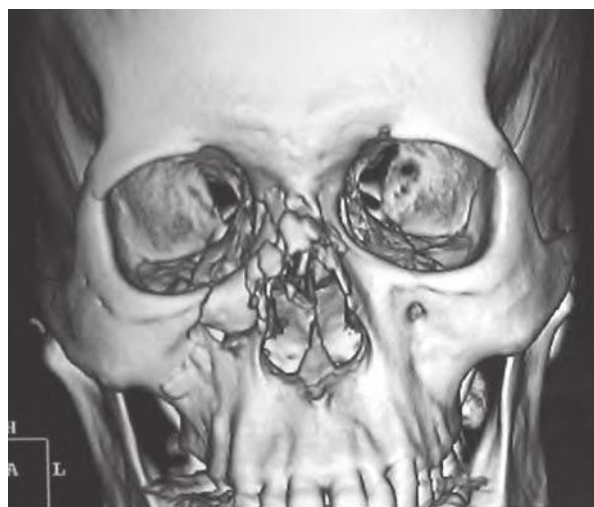


Fig. 40.42 Naso-orbito-ethmoidal (NOE) fracture; 3D CT reconstruction.

anatomical structures, namely the medial canthal ligament and the lacrimal apparatus, can also be compromised.¹⁰⁷

The fractures can be classified according to degree of comminution and status of the medial canthal ligament:

1. The central fragment is in one piece.
2. The central fragment is comminuted into manageable pieces, the canthal ligament remains attached to a fragment large enough to allow osteosynthesis.
3. The central fragment is comminuted into non-manageable pieces, the canthal ligament is either completely detached or attached to a fragment too small to allow osteosynthesis.

The whole detached complex may be also divided by a parasagittal fracture line and splayed to the sides, so that the nasal bridge is flattened and the canthal attachments are displaced laterally. Both detachment of the medial canthal ligament and its lateral displacement due to dislocation of its attachment lead to pseudohypertelorism and obtuse deformity of the medial palpebral angle.

Treatment

Proper restoration of form and fixation of NOE fractures usually require the wide access provided only by a coronal flap.¹⁰⁸ Wide exposure of the nasal bones and medial orbital walls is enabled by vertical incision of the periosteum over the nasal bridge. An intraoral approach to the paranasal areas of the maxilla or a transconjunctival approach to the inferior orbital rim and infero-medial wall are also often needed.

The central fragment must usually be repositioned ventrally by intranasal manipulation to re-establish projection of the nasal bridge and reconnect it to the frontal bone. Both nasal bones, if split and splayed, must be repositioned and held in the proper angle. The medial canthal ligaments must be restored to their correct position, preferably by exact reduction of their bone attachments. Various miniplates from different manufacturers are available to achieve these goals. The medial orbital walls are usually comminuted, and because the thinness of the lamina papyracea precludes use of any osteosynthesis devices, they must be reconstructed by autogenous bone grafts or implants.

In cases where the medial canthal ligament is detached or attached to an unusable bone fragment, it must be engaged and fastened in its proper position by a procedure known as medial canthopexy. It consists of suturing the ligament with a fine wire suture passed transnasally either to a drill-hole on the opposing undamaged side, or to a cantilevered miniplate. It is necessary to bear in mind that the position of the posterior arm of the ligament is what matters regarding the shape of the palpebral fissure and the position

of the lower lid. Therefore the canthopexy should be positioned deep enough in the orbit. Because canthopexy is a difficult and not very reliable procedure, every effort should be made during dissection to preserve the canthal ligament attachment to a bone fragment that could be stabilized by a mini- or microplate.^{109,110}

Fractures of the zygomatic bone

The zygomatic bones have a prominent position in the midfacial skeleton, making them prone to injury in different kinds of accidents. They are sort of keystones of the lateral facial skeleton, articulating with both calvarial (os temporale, os frontale, os sphenoidale) and other facial (maxilla) bones. These intimate contacts result in the zygomatic bone being only rarely broken in isolation. Fractures of the zygomatic bone usually transgress their strict anatomic limits or at least involve above-mentioned articulations. Fractures of zygomatic bone are present in over 40% of all facial fracture patients and are second only to nasal fractures in frequency.^{26-29, 111}

Fractures of the zygomatic bone are also referred to as fractures of the cheekbone or malar bone, fractures of the zygomatico-maxillary complex, zygomatico-orbital fractures, tripod or tetrapod fractures.⁶⁻⁸

The mechanism of injury is usually direct impact. The energy involved, site of impact, and direction of impact will affect the type of fracture (isolated zygomatic arch versus complex zygomatico-maxillary or zygomatico-orbital), the degree and direction of dislocation, and the degree of comminution.

Fractures of the zygomatic arch

Isolated fractures of the zygomatic arch are relatively rare, because for them to happen, traumatizing impact has to come from a lateral direction. On the other hand, they are frequent as a component of zygomatico-maxillary or Le Fort III fractures,¹¹² and are mostly caused by an indirect mechanism: displacement of the body of the zygoma posteriorly, leading to buckling of the zygomatic arch and fracture when the limit of its elasticity is overstepped.

The typical pattern of an isolated zygomatic arch fracture is of the letter M, with two fragments collapsed medially and often impinging on the masseter muscle or even the muscular process of the mandible (Fig. 40.43). Another possible pattern is one with only one site of major displacement, while the distant part of the fragment has just a greenstick fracture, or is only bent (Fig. 40.44).

Symptoms

The patient usually presents with limited or painful mouth opening. There can be visible depression of the facial contour at the fracture site, which later becomes camouflaged by developing edema.



Fig. 40.43 Depressed M-shaped fracture of the zygomatic arch with three fracture lines.



Fig. 40.44 Oblique fracture of the zygomatic arch as a component of a zygomatico-maxillary complex fracture. The zygomatic process of the temporal bone just has a greenstick fracture and is displaced medially. The body of the zygomatic bone, with its temporal process, is rotated.

Radiographic examination

The classical X-ray projection is an axial (submento-vertex) view of the skull. Fracture of the zygomatic arch can be also diagnosed on an occipito-mental view, and on a CT scan. CT examination can however miss this fracture if the slices are not thin enough and coronal scans or reformatting was not done. Ultrasonography is another diagnostic option which has been employed recently.

Treatment

Isolated zygomatic arch fractures are usually reduced by a limited transcutaneous approach, either transcutaneous bone hook reduction or Gillies temporal approach.¹¹³ Transcutaneous bone hook is faster, but less reliable and can lead to injury of the frontal

branches of facial nerve. The authors recommend it only for a fracture located anteriorly, near the body of the zygomatic bone. On the other hand, Gillies approach is safe and highly reliable. The elevator is introduced under the zygomatic arch, which is reduced while palpating and counteracting over-reduction with a finger (Figs 40.31, 40.45).

Open reduction of isolated zygomatic arch fracture is very rarely done, because only very posterior fractures are accessible from relatively easy preauricular incision and coronal incision is not justified in this instance.

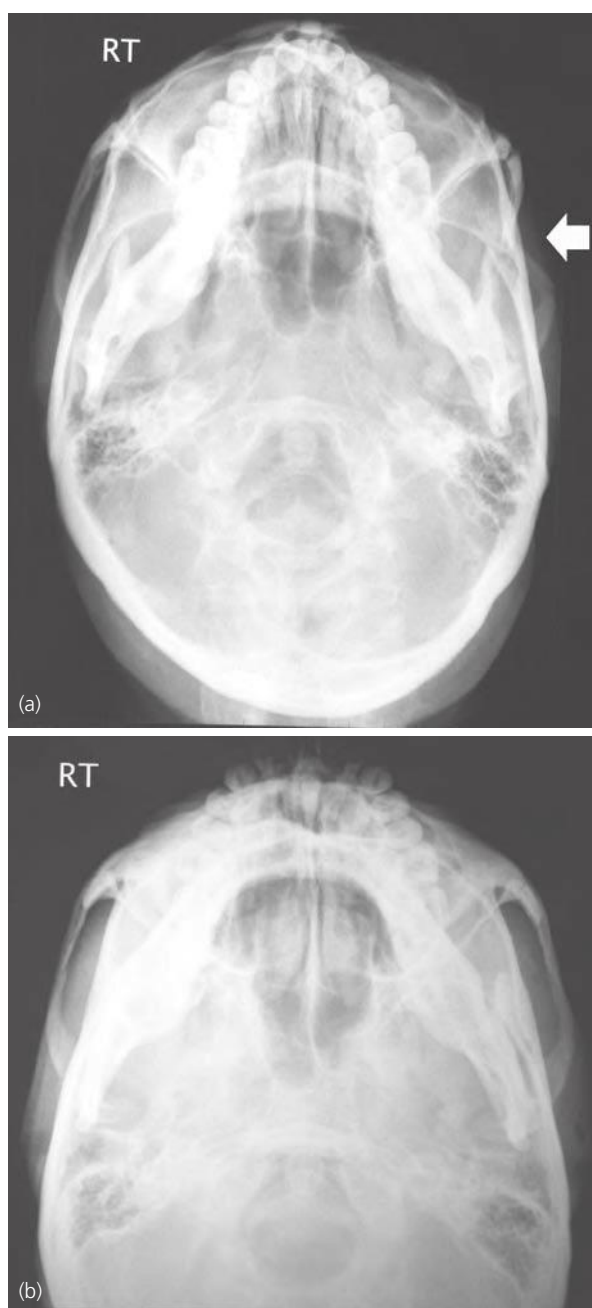


Fig. 40.45 (a) Depressed fracture of the zygomatic arch on a submento-vertex radiograph. (b) After reduction achieved through Gillies approach.

Fractures of the zygomatico-maxillary complex (zygomatico-orbital fractures)

Fractures of the zygomatico-maxillary complex (ZMC) are extremely diverse as to direction and degree of dislocation, or number of fragments. Many of these fractures are not displaced and therefore could have been easily overlooked in the past, when CT scans were not available or were not done routinely in all suspected facial fracture cases.

While some of the zygoma's four articulations with other bones can be fully interrupted and dislocated, others can be just of a greenstick character. This is mostly the case of zygomatic arch and fronto-zygomatic articulation. In the case of the widest articulation, with the maxilla, three areas should be considered separately: the zygomatico-alveolar crest, inferior orbital margin, and orbital floor.

The degree of fragmentation of fracture lines has an impact on the precision of reduction and stability of reduced fragments. Unfortunately many ZMC fractures have fragmented margins, especially in the area of the zygomatico-alveolar crest, which is also the most important support of the zygomatic bone in a vertical direction. As a result, the authors are skeptical about attempts to reduce ZMC fractures blindly and without fixation.

Symptoms

Symptoms of ZMC fracture will depend upon sites, type, and degree of dislocation. The most universal clinical finding is edema of overlying soft tissues, which develops during the hours after injury, and can later mask other symptoms or make their evaluation difficult (Fig. 40.46).

If the patient presents immediately after injury with a severely dislocated fracture, he/she can have considerable facial asymmetry and steps can be palpated on the zygomatic arch, inferior and supero-lateral orbital margins, as well as the zygomatico-alveolar crest. The body of the zygoma can be "bodily" dislocated or rotated, but mostly both types of movement

combine. It can cause loss of anterior and/or lateral projection of the zygoma, resulting in a flattened face and asymmetry. Bodily inferior dislocation with separation of the zygomatico-frontal suture will result in a change of position of the lateral canthus and therefore a downward slant of the palpebral fissure in the horizontal plane (Fig. 40.47). Dislocation of the orbital walls can lead to an increase (frequent) or diminution (rare) of orbital volume resulting in enophthalmos or exophthalmos. Limitation of ocular motility may be present either from injury or entrapment of ocular muscles. Because the fracture nearly always involves the infraorbital canal, anesthesia of innervation area of the infraorbital nerve is found in over 80% of patients.¹¹⁴ Bleeding from the nose is common. In cases of severe dislocation of the zygomatic arch or posterior displacement of the body of the zygoma, mouth opening can be limited due to interference with the coronoid process of the mandibular ramus and injury to the temporalis muscle. Orbital hematoma and subconjunctival suffusion usually develop during the first 24 hours.

All patients with ZMC fracture should be evaluated by ophthalmologist,¹¹⁵ not only to evaluate ocular position and motility, but also to exclude concomitant eye injuries, most frequent of which are corneal abrasions. If this examination is not done promptly, it can become increasingly difficult later on due to developing edema.

Radiological examination

Basic skull projections are useless in diagnosing ZMC fracture. An occipito-mental (Waters) view can give some initial orientation, but not full appreciation of the detailed fracture pattern. In the presence of clinical symptoms, a CT scan should always be done, not only axial, but also coronal slices, or coronal reconstructions, if positioning of the patient for coronal scans is not possible due to associated injuries (especially cervical spine injury). Recently, introduction of helical CT scanners that allow for reformatting in any



Fig. 40.46 Edema, periorbital hematoma, and temporal subconjunctival suffusion develop shortly after ZMC fracture.



Fig. 40.47 Downward slant of the palpebral fissure in the horizontal plane as a consequence of displacement of lateral palpebral canthus.

chosen level, has eliminated this problem (Fig. 40.48). 3D reconstructions are extremely helpful for appreciation of the type and degree of dislocation (Fig. 40.49). Radiological examination should answer the following questions:

1. Which articulations are dislocated and which only bent?
2. Is there any comminution of fracture lines?
3. Is there a loss of lateral or anterior projection?
4. What is the status of the orbital walls and orbital volume?

In evaluation of dislocations it can be useful to compare fractured ZMC with the mirror image of uninjured side.

Treatment

The goal of treatment is to establish preinjury anatomy and stabilize fragments in their anatomic positions so that the bone healing can take place. While this goal can be accomplished and its accuracy ascertained with relative ease in most facial fractures, in the case of ZMC it can be quite challenging. The reason rests with the 3D complexity of the zygomatic bone and difficulty in accessing some of its facets.

Proper reduction of ZMC fracture should result in precise alignment of six areas: zygomatic arch, supero-lateral orbital margin, lateral orbital wall, orbital floor, inferior orbital margin, and zygomatico-alveolar crest. Theoretically alignment of any three points of a 3D structure should result in other areas being aligned as well. However, some of these areas tend to

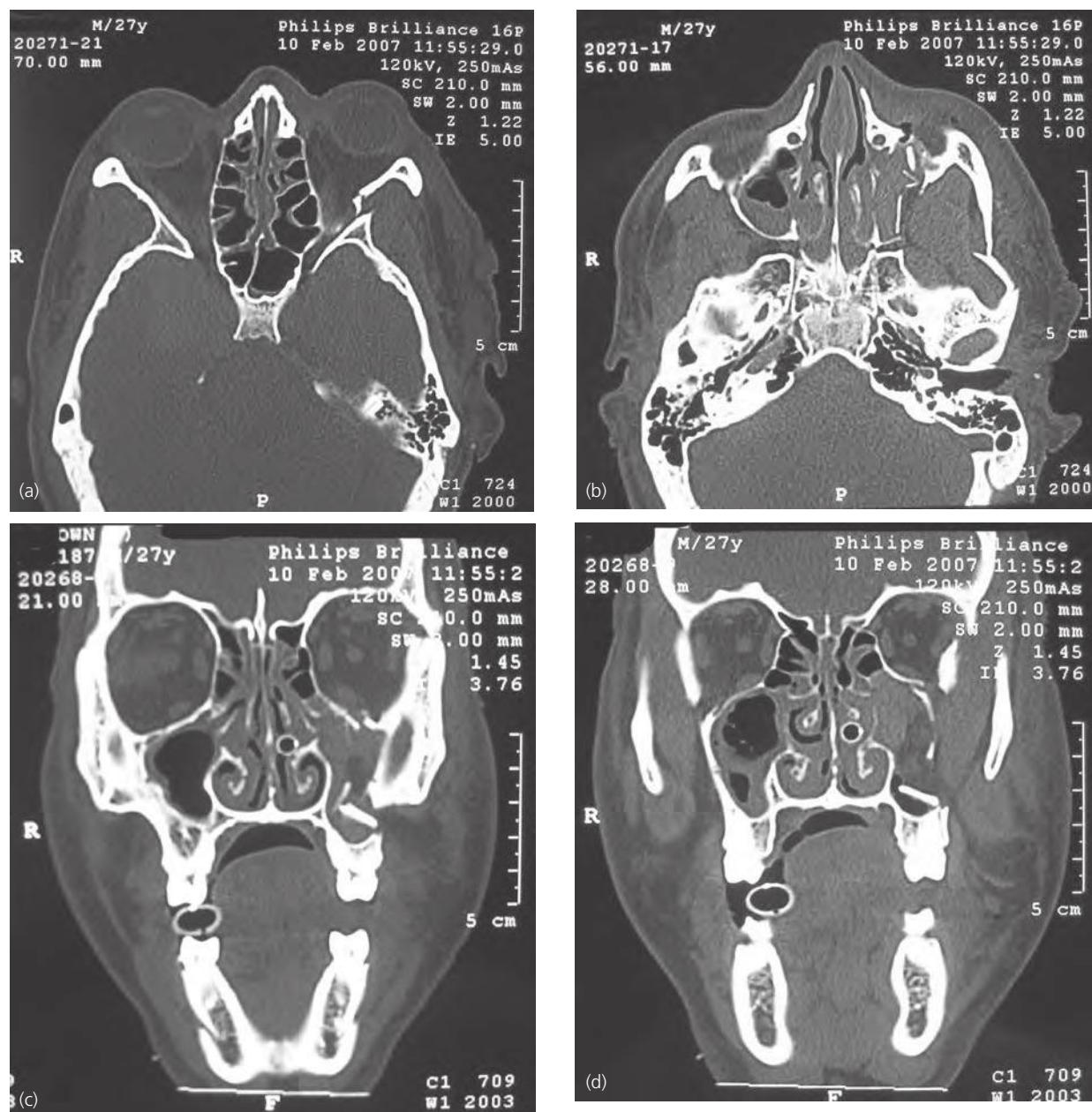


Fig. 40.48 CT examination of comminuted ZMC fracture. (a) Displacement of lateral orbital wall with interfragment. (b) Comminution of walls of maxillary sinus and depressed M-type fracture of zygomatic arch. (c) Depression and medial displacement of body of zygomatic bone, comminution of maxillary sinus wall, trap-door fracture of orbital floor. (d) Bone fragment impinging on lateral rectus muscle.

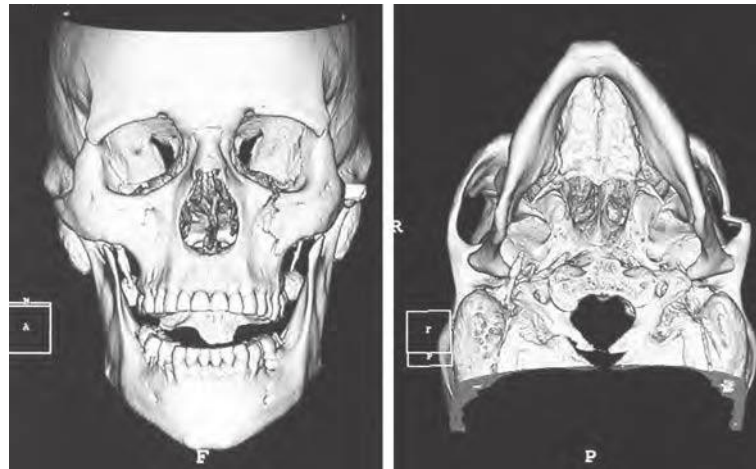


Fig. 40.49 3D CT reconstruction gives a good idea about the overall fracture pattern and type of dislocation but is insufficient for evaluation of the internal orbit. (a) Posterior rotation of the zygomatic body. (b) M-type fracture of the zygomatic arch.

be comminuted, making them unsuitable for verifying proper overall zygoma position. They are mostly the inferior orbital margin, the orbital floor, and also very often the zygomatico-maxillary crest. Other areas are rarely comminuted, but are accessible only through time-consuming coronal incision. They are the zygomatic arch and the lateral orbital wall. The last area, the zygomatico-frontal suture, is easily accessible and rarely comminuted, but it is also the least reliable indicator of proper reduction, often being the pivotal point around which the whole complex rotates. Minor dislocation here can result in major discrepancies in distant fracture lines.

After proper anatomic reduction the question of adequate fixation arises. This has been a controversial topic for a long time.¹¹⁶ Before introduction of miniplate osteosynthesis, ZMC fractures were mostly treated by the Gillies approach or transcutaneous bone hook reduction and surgeons relied on interlocking of fracture lines for fixation. In cases of unstable fractures, a wire suture was employed, and comminuted fractures were supported by external pin, antral strut, packing or balloon. Immediate radiological verification of proper reduction was usually not done. Subsequent post-treatment deformities used to be ascribed to postreduction dislocation of the zygomatic bone by the pull of the masseter muscle. However it can be argued that perhaps proper reduction was never achieved, which fact became obvious only after edema had completely subsided several weeks later.¹¹⁷ Even now some authors consider postoperative X-ray examination unnecessary¹¹⁸ in contrast with other reports¹¹⁹ on intraoperative CT imaging leading to immediate correction of unsatisfactory reduction of fracture.

Currently the need for stable fixation of the majority of ZMC fractures is recognized, with the exception of fractures without comminution of any fracture line. The number of sites that need to be plated and the strength of applied hardware, however, are still a matter for disagreement. The recommendations vary

from “minimized treatment” by single miniplate¹²⁰ to plating every accessible fracture line.¹²¹

Another controversy regards orbital exploration. Again various authors differ widely in their recommendations, from routine orbital exploration to orbital exploration only done in “cases of primary diplopia”.¹²⁰ Orbital walls are involved to some degree in every true ZMC fracture. This involvement varies from a simple fracture line resembling a crack to complete disruption with soft tissue prolapse, where the need for exploration and repair is obvious (Fig. 40.50). It is important to think not only about the pretreatment situation, but also about what happens to orbital walls during reduction. In most cases a simple orbital fracture, although dislocated, will be reduced together with other fracture lines even without orbital exploration.¹²² In some cases, however, the reduction maneuver can lead to soft tissue entrapment or further fragmentation and resultant insufficient reduction and restoration of orbital volume.

It is obvious from previous considerations that ZMC fractures differ widely in severity and different treatment approaches are necessary for their rational and successful management. Fractures without any comminution and without other dislocation than *ad axis* (fragments must remain in contact) in areas of the zygomatic arch and zygomatico-frontal suture can be treated by closed reduction (transcutaneous bone hook, screw or elevatorium through Gillies approach). If the fracture is unstable after reduction or cannot be reduced, the authors use an intraoral approach first to obtain reduction and stabilize the fracture by plating the zygomatico-alveolar crest. The intraoral approach is an obvious choice, because it has minimal morbidity and leaves no visible scar. The zygomatico-alveolar crest is a good place to evaluate reduction and apply a miniplate, which will act against the masseter pull and prevent tilting of the zygomatic bone downward and inward.¹²² Reduction at the inferior orbital margin can also be checked from this approach.

If the infraorbital margin is badly displaced or comminuted, or orbital exploration is required, either conjunctival or sub tarsal incision is indicated. Another option for simultaneous repair of supero-lateral and inferior orbital margins and orbital floor is conjuncti-

val incision with lateral canthotomy, which can provide access to both sites.⁸³ Exploration and repair of three sites is sufficient for precise reduction and stabilization of the majority of displaced fractures (Fig. 40.50).

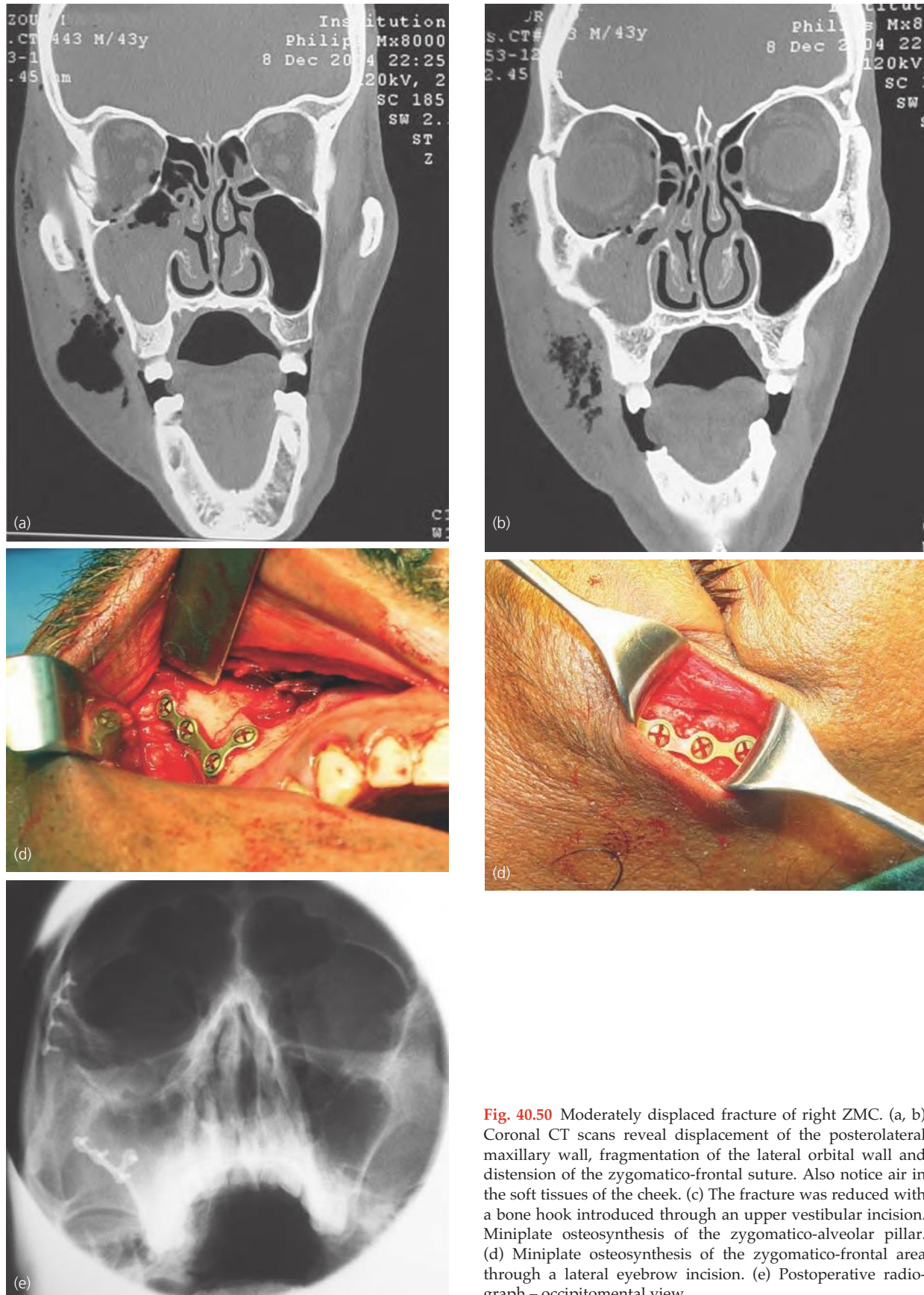


Fig. 40.50 Moderately displaced fracture of right ZMC. (a, b) Coronal CT scans reveal displacement of the posterolateral maxillary wall, fragmentation of the lateral orbital wall and distension of the zygomatico-frontal suture. Also notice air in the soft tissues of the cheek. (c) The fracture was reduced with a bone hook introduced through an upper vestibular incision. Miniplate osteosynthesis of the zygomatico-alveolar pillar. (d) Miniplate osteosynthesis of the zygomatico-frontal area through a lateral eyebrow incision. (e) Postoperative radio-graph – occipitomental view.

The exception is the displaced ZMC fracture with loss of anterior projection due to either overlapping (*dislocatio ad latus cum contractionem*) or laterally buckling fracture of the zygomatic arch. Such a fracture is usually the result of high-velocity injury and is accompanied by comminution in other sites making precise reduction difficult. In such cases the authors opt for a coronal incision that will provide excellent access to the zygomatic arch and enable its reconstruction and thus proper anterior projection of zygomatic bone.¹²³ Naturally the supero-lateral orbital margin is repaired from the same approach and, if necessary, the lateral orbital wall can be exposed after stripping the anterior attachment of the temporalis muscle to assist in the precision of the reduction.

Reconstruction of the orbital floor is mandatory in all cases of comminution, soft tissue prolapse to the maxillary antrum or increase in orbital volume. The authors' preferred material for this reconstruction is titanium mesh (Fig. 40.51). A detailed discussion of orbital repair is provided in Chapter 41.

Fractures of the orbit

The treatment of these fractures is covered in Chapter 41 in this book.

Subfrontal fractures

Le Fort II fracture

Fracture of Le Fort II type combines characteristics of the Le Fort I and NOE fractures. If present in bilateral symmetrical form, it resembles a pyramid. The fracture line crosses the nasal bridge, runs through the fronto-maxillary suture area into the orbit, continues posterior to the medial canthus attachment across the medial orbital wall to the orbital floor and from there leaves the orbit on its inferior margin near the zygomatico-maxillary suture to continue along this suture to the pterygo-maxillary suture (Fig. 40.52). Lower parts of the pterygoid processes of the sphenoid bone are broken off and attached to the central fragment.

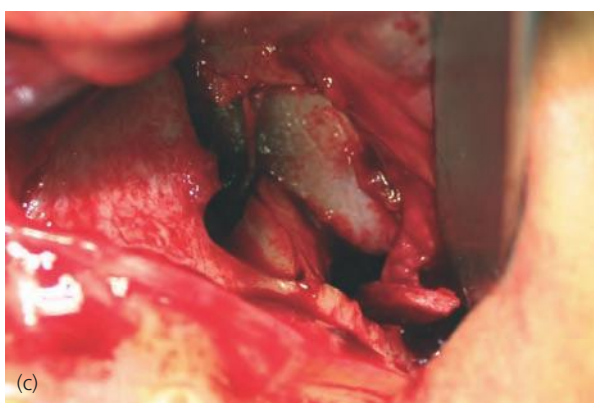


Fig. 40.51 Severely displaced ZMC fracture with loss of lateral and anterior projection. (a) Coronal CT scan reveals narrowing of left orbit, medial displacement of body of zygomatic bone and collapse of zygomatico-alveolar pillar. (b) Axial CT scan showing severe posterior and medial displacement of zygomatic bone with fragmentation of anterior and postero-lateral maxillary walls. (c) Exposure through an upper vestibular incision: fragmentation and collapse of zygomatico-alveolar buttress. (d) Exposure through sub tarsal incision: comminution of inferior orbital rim. (*cont'd*)

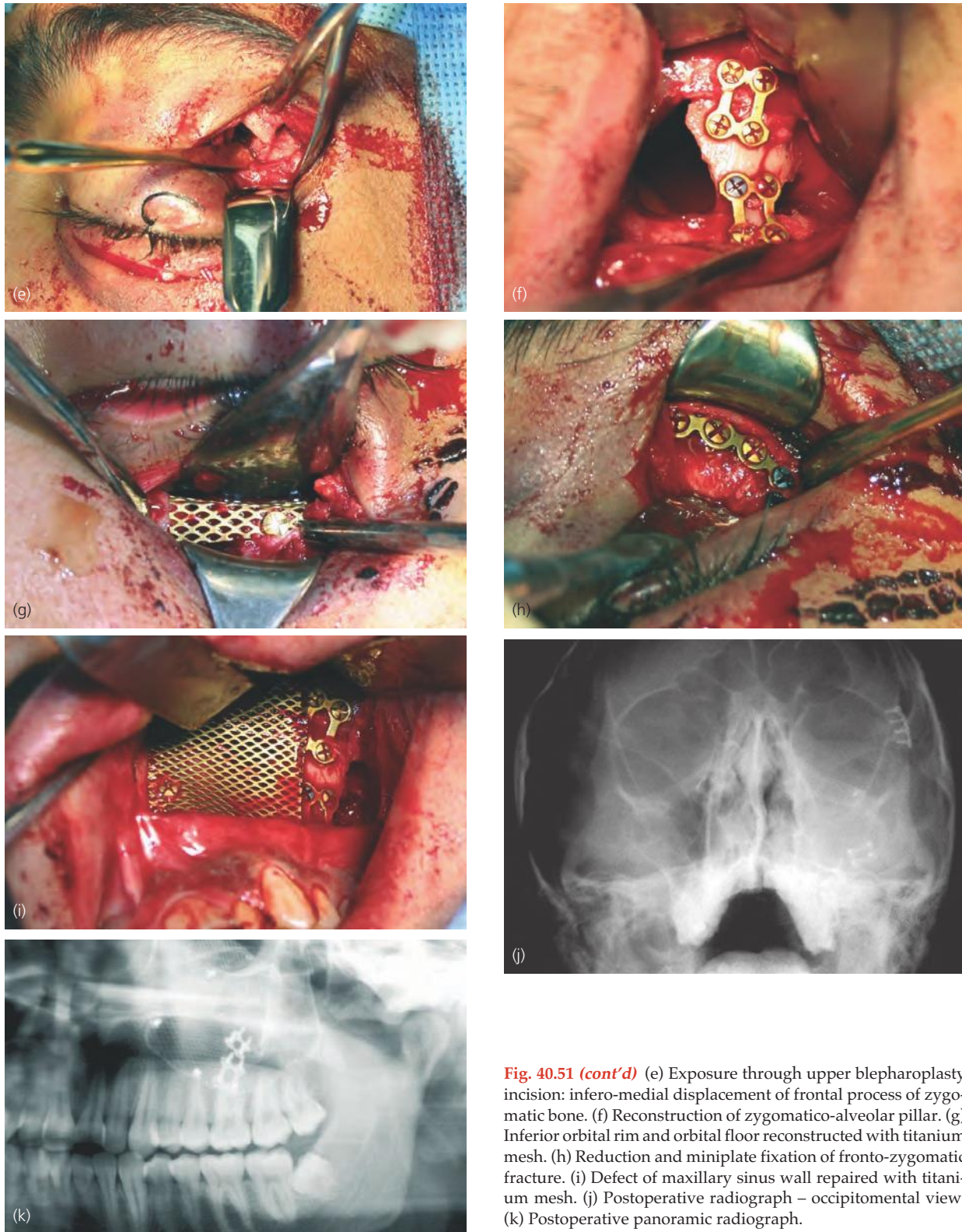


Fig. 40.51 (cont'd) (e) Exposure through upper blepharoplasty incision: infero-medial displacement of frontal process of zygomatic bone. (f) Reconstruction of zygomatico-alveolar pillar. (g) Inferior orbital rim and orbital floor reconstructed with titanium mesh. (h) Reduction and miniplate fixation of fronto-zygomatic fracture. (i) Defect of maxillary sinus wall repaired with titanium mesh. (j) Postoperative radiograph – occipitomental view. (k) Postoperative panoramic radiograph.

The fracture can be dislocated to various degrees, mostly dorso-cranially, like the Le Fort I fracture. This results in similar occlusal disturbance. Due to the upper component of fracture the nasal bridge is deformed and the medial and inferior walls of the orbit are disrupted.

Not infrequently the Le Fort II fracture is combined with a sagittal or parasagittal fracture of the hard palate.

Symptoms

Patients regularly present with nasal bleeding and eyelid edema and suffusion. Palpation of the nasal bridge and inferior orbital margins may reveal a step or crepitus. When the edema subsides, patients with major displacement can have a depressed central facial area – so-called dish face. Intraorally there is edema and hematoma in the upper vestibule and anterior open bite. Steps can be palpated on the zygomatic

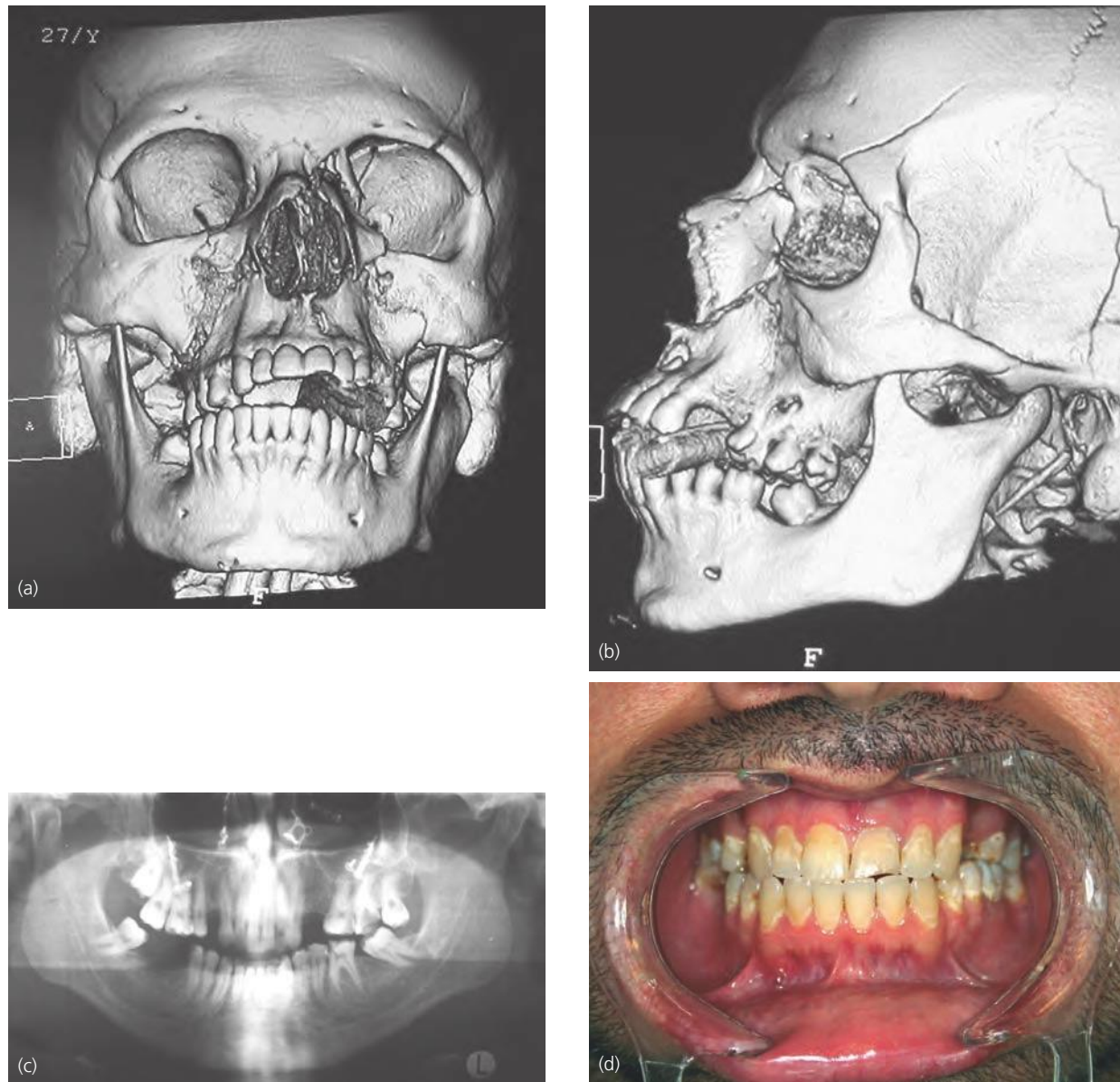


Fig. 40.52 Le Fort II fracture combined with left NOE and ZMC fracture. (a, b) 3D CT reconstructions show the pyramidal pattern of the maxillary fracture, comminution of the left medial orbital wall, undisplaced fracture of the left frontal bone, temporal bone and zygomatic arch; note displacement of the maxilla with anterior open bite. (c) Post-treatment OPG showing reconstruction of four maxillary buttresses, as well as inferior orbital rim. (d) Normal occlusion after treatment.

matico-alveolar crests. The upper dental arch may be mobile, and during its manipulation pathologic movement may be palpated over the nasal bridge and inferior orbital margins. However, more often than in Le Fort I fracture, the central fragment is firmly wedged in its new position and cannot easily be moved.

Radiological examination

CT scans, both coronal and axial slices, are necessary to fully evaluate the whole extent of fracture. 3D reformatting is extremely useful for overall appreciation of the fracture pattern.

Treatment

As in Le Fort I fractures, the key to proper reduction is occlusion, therefore treatment commences with

application of arch bars followed by mobilization of the central fragment. This can be quite difficult, because of the great complexity of the fracture line and the inaccessibility of some of its areas. Use of disimpaction forceps is frequently unavoidable. Proper mobilization is crucial for restoration of anterior facial height and prevention of postoperative anterior open bite. When passive positioning of the maxilla is not possible, a concomitant osteotomy may be the last resort to restore proper occlusion.¹²⁴

After the central fragment has been fully mobilized and central occlusion secured by maxillo-mandibular fixation, fracture lines are exposed from intraoral and lower eyelid approaches and the whole complex is rotated cranially with the mandibular condyles properly seated. This should result in alignment of the zygomatico-alveolar crests and infraorbital margins,

which are consecutively plated. In the absence of gross comminution there is no need to expose the nasal bridge that will be satisfactorily reduced. If there is comminution of NOE complex, it must be repaired through a coronal approach. Orbital walls should be explored in such a case and any large defects resulting from the fracture or its reduction should be repaired.

Le Fort III fracture

Fracture of Le Fort III type is sometimes also referred to as craniofacial separation, because it effectively separates the midfacial skeleton from the neurocranium. The fracture line divides the nasal bones and spina nasalis of the frontal bone, breaks the nasal septum, crosses into the medial wall of the orbit behind the posterior lacrimal crest, continues through the orbital processes of maxilla and zygomatic bone into the inferior orbital fissure, separates the zygoma from the ala major of the sphenoid, and runs through or next to the frontozygomatic suture. Separation is completed by fracture of the zygomatic arches and pterygoid plates of the sphenoid bone well above the pterygo-maxillary suture.

The pure pattern of the Le Fort III fracture is found only in a minority of cases; the midfacial complex is usually comminuted to some extent and other fracture patterns, like Le Fort I or Le Fort II, ZMC fracture or NOE fracture can be also identified. The fracture can be also complicated by paramedial split of the maxilla and palatal bone.

Symptoms

Patients quickly develop typical balloon-like edema of the face, binocular eyelid hematomas, and subconjunctival suffusions (Fig. 40.53). Nasal bleeding is common. The midfacial complex can be displaced to different extents, which can result in occlusal disturbance, usually anterior open bite and/or reversed



Fig. 40.53 Facial appearance of patient with Le Fort III fracture.

overjet. On manipulation with the dental arch, pathologic movement can be elicited and palpated over the nasal bridge and supero-lateral orbital margins.

Radiographic examination

Basic skull projections are useless in detection of this type of fracture. Only the occipito-mental (Waters) view of the skull can discover bilateral disruption of the zygomatic arches and superolateral orbital rims. Axial and coronal CT scans with 3D reconstruction are necessary to evaluate fully the pattern of the fracture, especially the extent of disruption of the fronto-nasal area.

Treatment

Restoration of proper occlusion is one of the main aims of treatment, therefore arch bars are first applied to the dental arches. Surgical access is through coronal incision that provides unimpeded exposure of all areas of alignment and osteosynthesis: zygomatic arches, supero-lateral orbital rims and fronto-nasal suture (Fig. 40.54). The midfacial complex must be properly reduced and this requires its mobilization via use of disimpaction forceps and intermaxillary fixation. Concomitant mandibular fractures are reduced and plated first. The maxillo-mandibular complex is then rotated cranially with properly seated condyles until full contact is achieved with the cranial base. At least the zygomatic arches and supero-lateral orbital rim should be plated. The frontonasal area is a less important fixation point because of inherent weakness of the underlying NOE complex, which often leads to comminution.

Fractures of the frontal bone

The squama of the frontal bone formally belongs to the neurocranium and forms underlying skeletal support of the upper facial third. However from a practical point of view the frontal bone is often involved in midfacial fracture patterns¹²⁵ as it is intimately connected to midfacial structures through the fronto-nasal, fronto-ethmoidal, fronto-maxillary, and fronto-zygomatic sutures. The frontal bone also forms the superior wall of the orbit.

The frontal bone is a strong bone with a double cortical layer and an interstitial, well developed diploe layer. Its weakness is the presence of the frontal sinus, which is developed to variable extent in different individuals. The anterior wall of the sinus can be broken in isolation by a direct mechanism (Fig. 40.55) or it can be broken and displaced together with large fragments. In these cases the posterior wall of the sinus is broken and displaced to the same degree (Fig. 40.56). However the posterior wall can be sometimes affected more than the anterior wall, or even fractured while the anterior wall remains intact. This phenomenon is explained by elastic deformation of bone and transmission of the traumatic force to the thinner posterior sinus wall. (Fig. 40.57).

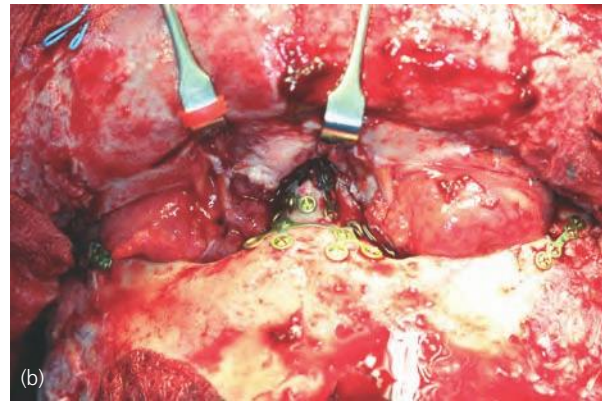
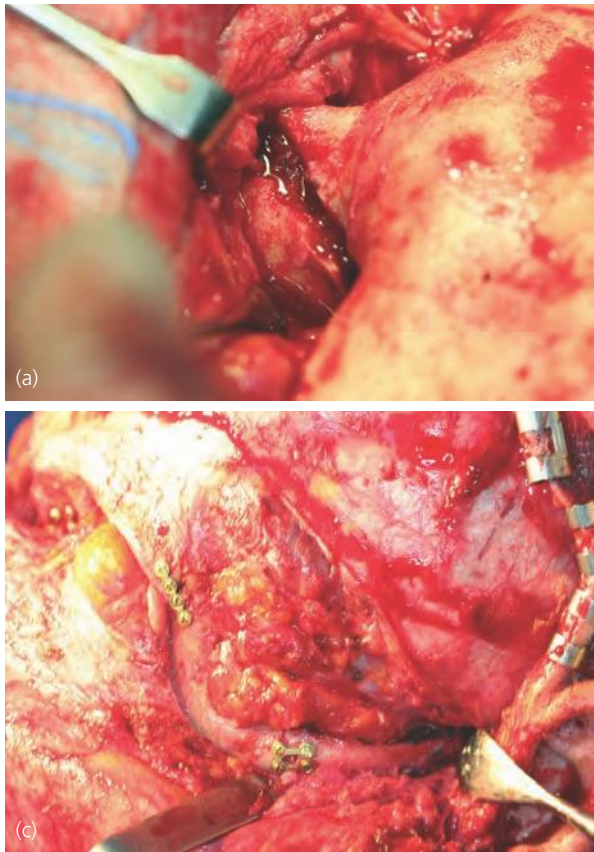


Fig. 40.54 Le Fort III fracture. (a) Fronto-nasal fracture exposed through a coronal incision. (b, c) Miniplate fracture repair in areas of fronto-nasal and zygomatico-frontal sutures, as well as zygomatic arch.

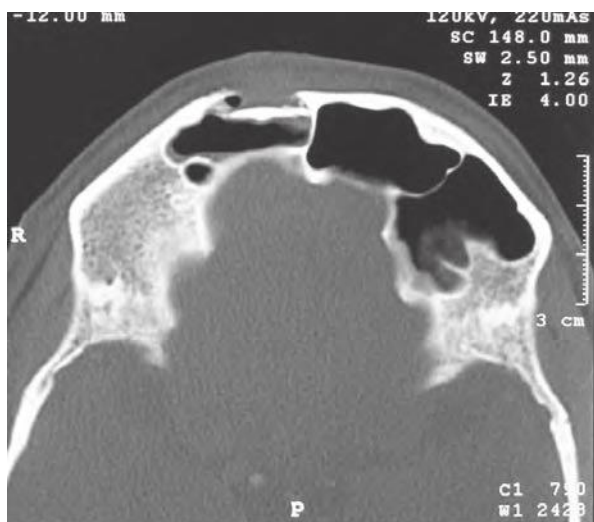


Fig. 40.55 Fracture of anterior wall of frontal sinus. Coronal CT.



Fig. 40.56 Severely displaced frontal bone fracture involving both anterior and posterior walls of frontal sinus. Coronal CT.

Symptoms

Frontal bone fractures are mostly caused by high-velocity mechanisms and consequently are often accompanied by intracranial injuries, such as brain commotion and contusion or intracranial bleeding, which can lead to alteration of consciousness.

Locally bruises, excoriations or lacerations can be seen. There is often bleeding from the nose. Gross displacement of fragments can lead to forehead deformity (Fig. 40.56), which is later masked by developing edema. Displaced fractures can be accom-

panied by disturbance of sensitive innervation in the areas of the supraorbital and supratrochlear nerves. Orbital ecchymosis and subconjunctival hematomas develop if the fracture extends into the orbital walls. In such cases damage to orbital structures and variety of related ocular symptoms may be present. Dislocated fractures are frequently associated with dural tears, which may or may not result in nasal cerebrospinal fluid (CSF) leak. Close cooperation with the neurosurgeon and ophthalmologist is therefore of utmost importance.

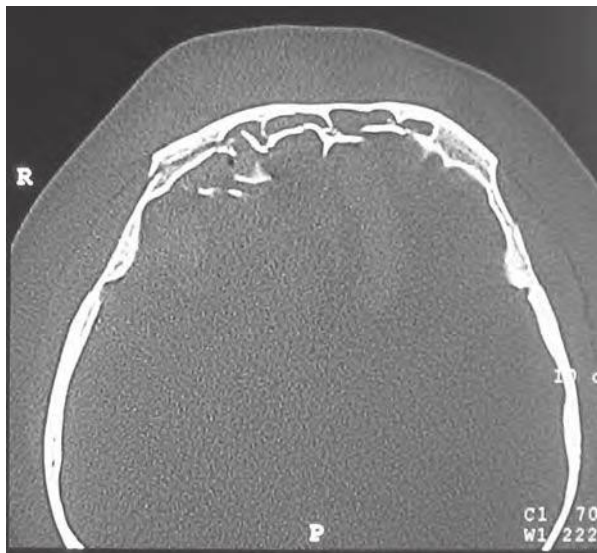


Fig. 40.57 Infrequent case of posterior wall of frontal sinus fracture with anterior wall intact.

Radiological examination

For a fully conscious patient with suspected skull fracture, basic X-ray projections of the skull may be sufficient. If the patient is unconscious or basic projections have roused suspicion of fracture, a CT scan of the brain and skull should be performed.¹²⁶ Axial slices give a good picture of the anterior and posterior walls of the frontal sinus, but coronal scans or reconstructions are necessary to evaluate the orbital roof and medial wall, and the fronto-nasal area. Coronal scans can also detect fractures of the cribriform plate of the ethmoidal bone, which usually run in an antero-posterior direction. In the case of comminuted fractures it is very useful for treatment planning to have 3D reconstruction.

Simple frontal bone fractures

Simple frontal bone fracture means fracture without displacement. For practical purposes, fractures with displacement less than the thickness of the outer or inner cortical layer are regarded as undisplaced. Because the frontal bone is not a site of attachment of any powerful muscles, there is no risk of secondary dislocation of fragments. The presence of dural tears is also improbable in undisplaced or minimally displaced fractures. Such fractures do not require any treatment except antibiotic cover if frontal sinus hematoma has developed.

Compound frontal bone fractures

Frontal bone fractures resulting from high-velocity injury tend to be comminuted with displacement of fragments that are mostly depressed. Rarely stress forces produced during impact can lead to outward buckling of fragments. Fractures can be associated with lacerations of the skin and underlying soft tissues and can thus communicate through laceration

with the external environment and become contaminated. Sometimes foreign bodies can be found in lacerations or even embedded in the fracture. Displaced fractures involving the frontal sinus must be considered contaminated, even if they do not communicate with any external wound, because of the continuity of the mucosal lining with the nasal cavity. Displacement of fractures intracranially can result in laceration of the dura mater with CSF leak, either directly through the wound or through the frontal sinus and fronto-nasal ostium to the nose. Rarely brain tissue can prolapse into the frontal sinus. Perforation of the dura mater violates the anatomic barrier preventing spread of infection from the nose and paranasal sinuses intracranially. If not repaired, whether or not there is a continuous CSF leak, the patient is at life-long risk of intracranial infectious complications like meningitis, encephalitis or brain abscess.¹²⁷

The frontal sinus is lined with respiratory mucosa containing mucinous cells. Therefore there is a potential for mucus retention and development of mucocele if the drainage of the sinus into the nasal cavity has been interrupted by displaced bony fragments.¹²⁸ Consequently, in such situations, drainage should be either restored or the sinus should be eliminated.

Treatment

Treatment of compound frontal bone fractures has four goals:

1. To seal dural perforations and isolate the intracranial space from potential sources of infection.
2. To ascertain patency of the fronto-nasal ostium, or to eliminate the frontal sinus if this patency cannot be maintained.
3. To restore the integrity of the frontal bone so that the brain is sufficiently protected from future insults.
4. To restore the contour of the forehead.

The fractured frontal bone is exposed via a coronal flap. During its dissection it is necessary to consider whether a pericranial flap will be required and proceed with a pericranial incision accordingly. If the frontal bone fracture is a component of a more complex midfacial fracture and some periorbital incisions are needed for repair, these should be performed first, before a surgical periorbital edema develops. Only in exceptional cases can an existing traumatic wound be used to address an isolated fracture of the anterior frontal sinus wall.

If only the anterior wall of frontal sinus has been broken and displaced, fragments can usually be lifted and removed with a pointed periosteal elevator. This makes the sinus cavity accessible for revision and debridement. Blood clots, loose bone fragments, and non-viable tatters of mucosa are removed, while healthy sinus mucosa is preserved. Consequently the fragments are reassembled, repositioned, and

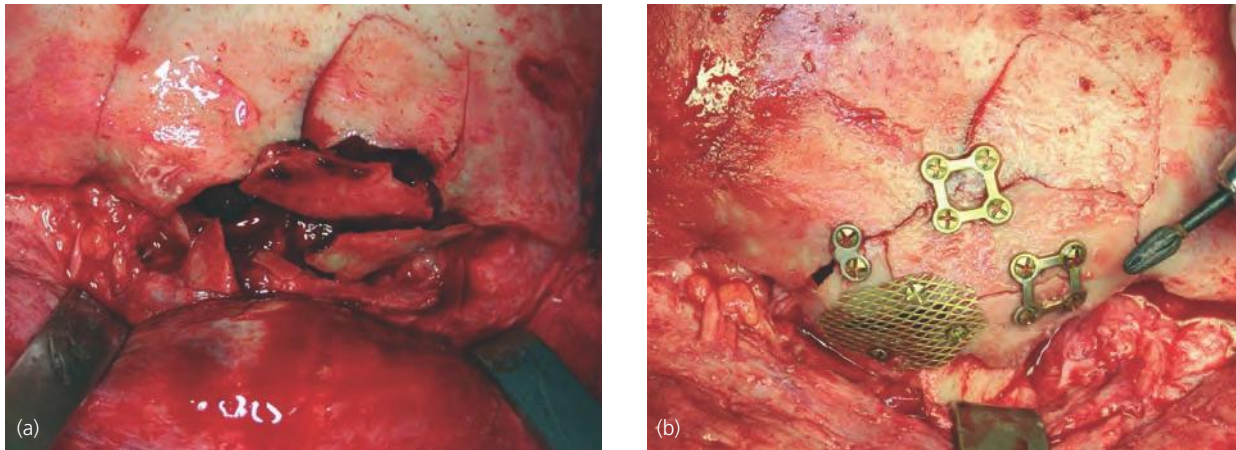


Fig. 40.58 Exposure of frontal bone fracture through coronal incision. (a) The supraorbital nerves were dissected free from the supraorbital foramina. Severe comminution of the glabellar area of the frontal bone. (b) Reduction of fragments and miniplate osteosynthesis. Missing bone replaced by titanium mesh. Minor irregularities are smoothed by a large bur.

stabilized with titanium plates or mesh and screws (Fig. 40.58).¹²⁹ Some trimming of fragment margins may be necessary to achieve reduction. Fragments too small to be engaged by hardware are discarded. Resulting gaps can be easily reconstructed with titanium mesh or left alone if not larger than 5 mm.¹³⁰

In cases where the full thickness of the frontal bone has been broken and displaced, fragments can be safely lifted only if buckled out. Attempts at prying out impacted full-thickness fragments can lead to further dural laceration, sagittal sinus or even brain injury. In such a situation low bi-frontal craniotomy encircling the whole fracture pattern is necessary. This provides wide access to the floor of the frontal sinus, the frontonasal ostium and the orbital roofs. It also enables the neurosurgeon to perform careful revision and repair of the dura mater of anterior cranial fossa. Craniotomy is also necessary in rare cases of displaced fractures of the posterior sinus wall without anterior wall involvement.

Management of injured frontal sinus and fronto-nasal ostium

Restoration of patency of the fronto-nasal ostium was traditionally attempted by insertion of a transnasal tube into the sinus, which was kept in place for several weeks or even months to serve as a stent for healing and re-epithelization.¹³¹ Results of such attempts were, however, often reported as disappointing due to scarring and resulting stricture of the neo-formed communication. On the other hand there are recent reports that the regeneration capacity of mucosal lining is sufficient to keep the communication patent even without stenting, if the bone frame was anatomically reduced.^{132,133}

Elimination of frontal sinus can be achieved by either obliteration or cranialization.^{126–130} In the case of obliteration, the mucosal lining of the sinus must be completely removed and peripheral osteotomy of sinus walls performed with a rotational instrument to get rid of microscopic extensions of the mucosa lining

into so-called Breschet pits. Unfortunately this can sometimes be very difficult if not impossible, especially in patients with extremely pneumatized sinuses that can extend far into the orbital roofs. These extensions are narrow and have egg-shell thin walls making the above described procedure unfeasible. After proper mucosal debridement the resulting cavity is filled by autogenous material: bone, muscle, fascia or fat grafts were used for the purpose. Again, in cases of extremely pneumatized sinus the necessary amount of material can be a hindrance. The large body of non-vascularized material can become infected. A better option seems to be spontaneous bone regeneration, where only the ostium of the debrided sinus is plugged by a graft and the cavity is left to the process of filling with blood and its subsequent organization and ossification.¹²⁶

Cranialization of the frontal sinus is a method of eliminating the sinus by removing its posterior wall and thus making it an extension of cranial cavity (Fig. 40.59). The cranialization procedure requires craniotomy and is indicated in cases of posterior sinus wall disruption. Naturally the mucosal lining has to be completely removed, including peripheral osteotomy, and the fronto-nasal ostium must be plugged. If dural tear was present, the intracranial space should be further isolated from the nose by an additional layer of vascularized pericranial flap introduced through a lower craniotomy slit.¹³⁴

When reconstruction of the orbital roofs is needed, this must be done before insertion of the pericranial flap. The same is true for reconstruction of the supra-orbital rims and NOE complex. After insertion of the pericranial flap these structures will no longer be accessible. Details of sequencing of panfacial fracture repair will be addressed in Chapter 41.

After completion of above-mentioned steps the fractured fragments and parts involved in the craniotomy are either reassembled on a side table or repositioned into the craniotomy defect in a jigsaw puzzle manner and secured by plates and screws. This can

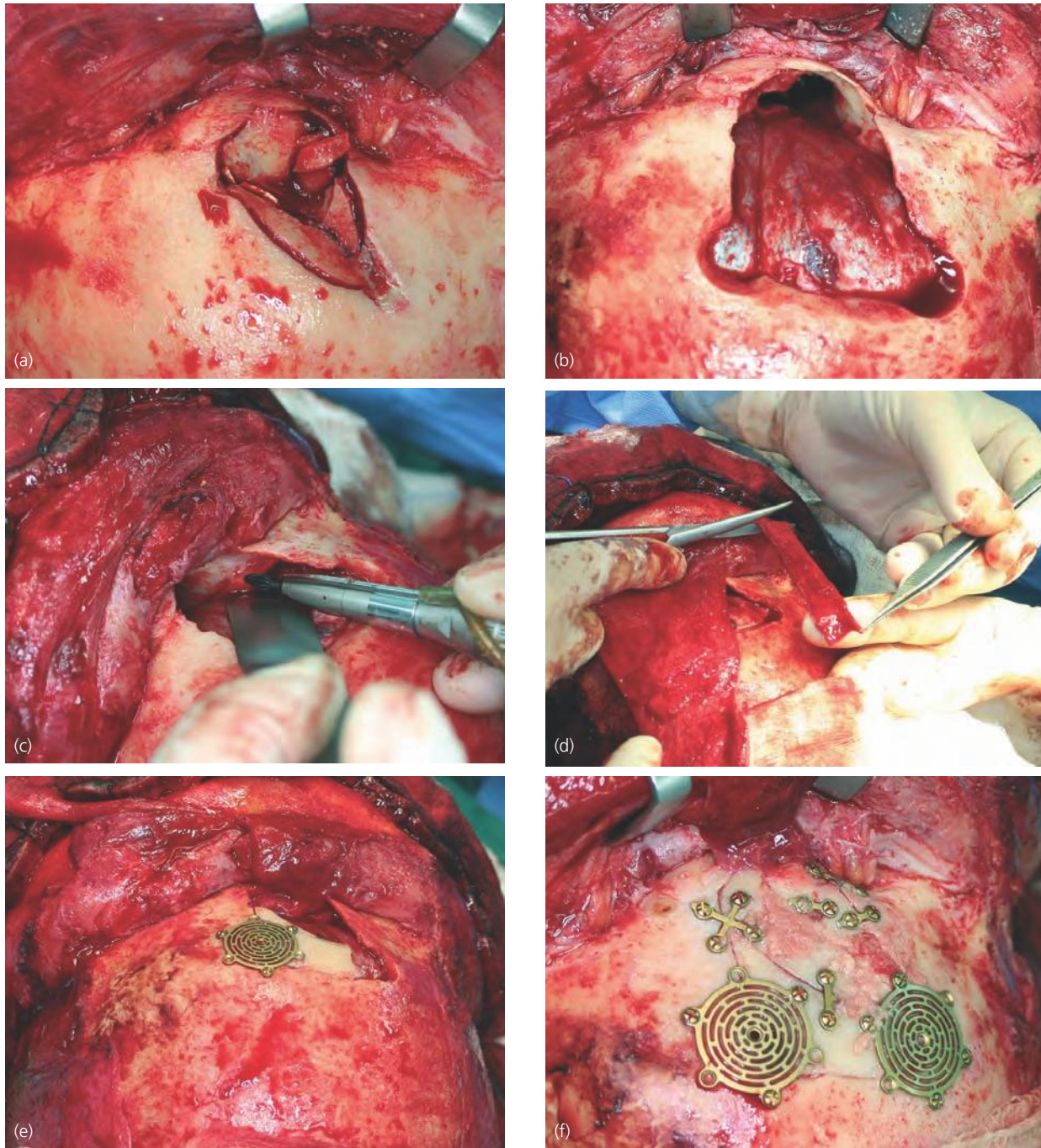


Fig. 40.59 Comminuted frontal bone fracture with involvement of posterior wall of frontal sinus. (a) Bird's eye view of glabelar area exposed through a coronal incision. Note freed supraorbital nerve and whitish foreign bodies. (b) Exposed dura mater after temporary removal of fragments and cranialization of frontal sinus. (c) Sinus mucosa is completely removed and peripheral osteotomy performed with a large bur. (d) A strip of pericranium is used to obliterate the fronto-nasal duct. (e) A pericranial flap is placed over the obliterated frontonasal duct to provide an additional isolating layer. (f) Reconstruction completed. Small irregularities are covered with bone dust and bur holes are covered with titanium plates to protect the underlying dura mater and brain.

be surprisingly difficult in the presence of numerous fragments, therefore marking of pieces and drawing their layout, or digital photography, is advisable before their removal. Sometimes a considerable amount of bone can be missing, either due to severe comminution or more often due to a previous urgent neurosurgical procedure. Unfortunately some neurosurgeons, who are not regular members of interdisciplinary teams, have a tendency to discard "useless"

bone fragments. The proper thing to do is to keep such fragments that cannot be left in place in a subcutaneous abdominal fat pocket for later reinsertion, of course after proper decontamination.

Larger defects are reconstructed by autogenous calvarial bone grafts or split ribs, smaller defects can be covered by titanium mesh. The coronal flap is then carefully repositioned and sutured in proper layers as described earlier.

Biodegradable screws and plates in management of midfacial fractures

Although titanium miniplates are considered the state of the art osteosynthetic device in craniofacial surgery, some controversy still exists regarding whether they should be removed after the healing period or whether it is safe to retain asymptomatic miniplates and screws until the end of the patient's life.^{135,136} More importantly, some miniplates may become symptomatic and their removal is then mandatory. Removal rates vary widely between 5% and 40%.¹³⁷

Common reasons for removal of miniplates include palpable or visible hardware, loosening of screws, pain, infection, wound dehiscence with exposure of hardware, and secondary corrective procedures.¹³⁸ In the mandible, the main reasons for removal are infection and exposure, but in the midface it is visibility, especially in the zygomatico-frontal region.¹³⁹ Moreover special attention is given to removal of metallic hardware in children, in whom it is considered to be a possible factor influencing facial growth. Due to remodeling processes, miniplates become buried in sites of bone apposition and their later removal can be extremely difficult, if not impossible.

Another important factor is ease of accessibility of some application sites. Intraorally introduced miniplates can be removed without considerable morbidity even under local anesthesia. Removal of periorbital miniplates can be more problematic, where repeated surgical access can lead to unfavorable scarring. The most difficult situation arises if the zygomatic arch or the frontal bone miniplates need to be removed. This requires repetition of exposure via a coronal flap, if the hardware was not introduced through traumatic wounds and additional scars are to be avoided.

A possibility for avoiding these problems is to use biodegradable osteosynthetic devices that will be eliminated by natural processes of enzymatic hydrolysis and absorption. The history of development of biodegradable osteosynthetic devices goes back to the early 1970s¹⁴⁰ and today most manufacturers of craniofacial osteosynthetic hardware offer their own biodegradable system. An ideal biodegradable implant should have the following properties: adequate strength and rigidity kept for appropriate time after insertion; malleability; no inflammatory or toxic tissue response; full absorption after fulfilling its purpose; acceptable shelf-life; easy sterilization; and, last but not least, reasonable price. So far there are no products available that fulfill these criteria without reservations.

Using biodegradable implants in management of midfacial fractures has no major long-term adverse effects beyond the total material resorption time and complications associated with the biodegradable

material could be considered minor.¹⁴¹ However the authors have identified several limitations. Firstly the implants are bigger than titanium miniplates used in identical indications. This is because of the inferior mechanical properties of biodegradable materials compared with titanium, which are made up for by increased bulk of material. The titanium systems have significantly higher tensile strength and stiffness compared to biodegradable systems.¹⁴² Because holes in the implants are more widely separated, it is sometimes impossible to engage all small fragments of comminuted fracture. The screws lack cutting threads and therefore require tapping. This again makes fixation of small fragments difficult. Screw heads break easily if greater torque is applied. The plates are not malleable at room or body temperature and need heating to adjust their shape. This makes use of tin templates necessary with most systems. Plates and screws are transparent and therefore poorly visible in operating fields. This altogether leads to elongated operation time.

Currently the authors consider biodegradable plates especially suitable for fixation of non-comminuted frontal bone fractures and orbital reconstruction.^{143–144}

Surgical technique

After proper exposure and reduction of the fracture, the tin template of shape corresponding to the chosen miniplate is adapted to the bone surface by finger molding (Fig. 40.60). The miniplate and template are then held together at one end by a dedicated pair of tweezers and immersed in a bath of sterile water heated to the temperature recommended by the manufacturer (usually between 50 and 60°C) for the recommended time (about 20–30 seconds). After removal from the bath, the plate and template are held in the open air so that the template is lower and the softened plate can assume the template's shape by drooping along the curvature of the template. The fall in temperature will make the plate rigid again. If the shape of the plate is not satisfactory, the procedure can be repeated a limited number of times. A final small adjustment can be made in the site of application with pressure from a heated molding pen. Drill holes are then created by a slowly rotating burr and a tap is used to cut threads in the bone. Screws are inserted using a screwdriver with a torque-limiting device to prevent the screw head being twisting off.

Recently a method has been introduced where resorbable pins inserted in the bone are welded to the resorbable plates using ultrasonic welding.¹⁴⁵ The polymer is anchored in the bone without the need for tapping in the bone. It is claimed that besides shortening operating time this method also results in better stability because all the structures are welded together into one piece.

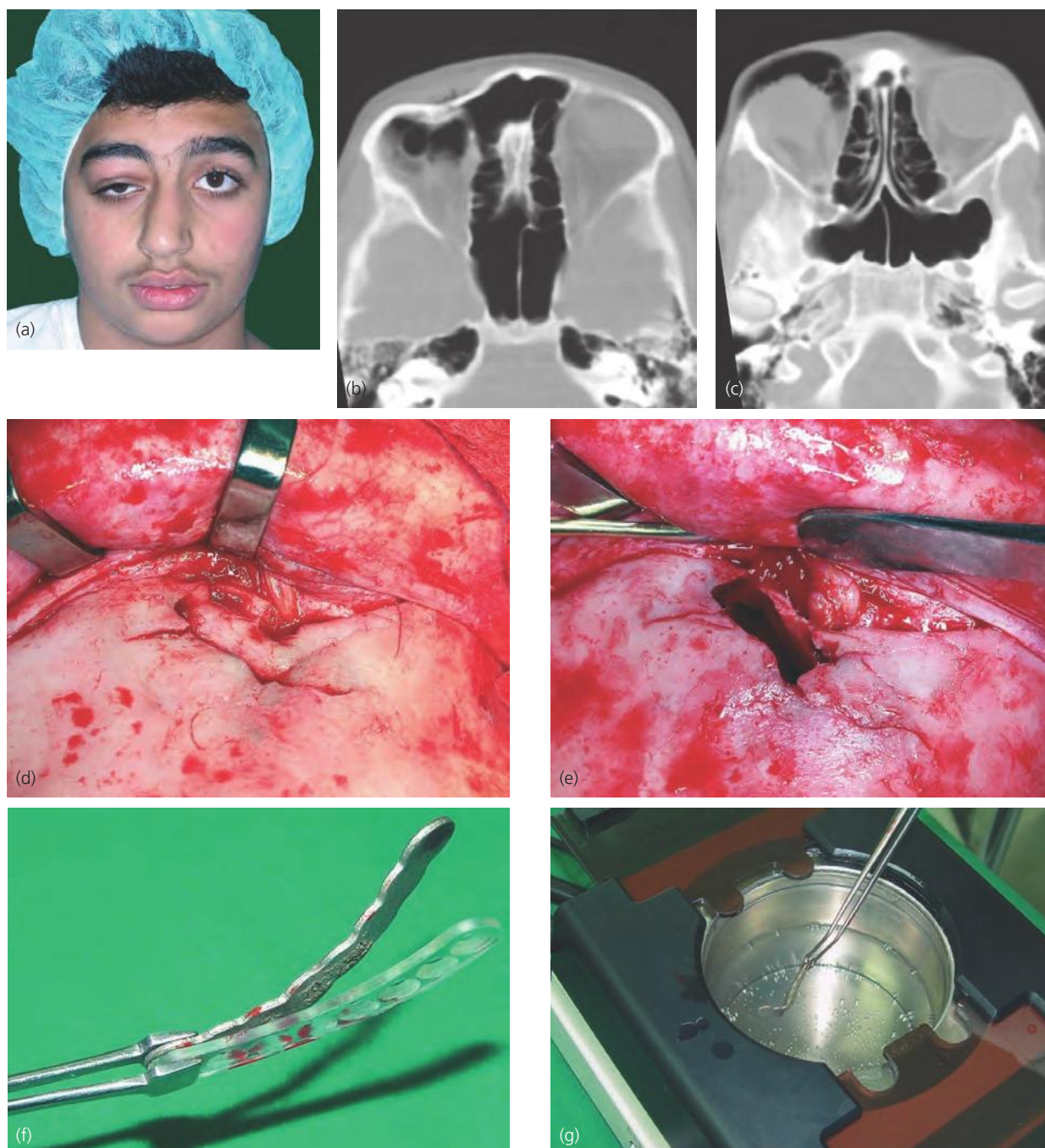


Fig. 40.60 Use of biodegradable miniplates in frontal bone fracture repair. (a) Clinical appearance of patient with marked deformity of supraorbital rim and upper eyelid edema. (b, c) Coronal CT scans showing depression of anterior wall of frontal sinus and soft tissue emphysema. (d) Fracture exposed through coronal incision. (e) Fragments temporarily removed to allow inspection of the frontal sinus and frontonasal duct. (f) Biodegradable miniplate is held together with template that was molded in fracture site. (g) Bath of hot sterile water softens the miniplate and allows it to assume the shape of the template.

Strategy of management of complex midfacial fractures

In this section general principles are described. The treatment of panfacial fractures is described in Chapter 41.

Timing of repair

There is no doubt that earliest possible repair of any injury, within a time frame of the first 24–48 hours

gives superb results and shortens the period of distress and suffering of a patient. This is easily achievable in simple injuries like facial lacerations, dentoalveolar injuries or even uncomplicated mandibular fractures; the patient must be medically fit, without any other injuries and the treatment facility must be well equipped, staffed, and organized for around-the-clock full performance. In practice all these conditions are only rarely met and comprehensive treatment of midfacial fractures, including those that are not complicated, often has to be postponed and staged.

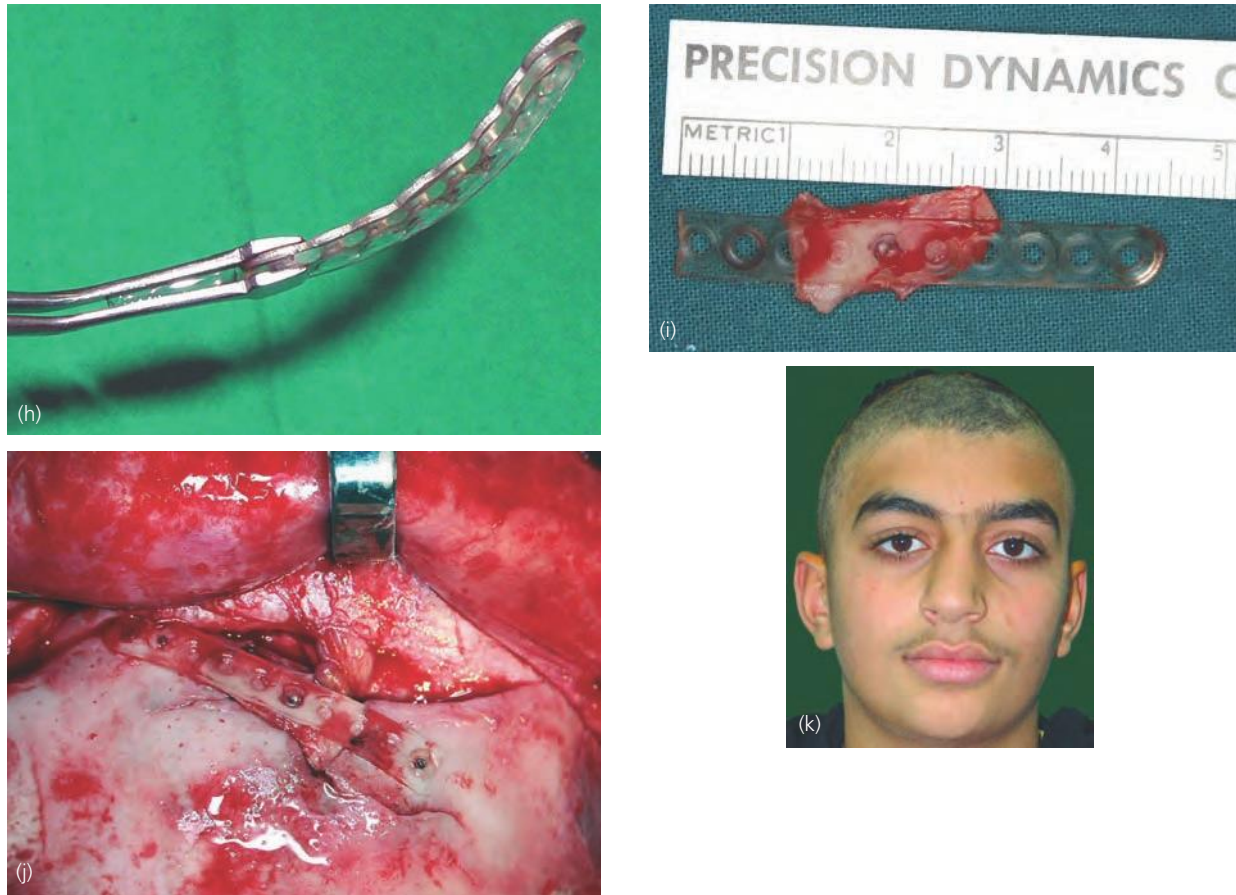


Fig. 40.60 (cont'd) (h) Template and miniplate after cooling. (i) Bone fragment fixed to miniplate on side-table. (j) Completed reconstruction. (k) Appearance of patient 1 month after surgery.

The following factors can preclude early management of midfacial fractures:

1. Concomitant life-threatening injuries, such as brain trauma, spine trauma, chest trauma, intra-abdominal bleeding, etc., which must be treated first, or which dictate postponement of surgical treatment.
2. Unavailability of necessary preliminary consultations (neurosurgeon, ophthalmologist).
3. Unavailability of full preoperative diagnostic imaging.
4. Unavailability of complete maxillofacial surgery team.
5. Unavailability of operation room or anesthesiologist (in smaller hospital usually occupied by urgent cases like appendectomies or Caesarean sections).
6. Unavailability of special equipment, e.g. osteosynthesis sets that were used in elective cases and are not yet resupplied and sterilized.
7. Underlying medical conditions requiring further investigations or compensation (diabetes, ischemic heart disease, blood disorders, etc.).

If the “golden period” of the initial hours after injury cannot be used for any reason, the developing post-traumatic edema makes some procedures difficult or even unfeasible (full eye examination, conjunctival or eyelid incisions). In such an instance it is better to

postpone definite treatment for several days until all conditions are favorable for successful and uncomplicated surgery.

Most authors agree that midfacial fractures can be treated without significant detriment to the result during the first 2 postoperative weeks. After that mobilization of fragments and their proper positioning become increasingly difficult due to callus formation and soft tissue scarring and contraction. After 3 weeks primary treatment is mostly impossible and surgical correction must be planned as a secondary reconstructive procedure. In such cases it is often preferable to postpone reconstruction for several months until there is maturation of calluses and scars and full recovery of patient's general condition. The usual reasons preventing primary treatment of midfacial fractures are brain injuries, cervical spine fractures, and nosocomial infections with multiresistant bacteria acquired during a long stay in the intensive care unit.

Anesthesia and airway management

Oral and nasal intubation

The usual way of securing the airway during surgical procedures is standard orotracheal intubation. This method can be employed in cases of limited midfacial trauma like ZMC fractures, NOE fractures, or frontal

bone fractures that do not require maxillomandibular fixation (MMF). However in cases of midfacial fractures involving occlusal disturbance, it is necessary to be able to bring the jaws into occlusion and repeatedly check the occlusion intraoperatively for adequate reduction. The same holds for cases with concomitant mandibular fractures.

In most maxillofacial trauma cases the airway is therefore secured by nasotracheal intubation (NTI) without interfering with MMF and the surgical approach.¹⁴⁶ Unfortunately, surgical reconstruction in patients with midfacial fractures sometimes precludes NTI, or requires switching the endotracheal tube from the nasal to the oral route at certain stages of the operation to make the nose accessible to surgery. This may compromise the airways.

NTI can be also problematic in patients with skull base fractures, potentially creating a communication between the nasal cavity and the anterior cranial fossa. Accidental passage of the tube into the cranial cavity is a very rare but catastrophic complication, dreaded by anesthesiologists.^{147,148}

Tracheostomy

A traditional method for airway control in such cases is tracheostomy. Despite being one of the most common surgical procedures, the tracheostomy has a

complications rate of 14–45% documented in the literature.^{149,150} Complications of tracheostomy include bleeding, injury to adjacent structures, surgical emphysema, pneumothorax or pneumomediastinum, blockage of tracheostomy cannula, displacement of cannula, tracheitis, cellulitis, pulmonary atelectasis, tracheo-innominate fistula, tracheo-esophageal fistula, tracheo-cutaneous fistula, tracheomalacia, granulation, tracheal stenosis, excessive scarring and failure to decanulate. Use of tracheostomy should, therefore, be considered judiciously.¹⁵¹ This is especially true in patients in generally good condition, which would require a tracheostomy only due to nasal intubation not being possible or being contraindicated.

Submental intubation

A method with specific special advantages for complex midface trauma cases is submental intubation. In 1986 Hernández Altermir¹⁵² described an alternative method of endotracheal intubation in maxillofacial trauma patients – submental endotracheal intubation (Fig. 40.61). The method gained wide acceptance and with some modifications is considered an attractive alternative to tracheostomy in the surgical management of selected cases of maxillofacial trauma.¹⁵³ The main consideration in choosing this method is the expected length of the postoperative period requiring

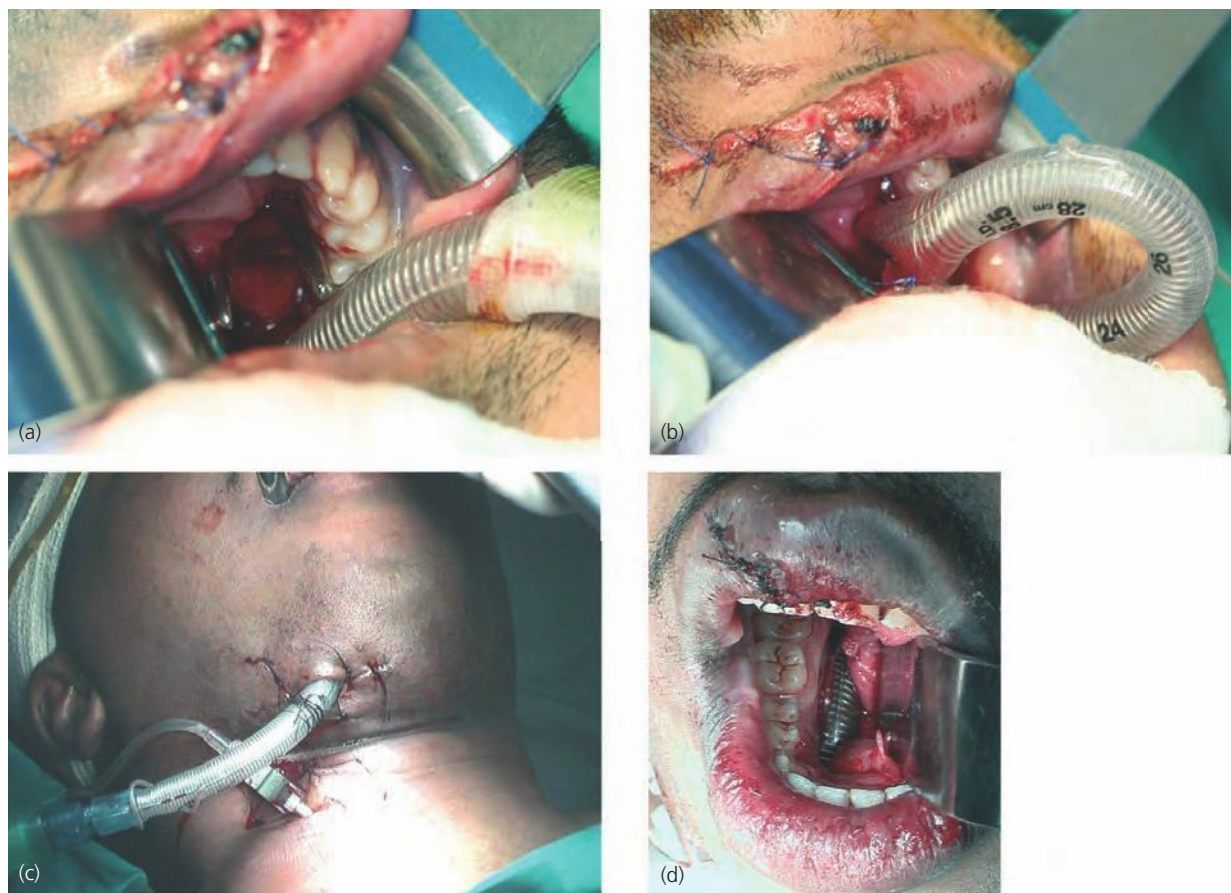


Fig. 40.61 Technique of submental intubation. (a) Large artery forceps is introduced into oral cavity through submental incision. (b) The tube is disconnected from the breathing circuit, the connector is removed, the end of tube is grasped by artery forceps and pulled outside through the dissected canal. (c) The tube is reconnected to the breathing circuit and secured in place by silk suture. (d) Position of the tube intraorally in the paralingual groove.

airway control. Usually the tube is switched back to the mouth at the end of the procedure and the patient is extubated. However some authors report on keeping patients on a submental tube for up to 48 postoperative hours. Submental intubation is contraindicated in patients requiring long-term airway maintenance.

Reported complications include detachment of the pilot balloon or its damage during externalization, damage to the cuff of the tracheal tube, infection of the submental wound, abscess formation in the floor of the mouth, salivary fistula, development of mucocele, and hypertrophic scarring. All these complications are relatively rare and avoidable with meticulous technique.

After the induction of general anesthesia the patient's trachea is intubated orally with a reinforced, spiral embedded, tracheal tube. It is important to release the sealed connector from the proximal end of the tube before intubation, so that it can be easily disconnected during the procedure. Patients already intubated by other types of tubes must have their tubes replaced.

A skin incision about 2 cm long is made parallel to the inferior border of the mandible in the submental area to one side of the midline. The right side is preferred over the left side whenever possible, because it allows better visualization of the position of the tube with direct laryngoscopy. Another incision is made intraorally at the junction of lingual attached gingiva and free mucosa of the floor of the mouth. Both incisions are connected by blunt dissection progressing from the outside to the inside through subcutaneous fat, platysma, deep cervical fascia and the mylohyoid muscle. Next a closed strong curved artery forceps is inserted into the mouth through the dissected canal, lateral to the genioglossus and medial to the digastric muscle.

At this point the endotracheal tube is briefly disconnected from the breathing circuit and the tube connector is removed. The deflated pilot tube cuff followed by the endotracheal tube are grasped by artery forceps and pulled outside in sequence. During this maneuver the tube is fixed in the mouth either manually or with McGill's forceps to prevent slipping from the trachea. The tube is then reconnected and secured to the skin of the submental area by strong silk suture, after verifying the unchanged tracheal insertion of the tube by auscultation of chest and checking the proper intraoral positioning of the tube in the paralingual groove (Fig. 40.61). At the end of the procedure the deflated pilot tube cuff and the tube are pulled back in the reverse order and the skin wound is sutured, while the intraoral wound is left to heal secondarily.

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Chapter 41

Orbital Reconstruction and Panfacial Fractures

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This chapter deals with the challenging surgical procedures of orbital and panfacial fractures. With the aim of restoring function and forms in all dimensions, preoperative planning and anatomic preformed implants must be of the highest standard. Intraoperative data acquisition using cone-beam technology and the combination with imaging fusion of preoperative planning data are replacing navigation procedures. The sequence of treatment is important.

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Orbital reconstruction

Introduction

In craniofacial trauma, nearly 40% of cases involve the orbital structures.^{1–3} Fractures of the orbital cavity occur mainly in the orbital floor medial to the infra-orbital groove and the nerve canal. Orbital floor fractures are regularly involved in combination with fractures of the medial wall.^{4–6} Due to the very complex geometry of the bony orbit, proper reconstruction after trauma is very challenging, especially if more than one wall of the orbit is affected. Therefore, the main aim of orbital reconstruction is to achieve

the correct preinjury anatomy of the skeleton. Nevertheless, post-traumatic facial deformities caused by incorrect reconstruction often result in enophthalmus, diplopia, and visual acuity disturbance due to enlargement of the orbital volume. In about 8.5% of treated patients with orbital fractures, a volume enlargement leads to enophthalmus, especially when the deep orbital cone is affected.^{6–8}

Secondary reconstruction of the orbit is necessary when the functional or esthetic result after primary repair is not satisfactory or when primary repair of the orbit was not done. The identification of stable anatomic landmarks in secondary reconstruction is often very challenging because of defects or scarring of the orbit. In these cases, more than one surgical procedure is often needed to get acceptable functional and esthetic results. Due to the complex anatomy, good results are achieved using computer-assisted surgery both in primary and secondary reconstructions. Therefore, a virtual three-dimensional (3D) reconstruction of the orbital cavity is mandatory using DICOM datasets. For the exact anatomic reconstruction of the orbital floor and the medial wall, an individually S-shaped cranio-caudal rise of the orbital floor and a retrobulbar bulge in the posterior aspect of the medial wall remain challenging.^{9,10}

Surgical anatomy

The orbital cavity is a pyramidal space. It is enclosed by bony walls and contains the ocular bulb, the peri-orbital fat, and accessory structures such as nerves and the ocular muscles. The ocular bulb itself occupies only one quarter of the bony orbital cavity. The medial orbital wall lies parallel to the antero-posterior axis, while the lateral orbital wall is inclined at 45 degrees to it. The lateral wall is part of the greater wing of the sphenoid and the zygomatic bone and therefore resists most severe impacts. In the posterior aspect of the orbital cavity there is a gap between the lateral wall and the roof, the superior orbital fissure, leading to the middle cranial fossa. The inferior orbital fissure, situated between the lateral wall and

the orbital floor, leads to the pterygopalatine and the infratemporal fossae. Although the bones of the medial wall are thin, the ethmoidal septa support the bone. In cases of trauma, they dissipate the forces and protect the ocular bulb. In the posterior part of the orbit lies the thicker bone of the sphenoid bone, enclosing the optic nerve.^{11,12}

The orbital roof is in a horizontal plane and separates the orbit from the anterior skull base. In the superomedial aspect of the orbital roof the frontal sinus obtrudes between the cranial and frontal bones. From a clinical point of view, orbital roof fractures often lead to proptosis and exophthalmos.¹³

The orbital floor is concave in the anterior third and becomes convex behind the globe, forming the postbulbar bulge. The bone is paper-thin, especially in the region of the infraorbital groove and at the junction between the floor and the medial wall. The canal containing the infraorbital nerve is normally separated from the floor and lies above the maxillary sinus. The most common site of orbital blow-out fractures is the anteromedial portion. Fractures at the posterolateral portion of the floor often lead to dislocation of periorbital fat into the pterygopalatine fossae.⁴⁻⁶

The orbital rim, derived from the frontal, maxillary, and zygomatic bones is the part of the orbit most resistant to trauma. In the lateral and inferior aspect it is thickest and weakest in the region of the frontozygomatic suture. The rim itself is a useful guide to anatomically important intraorbital structures. The infraorbital fissure lies 20 mm posterior to the orbital rim, the supraorbital fissure is 35 mm from the frontozygomatic fissure. The optic canal lies an average 45 mm from the inferior orbital rim. During surgical procedures, care of structures such as the medial canthal ligament and the lacrimal gland is mandatory. During preparation, the ethmoidal arteries must be identified and coagulated. As no important anatomical structure enters the orbit through the infraorbital fissure, dissection of the periosteum is essential for adequate exposure in managing orbital floor injuries.

Orbital soft tissues

The orbital septum is fixed at the orbital rim and acts as a barrier. In the presence of edema, blood, or infection it causes an unhindered rise in retrobulbar pressure.¹⁴ In this case a proptosis of the globe occurs and may lead to a decrease of microvascular circulation of the retina. If drainage of the retrobulbar space is necessary, the orbital septum is best incised in the lateral aspect, superior or inferior to the lateral canthal ligament.

The conjunctiva is a highly vascular structure. Subconjunctival hemorrhages therefore cease at the margin of the conjunctiva. In the medial aspect the tarsal plates of the conjunctiva are continuous with the medial canthal ligament. The ligament inserts outside the orbital rim on to the frontal process of the

maxilla and the posterior margin of the lacrimal fossa. The lateral ends of the tarsal plates merge with the lateral canthal ligament. It is attached to the frontal process of the zygoma. The integrity of these ligaments is very important from both a functional and an esthetic point of view. Preparation at the frontozygomatic suture and injury at the bony junction therefore lead to a downward sloping palpebral fissure. On the other hand, displacement of the bony attachment of the medial canthal ligament causes a lateral movement and leads to a traumatic telecanthus.

The periorbital fat acts like a cushion for eye rotation. Numerous fibrous septa run throughout the orbital fat. In entrapment of the septa, as is often seen in fractures involving the orbital floor, there is mechanical interference with free rotation of the ocular bulb. The integrity of the septae, the muscular tone of the extraocular muscles, and tissue hydration maintain the antero-posterior position of the bulb. The ocular bulb position changes if the bony orbital volume is altered, as in orbital fractures, resulting in an enophthalmos. Therefore, correct reconstruction of orbital fractures is mandatory to avoid esthetic and functional problems. The most common location of herniation of periorbital fat tissue is posterior into the maxillary sinus. In almost half of the cases, periorbital fat is also dislocated medially into the ethmoid cells.

The rectus muscle originates from a tendinous ring at the optic foramen. The long sagittal axis of the bony orbit modifies the action of the muscles. The medial and lateral rectus muscles move the ocular bulb only horizontally. The superior and inferior muscles, due to their oblique position forwards and laterally, bring about rotation, elevation, and depression of the bulb. Diplopia often follows orbital trauma, due to fractures of the orbital floor or the medial orbital wall. Failure of eye movement is not because of nerve palsy but results from edema, hematoma or even septal or fat entrapment. The maximum limitation of movement is often opposite to the field of action of the affected muscle.

The optic nerve is surrounded by dura, arachnoid, and pia mater. The dura fuses with the periosteum of the orbit. In the majority of orbital trauma cases, loss of vision results from edema or hematoma. Only in a few cases do fractures of the optic foramen result in vision loss.

Surgical approaches to the orbital cavity

Several approaches to the orbital cavity have been described in the literature. Besides the transconjunctival approach, access to the orbital cavity can be gained by subciliary, midtarsal, and infraorbital incisions. Because of the high risk of developing ectropion of the lower eyelid and a visible scar, the surgical approach to the orbital cavity should only be performed using a transconjunctival approach.

Transconjunctival approaches offer several advantages to the patient and the surgeon.¹⁵ It has been described especially for the treatment of orbital trauma.¹⁶ The approach produces no visible scar and therefore reduces the incidence of lower lid ectropion. An incision is made 5 mm posterior to the eyelid ridge in the inferior fornix. The postseptal area, which includes the palpebral conjunctiva and the extension of the orbital fat, is the preferred route for access to the orbital cavity. Following the incision of the periosteum, the basal, inferomedial, and inferolateral regions of the orbital cavity can be accessed.

Depending on the region of the orbit to be approached, an upper blepharoplasty approach is made. The orbicularis oculi muscle is incised and the superior and lateral orbital rim is exposed. The blepharoplasty and tranconjunctival incisions are connected by a subperiosteal dissection. After opening the periorbita, the ocular bulb is retracted to gain better visualization of the orbital cavity. After surgery, the conjunctiva is closed with a running resorbable 8/0 suture. The periorbita and the capsulopalpebral fascia do not need to be closed.

Investigation of orbital trauma

Useful techniques for the assessment of orbital trauma are listed in Table 41.1.

Plain radiography of the orbit can demonstrate bony margins clearly, although fractures of thin bony structures of the orbital walls are often overlooked. Therefore, 3D radiography techniques like computed tomography (CT) scans or magnetic resonance imaging (MRI) are more useful for assessment both of orbital fractures and soft tissue. Advances in imaging techniques, such as multiplanar and 3D reconstruction and 3D models, have opened up a new field of computer-assisted preoperative planning and computer-assisted surgery for orbital trauma and secondary reconstruction. Measurement of bulb position and orbital volume can be done preoperatively and compared with the non-affected side. During the operation surgical results can be compared with the normal side and symmetry can be achieved.^{17–23}

Table 41.1 Assessment techniques for investigation of orbital trauma.

History and clinical examination
Ophthalmological investigation and orthoptic assessment
Measurement of bulb position (Hertel index)
Diplopia assessment
Plain radiographs
Computed tomography (CT) scans
Magnetic resonance imaging (MRI)
Three-dimensional reconstruction and volumetric assessment
Computer-assisted preoperative planning
Surgical findings
Computer-assisted surgery

Diplopia is investigated using a linear light and questioning the patient about separation of two images. Additionally, a Hess chart can give reproducible results of ocular movement.

Complications of orbital trauma

A retrobulbar hematoma may occur rarely (<1%) in midface or orbital trauma, or due to the treatment of trauma. Normally, one or more of the posterior short arteries ruptures resulting in bleeding within the intraconal space. Compression of the remaining posterior short arteries leads to a change in the perfusion gradient of the ocular bulb. The rising venous congestion and edema of the optic nerve results in obstruction of the central retinal vessels. Irreversible damage of the retinal cells occurs after 15–20 minutes of vascular occlusion.

Diagnosis and treatment of a retrobulbar hematoma are shown in Table 41.2. Clinically, decreasing visual faculty and the start of blindness is obvious. Often, pain, diplopia, and ophthalmoplegia are evident. A forward displacement of the iris obstructs the drainage of the aqueous from the anterior chamber leading to proptosis of the ocular bulb. The bulb is tense to palpation with increased ocular pressure on tonometry. The pupil dilates with a lack of direct light reflex.

Medical treatment should start by reduction of the intraocular pressure by dehydrating the patient using a combination of intravenous mannitol (250 mg 20%) and acetazolamide (500 mg). Circulatory spasm and intraorbital edema are treated using high dose of steroids (dexamethasone 4 mg/kg as a bolus, then 2 mg/kg every 6 hours for 24 hours). If conservative treatment fails, preparation for surgery should start. The aim of a surgical procedure is decompression of the intraconal space. It is approached by a four-quadrant incision. The intramuscular septum is divided by a medial and lateral upper and lower eyelid incision and preparation of the intraconal space. Insertion of a drain might be useful.

Table 41.2 Diagnosis and treatment of retrobulbar hematoma.

Diagnosis
Pain
Decreasing visual faculty and onset of blindness
Proptosis
Onset of ophthalmoplegia
Tension of the ocular bulb
Ecchymosis
Dilated pupil
Loss of visual evoked potentials
Treatment
Surgical decompression
Mannitol IV (250 ml, 20%)
Acetazolamide IV (500 mg)
Dexamethasone 4 mg/kg as a bolus, then 2 mg/kg every 6 hours for 24 hours

In the case of severe impact from the lateral aspect of the orbit, the edges of the superior orbital fissure might be approximated causing superior orbital fissure syndrome. Due to compression of the neurovascular structures passing through the fissure, edema of the periorbital tissue occurs with proptosis of the ocular bulb. The third, fourth, and sixth nerves are affected, resulting in complete ophthalmoplegia and ptosis. The direct and consensual light reflexes are diminished or lost. A specific sign might be disturbed sensation at the forehead because of involvement of the frontal branch of the trigeminal nerve. Monitoring of the optic nerve is always mandatory. Where there is an intact optic nerve, a conservative approach is proposed. Partial or complete recovery is reported within 6–12 months. Operative decompression of the superior orbital fissure is controversial.

In an extensive injury of the orbit with extension to the skull base, a carotidocavernous fistula can occur resulting in a rare but serious complication. The internal carotid artery within the carotid sinus is damaged. Clinically, a pulsation exophthalmos is visible. On auscultation, a murmur is obvious over the frontal region. Performance of arteriography confirms the diagnosis. The treatment of choice is embolization or transcranial closure.

Preparation for orbital surgery

In the clinical assessment, symmetry of the eye and the zygoma should be examined in all three planes, from in front, superiorly, and inferiorly. Sensations of the skin around the orbit, especially from the infra-orbital, the supraorbital, and the zygomaticofrontal nerves should be recorded. Patients are asked to bring pretrauma photographs. Preoperative clinical photographs are requested.

Clinical investigations also include an ophthalmological examination to determine visual acuity, visual fields, accommodation, eye movement and double vision, and intraocular pressure.

Regarding radiography, occipitomenal projections might be useful to identify bony trauma and fractures of the orbital rim. Zygomatic displacement is also assessed in comparison to the unaffected side. Nevertheless, 3D techniques like CT or MRI scans are more useful in the diagnosis of orbital trauma. The CT imaging modality has been shown to be the diagnostic technique of choice for demonstrating the complexity of these skull fractures.^{17–23} Multiplanar reconstruction with 1 mm slices provides improved preoperative clinical information and thereby enables accurate preoperative surgical planning.

Computer-assisted surgery in orbital surgery

Computer-assisted surgery (CAS) has proved to be an essential tool for preoperative planning and intraoperative control of the precise reconstruction in craniomaxillofacial trauma.^{24–36} Stereotactic guidance

for intracranial surgery dates back to the early 1900s. Frameless surgical navigation (or CAS) systems were subsequently developed.^{37,38} These CAS systems are in common usage for neurosurgery, endoscopic sinus surgery, and craniomaxillofacial surgery.²⁴

A typical maxillofacial CT data set is in DICOM format when recorded from the CT scanner. It is composed of approximately 150 1 mm, non-overlapping slices, obtained with no gantry tilt. Virtual reconstruction of these images in every slice thickness is possible, but due to the resolution of the available scanners this is not practical.³⁹ Voxels represent the volumetric components of the 3D dataset. Pixels are represented in the slice distance. Ideally the square blocks would have isometric length but due to the edge length (0.3–0.5 mm) of the CT scanner detectors, this is not possible. The third dimension is determined by the table feed which is at least 1 mm and is the limiting factor.^{40,41}

All CAS systems incorporate a computer digitizer to track the location of the patient and instruments in space. Four different digitizer modalities have been designed: optical, electromagnetic, electromechanical, and ultrasound. Only the electromagnetic and optical systems are in common use. Each system has unique registration procedures, advantages, and disadvantages.^{42–45}

After importing the CT dataset, performing the preoperative planning, and generation of a “virtual patient”, a registration process is necessary to integrate the virtual and actual datasets. This defines both the anatomy and the location of the patient in the operating room. The registration process involves identification of specific anatomic landmarks or fiducial markers applied to the patient before obtaining the CT scan. The landmarks or fiducials are then identified on the virtual patient. The fiducials used for registration can be separated into two types: invasive and non-invasive. Invasive fiducial markers (e.g. titanium screws) are rigidly fixed to the skull before the CT scan and remain in place until the procedure is complete. Non-invasive fiducial markers can be subdivided into three types: adhesive markers, dental appliances, and anatomic landmarks.^{36,40,46–49} Both the adhesive markers and the dental appliances must be applied to the patient prior to acquiring the CT dataset, while anatomic landmarks are inherently present.

Preoperative planning

After importing the CT dataset of a trauma patient, several different preoperative planning tools are available depending on the defect (Table 41.3).

Unilateral defects

For unilateral defects of the orbital cavity, mirroring the unaffected side to the affected side is a well known standard procedure. After segmentation of the corresponding area of interest, a mirroring procedure is

Table 41.3 Available virtual planning tools for different trauma.

Planning tool	Trauma
Mirroring	Unilateral defects
Virtual implants/reference skull	Bilateral defects
Individual designing (CAD/CAM)	Bony defects

easy to perform in most 3D modeling software. After this, however, an additional adaptation to the remaining bony structures has to be done, due to the physiological asymmetry of the human skull.^{24–26,50}

Bilateral defects

The mirroring protocol is not possible in defects involving both sides of the skull. Therefore, two possibilities were created in the last few years. The first solution is having a database with segmented atraumatic skulls from other patients which can be imported as a reference skull. After fusion and adjusting the selected skull into the patient dataset, virtual repositioning of the dislocated bone fragments is possible considering the ideal shape of the skull. In addition,

the virtual planning can be exported (STL-format) and printed out via a stereolithographic device or a 3D printer (Fig. 41.1).

The other solution is application of virtual bony spare parts for bilateral defects, where no bony fragments can be repositioned (e.g. orbital floor, medial wall). Standardized implants can be imported into the patient dataset and placed in the area where reconstruction is needed. With regard to the orbital cavity, virtual implants are available which represent the definitive physical implant; this is then inserted during surgery (see later section, Preformed orbital implants) (Fig. 41.2).

Bony defects

CAD/CAM procedures are used when large defects of the calvarium need to be covered by alloplastic material, such as titanium, ceramic implants or peak. Several companies offer the service for producing these individual implants. After sending the patient-specific dataset to the company via the internet, planning proposals are sent back to the surgeon. After approval, the individual implant is produced and sent to the hospital for implantation (Fig. 41.3).



Fig. 41.1 (a) Patient dataset after primary insufficient reconstruction including a dataset of an equivalent unaffected midface dataset (green) after image fusion. (b) Bony fragments after reposition to the ideal position given by the unaffected and fused dataset. (c) 3D model including all segments (color coded) where a reosteotomy has to be done.

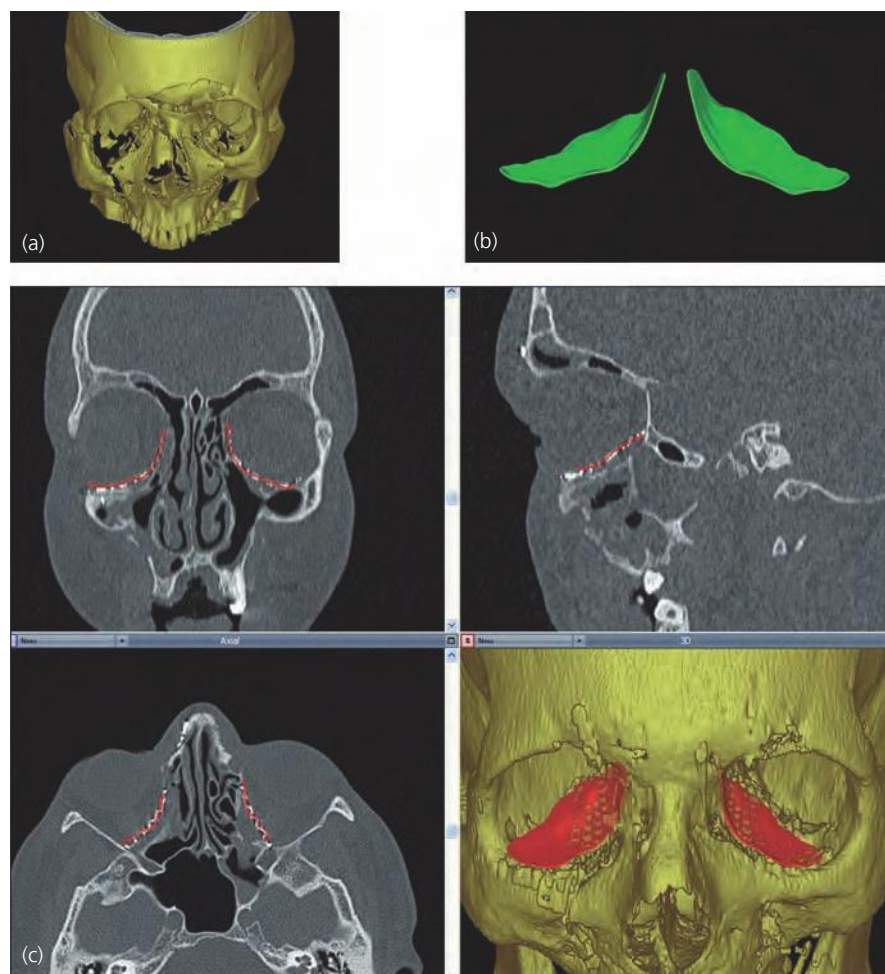


Fig. 41.2 (a) Patient CT dataset with severe midface trauma. The mirroring procedure is not possible. (b) Standardized virtual orbital floor implants available in a database. (c) Postoperative control CT dataset after image fusion with the preoperative planning. The two inserted preformed titanium meshes are at the same level as the preoperative planned one.

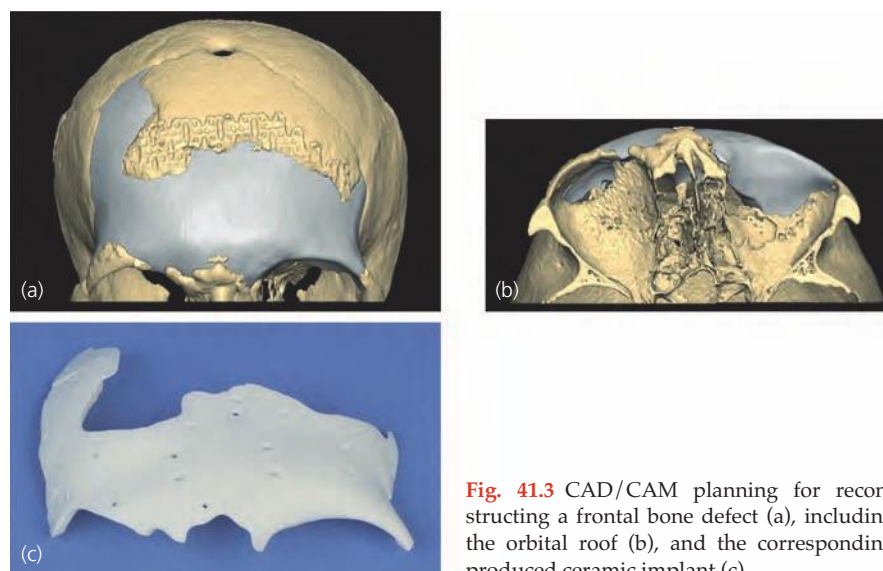


Fig. 41.3 CAD/CAM planning for reconstructing a frontal bone defect (a), including the orbital roof (b), and the corresponding produced ceramic implant (c).

Material for orbital reconstruction

There is still no consensus on the ideal material for reconstruction of the orbital cavity.⁵¹ In general the material should be easily cut and sized in the operating room, and able to be shaped to fit the orbital contours and retain its new form without memory. It

should allow fixation to host bone by screws, wire, suture, or adhesive.⁵²

Depending on the defect size, lyophilized dura, silastic, polyethylene or polydioxanone sheets, hydroxyapatite blocks, titanium meshes, ceramic inlays, or autogenous bone grafts are recommended.^{2,52-60} Elastic materials are often unable to with-

stand static or dynamic stresses in large defects, are prone to foreign body reaction and implant exposure, and only fibrinous connective tissue remains after resorption, with insufficient bone regeneration.^{2,61–63} The disadvantages of autologous bone grafts are rigidity and the requirement of a donor region. They cannot be flexibly adapted to the individual anatomic situation and morbidity of the donor region occurs. In addition, further resorption and displacement of the implanted grafts often occurs if they are not precisely positioned and fixed.^{64–66} Titanium meshes have been used to connect large defective areas in the internal orbit in order to minimize postoperative displacement and facilitate correction.^{52,60,66} They are biocompatible, rigid enough to cover large defects, and deformable, allowing adaptation to individual anatomy (Fig. 41.4). However, adjusting to the individual anatomic requirements can be time consuming and difficult, especially if the deep orbital cone is affected or scarring renders the identification of stable posterior landmarks difficult.^{67–69}

Preformed orbital implants

Intraoperative 3D bending of titanium mesh for the internal reconstruction of the orbital cavity remains challenging, and navigation-guided control of titanium mesh reconstruction is time consuming.^{24,35} Free-hand placement of titanium mesh or calvarian split grafts is difficult for restoring the vertical height of the reconstruction in the posterior aspect of the orbital floor and medial orbital wall. The anatomical reconstruction of the so-called retrobulbar bulge area is most important to prevent eophthalmos deformity. Stereolithographic models or individual alloplastic implants can be used in these situations.

Spiral CT datasets with a 1 mm collimation/slice thickness are acquired. At a workstation (e.g. Voxim, IVS Solutions AG, Chemnitz, Germany) virtual reconstructions can be performed by mirroring the individually defined 3D segment from the unaffected side on to the defective side, which results in a new sub-volume. The resulting sub-volume file can be exported as an STL object to allow it to be read by CAD/CAM machines. The prepared data file then can be sent to a stereolithographic machine, which selectively polymerizes laser-curable liquid resin layer by layer, resulting in the template.^{31,32} This is an accurate process (error about 0.2%) and the fabrication time for these templates is less than 5 hours. Any cutting to size and adaptation of the titanium mesh that is required can be done on the template by hand. Afterwards it is removed and sterilized before the operation (Fig. 41.5).

However, CT scanning procedures are commonly performed in an axial manner which omits segments located parallel to and in between the scanned layers. Therefore the missing information of the orbital floor leads to formation of pseudo foramina in virtual imaging and in stereolithographic models.

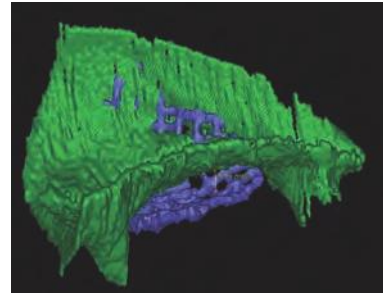


Fig. 41.4 Segmentated postoperative dataset of an orbital cavity (green) treated with a common titanium plate (blue). Not considering the shape of the orbital floor has led to an increase in the orbital volume, because the implant was placed into the maxillary sinus.

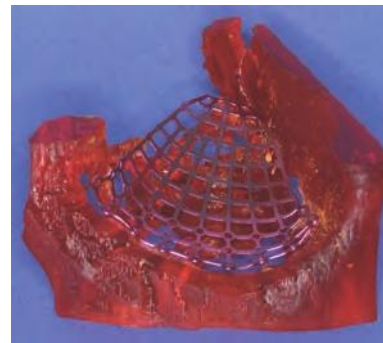


Fig. 41.5 Stereolithographic model of a right orbital cavity. With such a model, individual adaptation of titanium mesh is possible.

Furthermore this technology involves higher costs and greater logistic challenges.^{2,30,70}

Efforts have been made to produce standardized anatomic preformed 3D implants for orbital reconstruction. These would enable optimal reconstructions in more centers without having additional devices like intraoperative navigation or intraoperative imaging. First basics were created by analyzing and classifying the human orbital floor.⁷¹ By performing CT data analysis the surface of the orbital cavity could be evaluated and this showed that the variation in the depth of the orbital floor was low amongst patients. By clustering the data, the 95% confidence interval was within 1 mm. Age has no influence on the shape of the orbital wall, once the bony skeleton has matured.⁷² Additionally, the volume and morphology of the orbital cavity were constant and steady after the age of 17 years.^{73–75} The extent and width of the orbital rim is reported to show a strong correlation with the volume of the cavity. Depending on ethnic groups, mean values for the volume can be found in the literature ranging from 20–26 cm³.^{74,76,77} By using these results, standardized orbital cavities could be rebuilt and a new type of three-dimensional orbital implant could be designed (Fig. 41.6).

Only two different sizes are necessary to cover the area of interest in a standardized way at the orbital cavity; these two sizes are available in male and female types. The average implant error was less than 1 mm for all orbits studied.⁷⁸ Anatomic structures and landmarks can be coded in these implants. With



Fig. 41.6 Standardized preformed orbital implant (for patient's left side).

regard to the anatomic reconstruction demands, the following structures determine the implant borders: anterior – lower orbital rim; posterior – deep orbital cone; lateral – beginning of lateral orbital wall; medial – frontoethmoidal suture and nasolacrimal duct leading to retraction of the antero-medial wall. The implant has to be placed on remaining stable bony contours at the anterior and dorsal aspect of the orbital cavity (Fig. 41.7). The insertion of the implant itself is possible using a transconjunctival approach without canthoplastic extension.

For preoperative planning the virtual implant can be used to perform a reconstruction of the orbital floor and the medial orbital wall, which can simultaneously be used for intraoperative navigation without additional segmentation procedures. Additionally, this information determines the correct size of the implant.

A true-to-original reconstruction of the orbital walls, especially of the transition area between the

orbital floor and medial orbital wall are key procedures in orbital trauma management, and make a major contribution to the restoration of function and esthetics of the midface. Often the medial wall is simultaneously affected, which is one of the main reasons for postoperative problems of volume excess, dysfunction, and enophthalmus.^{1–6}

Compared to other methods, standardized orbital 3D implants have logistic and financial advantages, as no extra models have to be produced.^{28,30,34,79,80}

Therefore, virtual planning, implant fabrication, and surgical procedure are possible on the same day. This allows treatment on the same day, even in an emergency situation. However, trauma to the orbital roof or the skull base still cannot be considered.⁸¹ This can be a problem in malformations or defects where no remaining anatomical landmarks for correct placement exist. These virtual implants have their limitations, therefore, for preoperative planning.

Intraoperative imaging and postoperative control

CT with primary coronal sections (earlier) and multislice systems with secondary coronal reconstructions (nowadays) are accepted standards for preoperative diagnostic, planning, and postoperative control of midface fractures.²³ It has been repeatedly advocated that postoperative conventional radiography, cone-beam computed tomography (CBCT) or CT should be performed for control and self-correction for the surgeon.⁸² However, postoperative

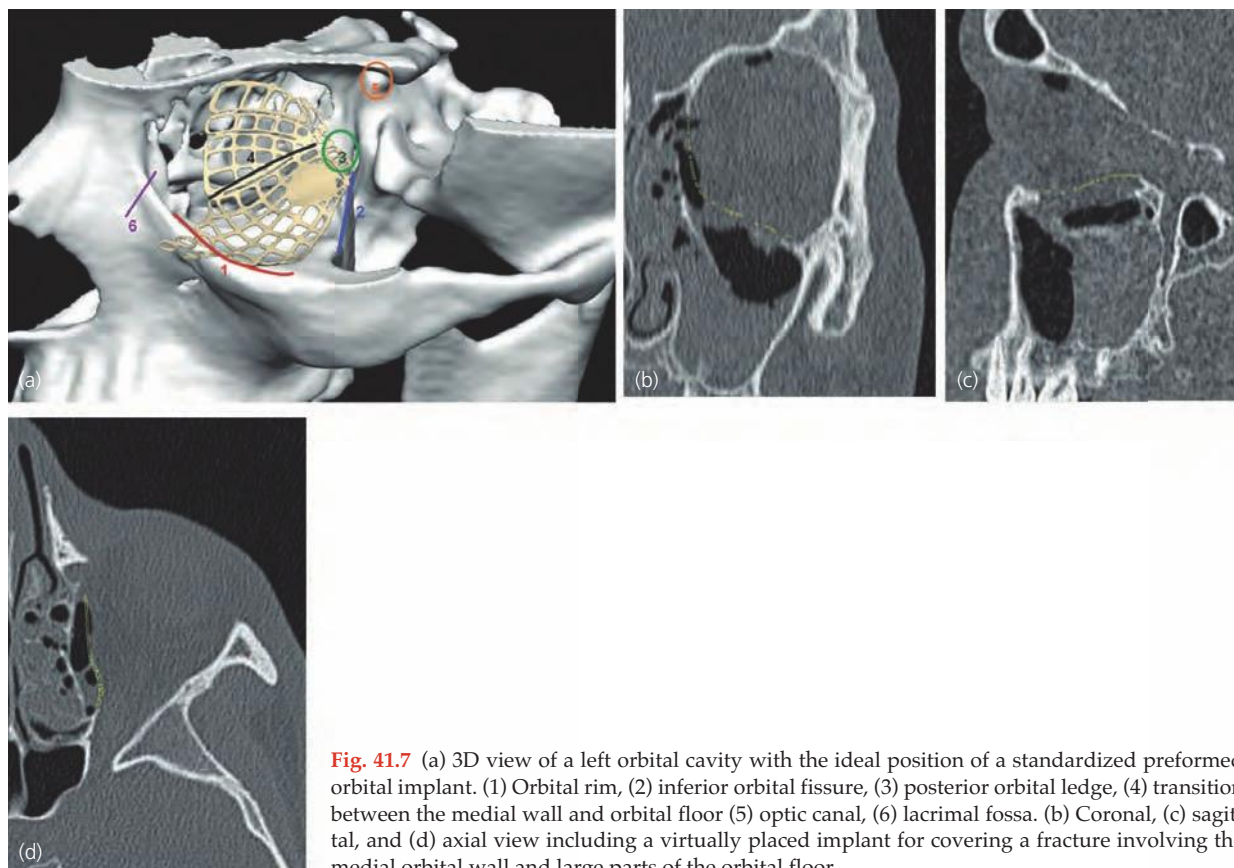


Fig. 41.7 (a) 3D view of a left orbital cavity with the ideal position of a standardized preformed orbital implant. (1) Orbital rim, (2) inferior orbital fissure, (3) posterior orbital ledge, (4) transition between the medial wall and orbital floor (5) optic canal, (6) lacrimal fossa. (b) Coronal, (c) sagittal, and (d) axial view including a virtually placed implant for covering a fracture involving the medial orbital wall and large parts of the orbital floor.

controls outside the operation room are too late if instantaneous corrections are necessary and often a secondary reconstruction is not performed.⁸³

Intraoperative 2D fluoroscopy with a mobile C-arm system is well established in many surgical fields such as orthopedics and neurosurgery, but it is hardly used for craniomaxillofacial surgery, because projection fluoroscopy does not reveal the complex 3D structures of the filigree midface bones and cavities.^{84–86} The performance of intraoperative CT imaging for control of the surgical result of the head and neck region has already been described for orbito-zygomatic fractures.^{87,88} The advantage is in the possibility of getting intraoperative feedback for necessary surgical correction. However this technique involves enormous technical and financial expense and it must be kept in mind that during visualization of the midface region, sensitive structures like the lens are exposed to radiation.⁸⁹

The 3D rotational X-ray technique is well known from cardiovascular interventions.⁹⁰ New mobile C-arm systems such as Siremobil Iso-C^{3D} (Siemens AG, Medical Solutions, Erlangen, Germany) or Pulsera 3D-RX (Philips Medical Systems, Best, Netherland) permit intraoperative 3D imaging of bone structures.^{84,91} The C-arm images are comparable to CBCT images, and have shown no statistical difference in assessing the facial skeleton compared with CT.^{92,93}

The C-arm provides multiple images that are acquired about an isocentric point. The data provide tomographic images that may be reconstructed into 3D images like other 3D modalities. By panning the C-arm over the surgical field, no repositioning of the patient is necessary, and a fast intraoperative update

or postoperative control can be evaluated. By importing the C-arm dataset into the virtual planning dataset (diagnostic CT), automatic image fusion of both datasets leads to fast feedback about preoperative planning and the actual surgical situation (Fig. 41.8).

Conclusion

Orbital reconstruction still is a challenging surgical procedure. Preoperative planning and anatomic preformed implants are currently the technologies which define the highest standards in orbital reconstruction. Intraoperative data acquisition using cone-beam technology and the combination with the imaging fusion of the preoperative planning data are displacing navigation procedures and postoperative control CT scans.

Planning and sequencing of the treatment of panfacial fractures

Panfacial fractures are injuries of the facial skeleton involving the mandible, the midface, the naso-orbital-ethmoid area, and the frontal bone. The aim of the treatment in severe facial injuries is 3D reconstruction of the preinjury facial projections and restoration of form and function.

Diagnostics

CT scan with axial, coronal, sagittal, and 3D views is performed for diagnostics, often in the unconscious patient. The information of the CT scan concerning

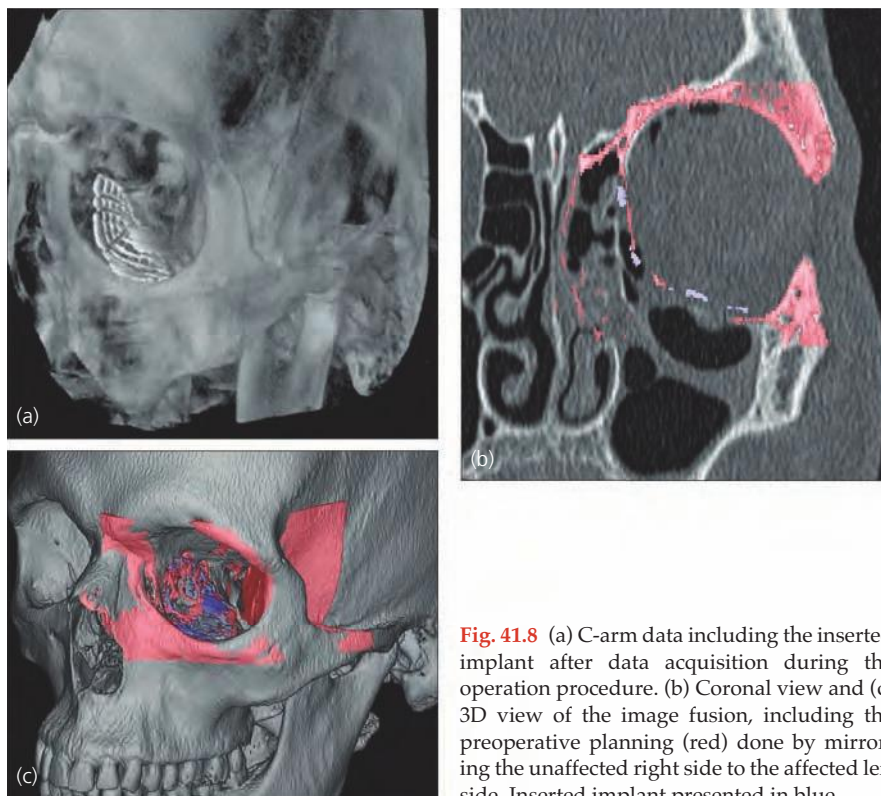


Fig. 41.8 (a) C-arm data including the inserted implant after data acquisition during the operation procedure. (b) Coronal view and (c) 3D view of the image fusion, including the preoperative planning (red) done by mirroring the unaffected right side to the affected left side. Inserted implant presented in blue.

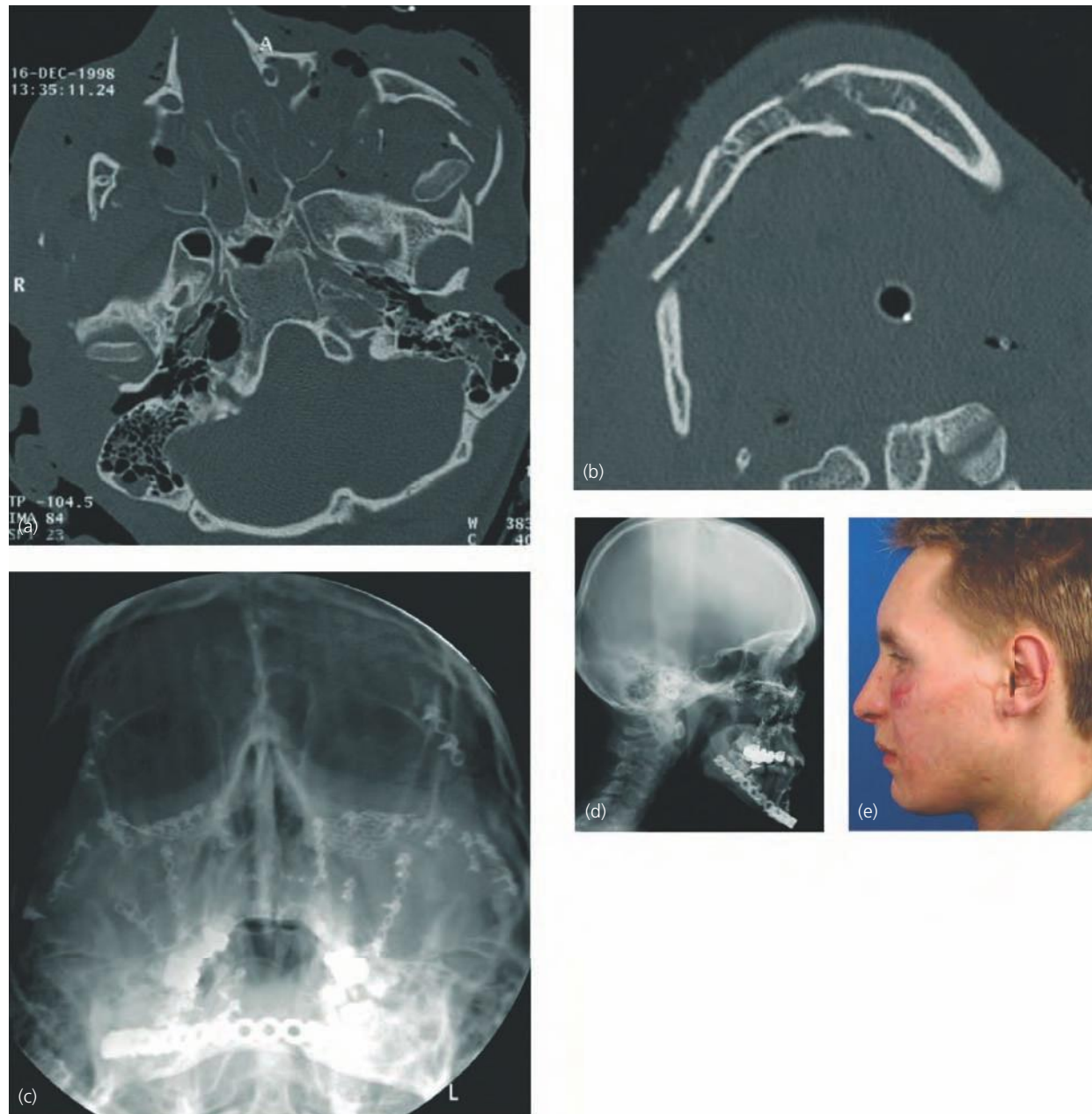


Fig. 41.9 (a) The axial view of a CT scan demonstrates a panfacial fracture with a comminuted bilateral centralateral midface. Loss of facial projection is noted by evaluation of the displaced fractures of the zygomatic arches. (b) Comminuted mandible fractures are seen. (c, d) Postoperative radiographs using coronal and lateral views demonstrate the result after open reduction and osteosynthesis. Note the miniplate osteosyntheses in the midface are located along the facial buttresses including the zygomatic arches. The mandible was reconstructed using a load-bearing reconstruction plate. Both orbital floors were repaired primarily using titanium mesh. (e) Following open reduction and osteosynthesis, 3D facial reconstruction was achieved.

the degree of dislocation and comminution of the fractures is important for sequencing of the treatment. Sequencing of the treatment is based on the clinical evaluation and the information of the CT scan concerning important areas such as the naso-orbital-ethmoidal (NOE) complex, the inner orbit, the zygomatic arches, the central midface with possible sagittal fractures, and the mandible with possible shortening of the ascending ramus due to fractures of the condylar process and lingual gapping of mandibular fractures, which can cause a widening of the lower face.

The diagnosis of intracranial injuries is important for primary management and for the planning and

timing of the treatment. Involvement of the frontal bone, the frontal sinus, and the skull base with intracranial injuries is common, and often an interdisciplinary treatment together with the neurosurgeon is indicated.

Radiographs are not indicated for the diagnosis of severe craniofacial trauma as no diagnostic information is added following a CT scan.

Airway

In severe panfacial injuries restoration of occlusion is important for reconstruction of the facial skeleton. When the occlusal unit needs to be reconstructed

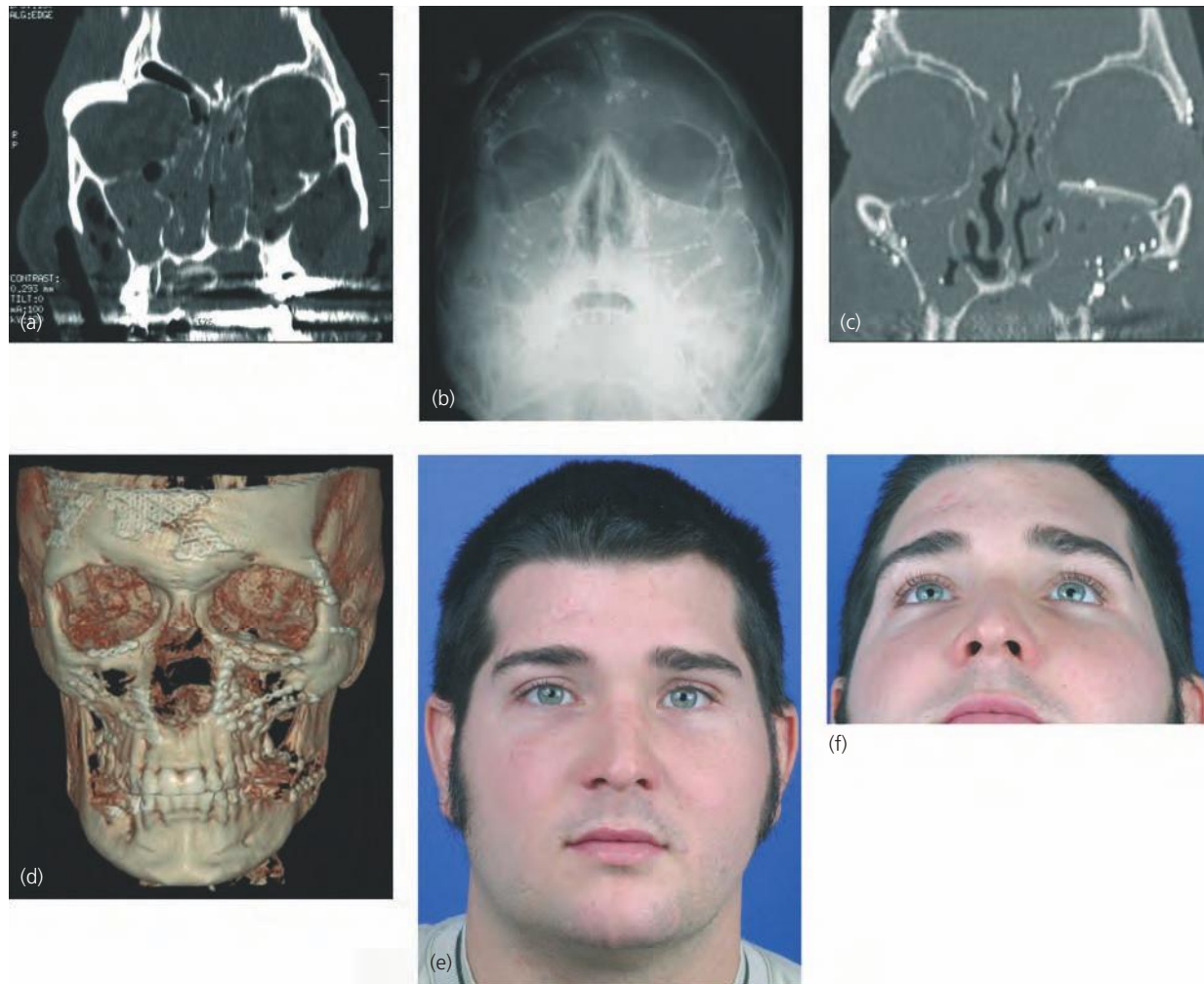


Fig. 41.10 (a) The coronal view of a CT scan demonstrates a panfacial injury with severe midfacial fractures and bilateral deformation of the orbit. (b) The postoperative radiograph demonstrates the location of the miniplates. (c) Primary bone grafting using calvarian split grafts harvested by a coronal approach were used for the primary reconstruction of the left orbital floor. (d) A 3D reconstruction of a cone-beam CT scan demonstrates a stable result with 3D reconstruction of facial dimensions 8 years postoperatively. (e, f) Clinically, temporal hollowing can be seen, caused by an injury of the temporal muscle from an incorrect coronal approach at the time of immediate treatment of the cranial injuries.

endotracheal oral intubation should be avoided intraoperatively in dentate patients. Nasal intubation is often performed, however when nasal intubation is not indicated due to severe central midfacial and nasal injuries, the endotracheal tube can be placed via a submental incision. Tracheostomy is only recommended when long-term postoperative intubation is indicated due to concomittant injuries.

Approaches

Coronal, transconjunctival, and transoral approaches are performed. These surgical approaches are described in Chapter 40. In comminuted mandibular and condyle fractures, approaches such as submandibular, preauricular, retromandibular, or transoral endoscope-assisted approach, may be used depending on the preference of the surgeon (see Chapter 43). Bone injuries are treated prior to soft tissue injuries. Extended lacerations can be used for open reduction and fixation of facial fractures. All fracture sites need to be exposed before reduction and fixation are per-

formed. The coronal approach is performed when open reduction and fixation of fractures of the NOE complex, the frontal bone including the frontal sinus, and the zygomatic arches are indicated. When a coronal approach are performed, this facilitates harvesting of calvarian bone grafts.

Timing

Treatment should be performed during the first 7 days after trauma. Surgical treatment via a coronal approach is recommended for reconstruction of the outer frame of the face and restoration of facial projection by control of the frontal bar, the NOE complex, and zygomatic arches. A combined neurosurgical and craniomaxillofacial treatment is recommended when injuries of the frontal skull base and fractures of the posterior wall of the frontal sinus are present. Reduction of NOE, midfacial, and mandibular fractures may be performed first when surgery of the anterior skull base or for fractures of the posterior wall of the fontal sinus is indicated at a later stage,

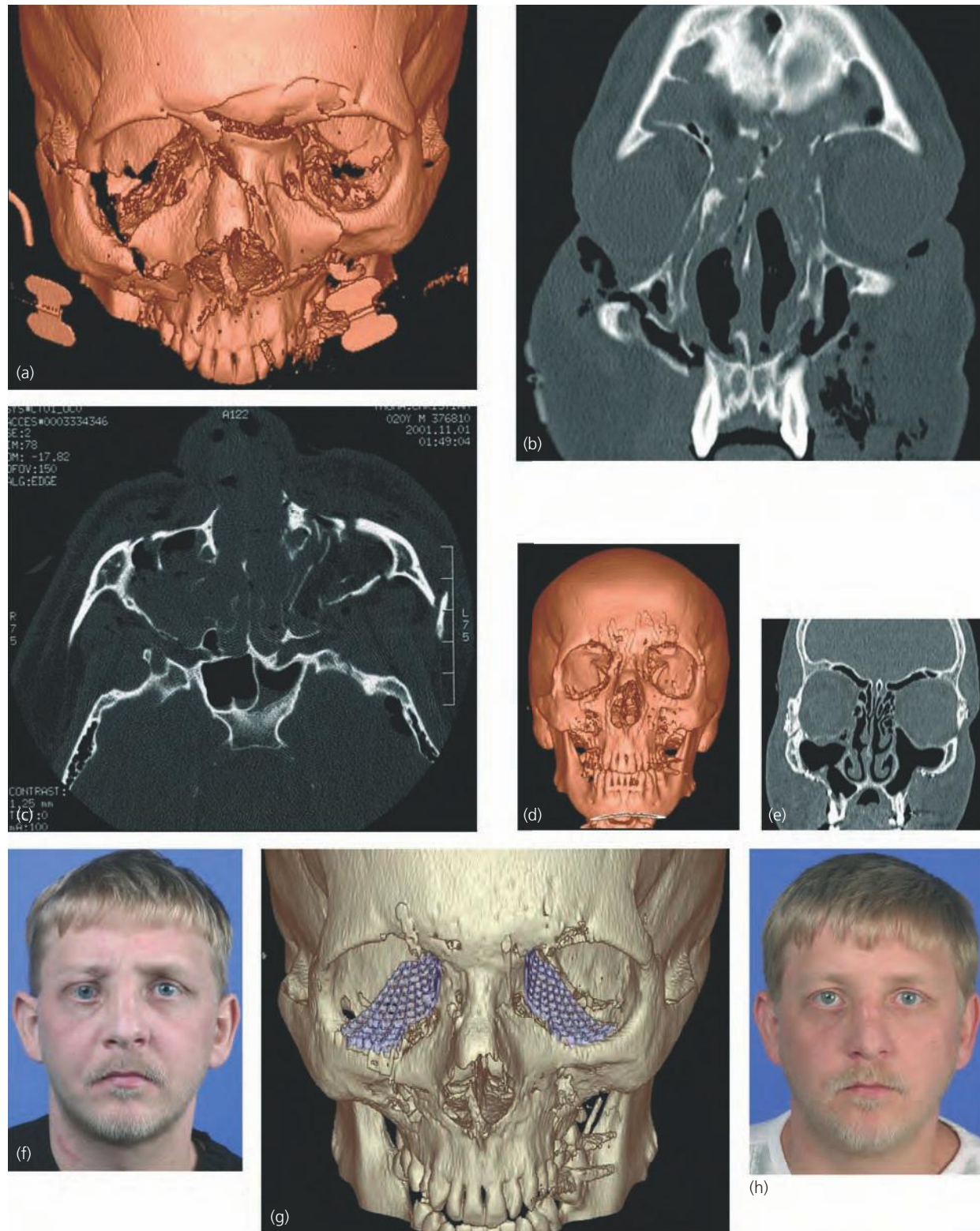


Fig. 41.11 (a, b, c) Panfacial trauma with impression of the NOE complex and deformation of both orbits and involvement of the frontal sinus is demonstrated by 3D reconstruction, axial and coronal view of the preoperative CT scan. (d) The 3D reconstruction and coronal view 4 months postoperatively show restoration of the facial projections with narrowing of the intercanthal distance. (e) Following functional reconstruction of the frontal sinus and the nasofrontal duct, pneumatization of the paranasal sinus system is demonstrated. Normal sinus function is of importance to avoid long-term complications such as mucocele formation and sinusitis in the frontal region. (f) As the visual function was unclear in the unconscious patient, reconstruction of the inner orbit was not performed primarily. After recovery at 4 weeks postoperatively an inferior position of the left canthal ligament and bilateral enophthalmos with severe diplopia was noted. Following submandibular intubation an inconspicuous scar is located at the right submandibular region used for the primary repair of the panfacial injuries. (g) Secondary bilateral orbital reconstruction was performed using computer-assisted planning and intraoperative control of the placement of two individually preformed titanium mesh implants. The bilateral orbital reconstruction and the fixation of the right canthal ligament using a transnasal wire are demonstrated in a 3D reconstruction of a CT scan after corrective surgery. (h) Following bilateral orbital reconstruction and medial canthal ligament fixation, a satisfying esthetic and functional result was achieved, without enophthalmos or diplopia.

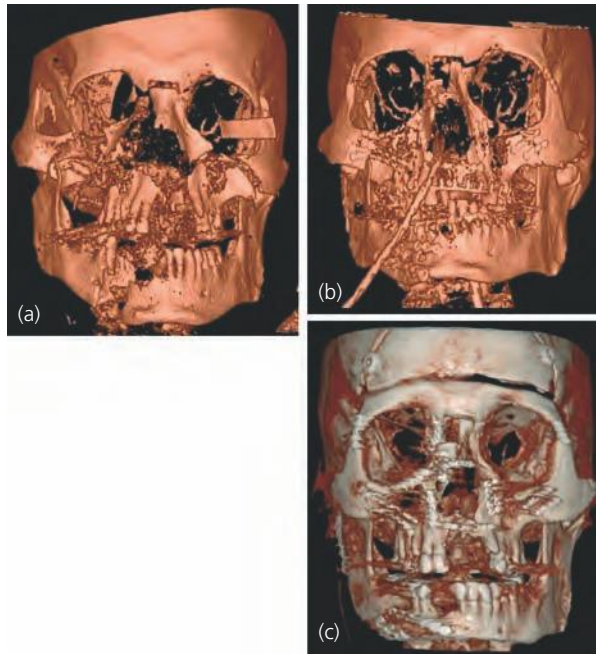


Fig. 41.12 (a) Panfacial trauma with severely dislocated midfacial fractures is demonstrated by 3D reconstruction of a CT scan. Due to intracranial injuries a neurosurgical treatment was not indicated at an early stage. To prevent difficulties of fracture reduction later than 2 weeks post-trauma, an open reduction of the periorbital, midfacial, and mandible fractures was performed without a coronal approach. (b) Extensive soft tissue swelling of the right zygomatic complex area did not allow anatomic reduction in this region. (c) During a second operation when a craniotomy for the treatment of the frontal skull base was performed, anatomic reduction of the right zygomatic complex was performed. Following the initial closed reduction of the NOE fractures, an anatomic reduction of the intercanthal distance was not achieved.

due to concomitant intracranial injuries, as precise fracture reduction after 14 days may be difficult. However, reduction of NOE fractures without a coronal approach may cause unsatisfying results. Sagittal fractures of the midface and the palate should be treated as early as possible, as reduction after several days may be difficult. In cases when early surgical treatment is not possible, transpalatal wires can be placed, even in the oral intubated patient, for closed reduction of sagittal palatal fractures to avoid widening of the midface.

Late reconstruction after 14 days leads to unsatisfactory results due to healing of fractures in malposition. Scarring with thickening of soft tissues and shrinkage of the overlying soft tissue envelope may cause unreparable deformities.

Therapy

Treatment strategies and sequencing of the treatment such as “top to bottom” or “bottom to top” have been described. A practical approach is to reconstruct the outer frame, with realignment of the zygomatic complex including the zygomatic arches and frontal bar, first. Reconstruction of the straight zygomatic arch is

important for restoration of the anterior–posterior facial projection.

In NOE fractures, reduction of the central fragment and exact repositioning of the medial aspect of the orbit with attachment of the medial canthal ligament is important, to avoid telecanthus deformity and broadening of the nose. Transnasal wiring can be used. A telecanthus deformity and broadening of the nose are difficult to correct secondarily and cause an unsatisfactory facial appearance. Reconstruction of the central midface and of the outer orbital frame is then performed. Widening of the face and enlargement of the orbit with enophthalmus deformity will result when the medial aspect of the orbit is not reduced adequately, as the infrarobital rim and the zygomatic complex will then be realigned based on the medial orbital fractures in malposition. Reconstruction of the paranasal and zygomatic buttresses is important for restoring the vertical height of the midface. Usually one or more buttresses can be reconstructed. A meticulous fracture reduction, even of small fragments, is recommended, especially in the buttress areas. Bone grafting may be indicated to achieve stable long-term results in areas with defects. Calvarian bone is the bone graft of choice. Mini- high and micro-osteosynthesis systems, such as 1.3 mini-plate systems, are recommended; they provide sufficient stability as mainly compressional forces are present in the midfacial area. The 1.3 plates are user friendly and allow for precise reduction and fixation without much bending of the plates, as the micro-plates adapt easily to the bone surface when the screws are tightened. In fractures with defects, stronger osteosynthesis systems such as 1.5 or 2.0 may be indicated.

An important factor for sequencing of the treatment is the occlusion present and the involvement of the mandible. In the dentate patient, restoration of the occlusion is important. In the edentulous patient, dentures may be helpful for reconstruction of facial height. Fractures of the anterior mandible frequently show lingual gapping which may cause widening of the face. Dislocated fractures of the mandibular condyle region and the ascending ramus often cause shortening of the ascending ramus which results in a unilateral or anterior open bite and incorrect vertical height of the face. When these fractures are present it is recommended to repair the mandible first before the occlusal unit is reconstructed. Correct mandibular reconstruction is not only important for restoration of the occlusal unit, but also to avoid broadening of the face. A broad face with an incorrect vertical height will result when the midface is realigned to a widened mandible with a shortened ascending ramus. For the reconstruction of comminuted mandible fractures a load-bearing osteosynthesis using rigid osteosynthesis plates such as 2.0 locking or reconstruction systems is indicated. An anterior rotation of the mandible with distraction of the condyles out of the temporal fossa should be avoided. When maxillomandibular

fixation (MMF) is applied, forward rotation of the mandible will cause a shortening of the midface and an anterior open bite when MMF is released. Anterior guiding elastics may be helpful to close an anterior open bite postoperatively. Arch bars should not, therefore, be removed at an early stage. For postoperative functional treatment using guiding elastics arch bars are better than MMF screws.

Reconstruction of the inner orbit is addressed last, following reconstruction of the outer frame, realignment of the NOE complex and the midface, and reconstruction of the mandible and of the occlusal unit. Repair of the walls of the inner orbit is of utmost importance for restoring ocular function and to avoid enophthalmos and diplopia. Furthermore the medial canthal ligament needs to be repaired when detached from the central fragment of the NOE complex to avoid telecanthus deformity. As the treatment of panfacial fractures is time consuming, orbital reconstruction by a second surgical team is recommended.

When the soft tissue envelope has been detached from the facial skeleton, resuspension of the midfacial soft tissue is mandatory to avoid soft tissue sagging, especially in the periorbital and midfacial region. Periosteal suspension sutures are used in the temporal and supraorbital areas.

Intraoperative imaging using cone-beam CT scan allows intraoperative control of the anatomic reduction of complex facial fractures, with reconstruction of the facial projections, to avoid secondary corrective surgery.

Summary

The challenge of panfacial fractures can be best met by careful preoperative planning and correct sequencing of treatment.

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Chapter 42

Mandibular Trauma – Principles of Treatment

William Chung and Bernard J. Costello

Over the course of the past three decades, there has been a rapid development of techniques used in the management of mandibular trauma. Today's management of mandible fractures has allowed for decreased infection rates, increased rigid fixation over fracture segments with improved bony union, and a reduction in the use of wire maxillomandibular fixation, resulting in a faster return to function. The goal of this chapter is to present an overview of general treatment principles and outline advances in the management of mandibular fractures.

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Historical background

Contemporary management of mandible fractures has allowed for a predictable outcome with decreased infection rates, increased rigid fixation over fracture segments with improved bony union, and a reduction in the use of wire maxillomandibular fixation, resulting in a faster return to function.¹ The diagnosis and treatment of mandibular fractures was documented as early as 1650BC in an Egyptian papyrus.^{2,3} Hippocrates described the use of external bandages to achieve fracture immobilization.⁴ Salicetti^{5,6} and Gilmer^{7,8} both reported the use of wires to perform maxillomandibular fixation in the treatment of mandibular fractures. Other early methods of fracture repair coincidentally resembled elements of more modern principles of rigid fixation and continued to revolve around Hippocrates' concept of immobilization.⁹

The management of mandibular fractures for the greater part of the 20th century was limited to the

application of maxillomandibular fixation, Gunning-type splints, or external frames used in combination with pin fixation.^{10,11} Fracture treatment by an open approach with direct transosseous wiring was eventually introduced, but was reserved for select cases involving the "edentulous" posterior mandible (i.e. ramus/angle) or in completely edentulous patients.¹² Luhr reintroduced the idea of using miniature bone plates in the repair of mandibular fractures in 1968 and 1972.¹³ In 1976, Speissl and others continued to advance the techniques of open reduction and internal fixation (ORIF) and developed the principles now advocated by the Arbeitsgemeinschaft für Osteosynthesefragen (Association for Osteosynthesis/ Association for the Study of Internal Fixation (AO/ ASIF)).¹⁴ In 1973, Michelet introduced techniques for mandibular fixation using smaller plates along ideal lines of osteosynthesis and Champy further refined and applied these techniques in clinical practice.^{15,16}

Etiology and epidemiology

The etiology of mandibular fractures includes assault, motor vehicle accidents, work- or sports-related injury, falls, projectile missiles, and pathologic fractures. The most common cause of mandibular fractures worldwide is motor vehicle crashes followed by assaults and occupational incidents.^{17–21} The circumstances and pattern of mandibular fractures are also quite variable depending on the population studied and the environment in which they live. More developed countries are likely to have a higher incidence of motor vehicle-related fractures, while underdeveloped nations typically report isolated fractures arising from altercations.^{17–21}

A number of studies that evaluated the location of mandibular fractures report the body as the most common site, at about 33%.^{18–21} Fig. 42.1 depicts the percentage of mandibular fractures based on anatomic location. The condyle and angle were also frequently reported at approximately 29% and 23%, respectively. Fractures occurred at the symphysis in nearly 9% of cases, while all other anatomic locations were infrequently involved. Depending on the mechanism of injury, fractures of the mandible comprise 10–25% of all facial fractures.²² Mandibular fractures are routinely reported as isolated from other facial fractures. Most studies that evaluated the number of mandibular fractures per patient reported a mean of 1.5–1.8 fractures.^{17–21} In numerous studies, more than 50% of all reported mandibular fractures occurred as multiple fractures.^{23–24} Frequent combinations of multiple mandibular fractures are angle and contralateral body, bilateral angle or body, and condylar and contralateral body.²⁵ Assaults typically resulted in left-sided fractures as most individuals are right-hand dominant.

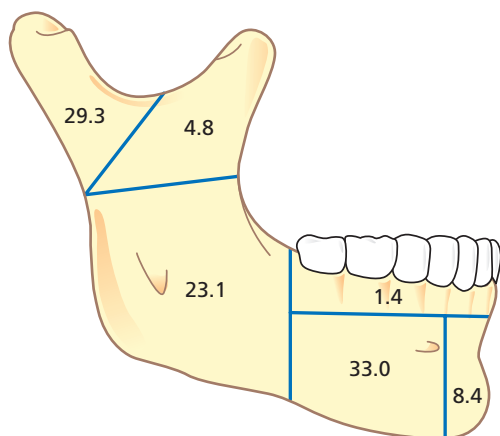


Fig. 42.1 Percentage of mandibular fractures based on anatomic location. (Figure 22-19, p. 413. Chacon G, Larsen P. Principles of management of mandibular fractures. In: *Peterson's Principles of Oral & Maxillofacial Surgery* (Milorio M, ed.), 2nd edn, Vol. 1. Hamilton, Ontario: BC Decker Inc, 2004. Reproduced with permission.)

Classification of mandibular fractures

Numerous classification schemes exist for mandibular fractures. These injuries have been classified based on the location of teeth relative to the fracture, anatomic region of the fracture, and fracture pattern. One particular classification scheme for angle and body fractures correlates the direction of the fracture line on a plain film with the role of the masticatory muscles on the fracture segments. This scheme describes the principle of *favorableness* in a horizontal and vertical plane. Figs 42.2–42.5 illustrate the principles of a favorable vs unfavorable fracture in both the horizontal and vertical plane. A fracture is favorable in a horizontal plane if the fracture resists the superior pull of the proximal fracture segment by the masseter and temporalis muscles. A fracture is favorable in the vertical plane if the fracture resists the medial pull of the proximal segment by the medial pterygoid muscle. Table 42.1 outlines classification schemes based on anatomic location and fracture pattern.

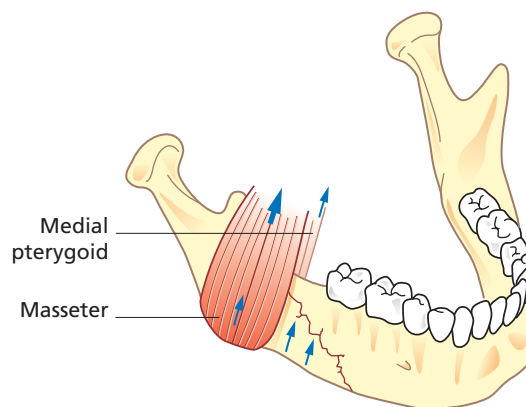


Fig. 42.2 Horizontally favorable fracture. (Figure 18-7, p. 488. Barber HD, Bahram R, et al. Mandible fractures. In: *Oral & Maxillofacial Trauma* (Fonseca R, Walker RV, et al., eds.), 3rd edn. Vol. 1. St. Louis, MO: Elsevier Saunders, 2005. Copyright © 2005 Elsevier.)

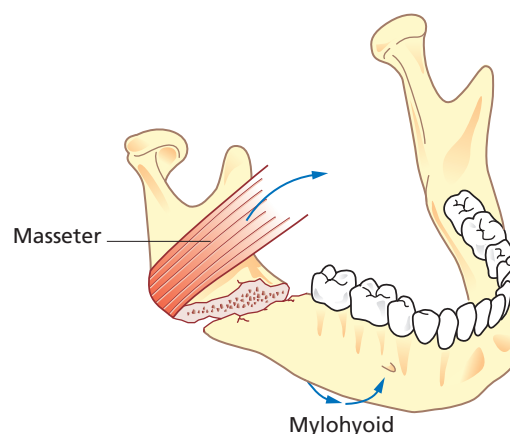


Fig. 42.3 Horizontally unfavorable fracture. (Figure 18-8, p. 488. Barber HD, Bahram R, et al. Mandible fractures. In: *Oral & Maxillofacial Trauma* (Fonseca R, Walker RV, et al., eds.), 3rd edn. Vol. 1. St. Louis, MO: Elsevier Saunders, 2005. Copyright © 2005 Elsevier.)

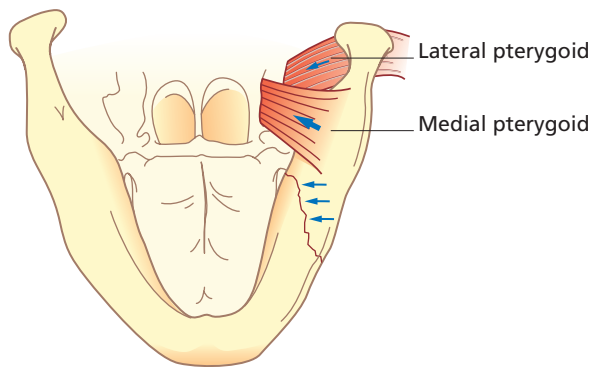


Fig. 42.4 Vertically favorable fracture. (Figure 18-6, p. 488. Barber HD, Bahram R, *et al.* Mandible fractures. In: *Oral & Maxillofacial Trauma* (Fonseca R, Walker RV, *et al.*, eds.), 3rd edn. Vol. 1. St. Louis, MO: Elsevier Saunders, 2005. Copyright © 2005 Elsevier.)

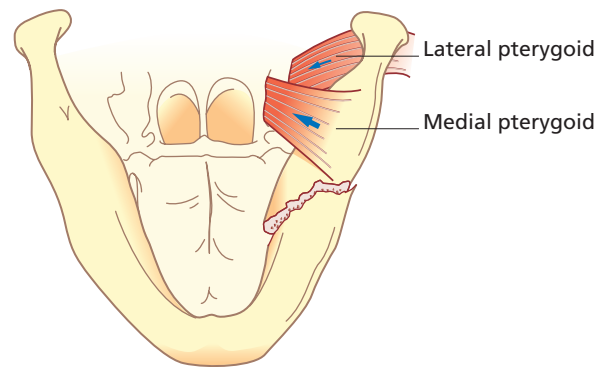


Fig. 42.5 Vertically unfavorable fracture. (Figure 18-5, p. 488. Barber HD, Bahram R, *et al.* Mandible fractures. In: *Oral & Maxillofacial Trauma* (Fonseca R, Walker RV, *et al.*, eds.), 3rd edn. Vol. 1. St. Louis, MO: Elsevier Saunders, 2005. Copyright © 2005 Elsevier.)

Table 42.1 Classification of mandibular fractures.

Anatomic region
Symphysis – fracture involving the area between the lateral incisors extending vertically through the inferior border of the mandible
Parasymphysis – fracture between the mental foramen and the mesial aspect of the canine extending through the inferior border of the mandible
Body – fracture between the mental foramen and the distal aspect of the second molar extending through the inferior border of the mandible
Angle – fracture between the distal aspect of the second molar and the posterior attachment of the masseter muscle extending through the inferior border of the mandible
Ramus – fracture extending horizontally through the anterior and posterior borders of the ramus or extending vertically from the sigmoid notch to the inferior border of the mandible distal to the second molar
Condylar process – fracture extending from the sigmoid notch to the posterior border of the ramus
Coronoid process – fracture involving the coronoid process
Alveolar – fracture confined to the tooth-bearing segment of bone
Fracture pattern
Greenstick – fracture through one cortex and no discontinuity of bone. No mobility of fractured segments. Common in children
Simple – fracture does not communicate with the external environment. A closed fracture
Compound – fracture communicates with the external environment either through the skin or the periodontal ligament of a tooth. An open fracture
Comminuted – multiple segments of bone as a result of greater force from the trauma
Complex – fracture involving damage to adjacent structures
Telescoped or impacted – one fractured segment is driven into the other segment
Pathologic – fracture resulting from normal function in an area of diseased bone
Terms associated with temporomandibular joint (TMJ) fractures
Displaced – movement of the condylar (proximal) segment relative to the remainder of the mandible. Although the condyle is malpositioned, it is still within the confines of the glenoid fossa and has not ruptured the capsule of the TMJ
Dislocated – the condylar (proximal) segment is no longer within the confines of the glenoid fossa
Extracapsular – fracture not involving the capsule of the TMJ
Intracapsular – fracture occurring within the TMJ capsule

Patient evaluation

The evaluation of mandibular fractures begins with obtaining a careful history. Information about the mechanism of injury often suggests a specific fracture pattern and may provide the surgeon with valuable insight regarding the potential for concomitant inju-

ries. In addition to focused questions about the traumatic event, the surgeon must carefully review the patient's past medical and surgical history, medication use, and any drug allergies. Pretrauma temporomandibular joint (TMJ) dysfunction and any associated non-surgical or surgical treatment should be carefully documented.

During the physical examination immediate attention must always be given to any problems associated with airway compromise or hemorrhage. Once the airway, breathing, and circulation have been adequately assessed, a neurologic evaluation should be performed. Standard trauma protocols such as those described in Advanced Trauma Life Support guidelines from the American Red Cross should be used during a comprehensive examination.²⁶ When a mandibular fracture is suspected, a thorough examination of the entire maxillofacial region is critical and should be carried out prior to ordering any radiographic studies.

A number of signs and symptoms are highly suggestive of a mandibular fracture (Table 42.2). Occlusal changes are one of the most common physical findings in patients with fractures of the mandible. Occlusal changes may be the result of dental fractures, fractures of the alveolus, trauma to associated structures of the TMJ, fractures of the maxilla, or contusions of the masticatory muscles. It is important to consider that the patient may have had a pre-existing malocclusion prior to the injury.

Deviation of the mandible on opening is indicative of a condylar fracture. In a unilateral condylar fracture, the mandible deviates to the side of the fracture due to the unopposed action of the lateral pterygoid muscle on the unaffected side and the lack of translation on the affected side. An open bite subsequently develops on the unaffected side and the posterior teeth contact first on the affected side. When bilateral condylar fractures occur the mandible does not necessarily deviate. However, the posterior occlusion does contact prematurely and an anterior open bite subsequently develops.

An altered range of motion can occur with mandibular fractures. The most common cause of limited opening is from pain and reflex guarding associated with the trauma. Decreased opening also occurs when a depressed zygomatic arch fracture mechanically impinges upon the coronoid process of the mandible. Muscle contusions and edema overlying any fracture site can also contribute to limited mouth opening.

A fracture should be considered when the force of a trauma is great enough to cause a laceration involving the perioral structures. Gingival lacerations may

be associated with either dental or alveolar fractures or they may indicate an underlying mandibular fracture. A more extensive degloving injury of the oral tissues may also be consistent with a fracture. A laceration to the chin should raise the surgeon's suspicion for not only a symphysis fracture, but also subcondylar fractures. Sublingual ecchymosis is also consistent for a mandibular fracture.

Lacerations of the lower lip or chin that involve a terminal branch of the mental nerve result in some degree of neurosensory deficit in this region. However, a neurosensory deficit along the distribution of the inferior alveolar nerve occurs when a displaced fracture traverses either the angle or body of the mandible. A non-displaced fracture typically does not manifest any neurosensory changes.

Changes in facial contour are often consistent with mandibular fractures. Bilateral angle or subcondylar fractures often displace the anterior mandible inferiorly, causing an elongated facial appearance. Bilateral parasymphyseal fractures often cause loss of chin projection. Any displaced fracture also creates loss of normal mandibular arch form.

A thorough otoscopic examination should be routinely performed during the evaluation of all maxillofacial trauma patients. However, the otoscopic examination plays a particularly important role for fractures of the condylar process. Dislocation fractures of the condyle can produce bleeding in the external auditory canal if the dislocated condyle tears the lining of the anterior wall of the canal. Basilar skull fractures may also be diagnosed when an otoscopic exam reveals blood behind the tympanic membrane.

Imaging

In clinical situations where the mechanisms of injury or physical examination are suggestive of a fracture, radiographic studies are necessary to confirm the diagnosis and plan subsequent treatment. An overview of radiographic methods in oral and maxillofacial surgery is given in Chapter 2. In the following paragraphs, specific considerations for mandibular fractures are presented.

When an isolated mandibular fracture is suspected, the routine radiographic assessment may consist of a panoramic radiograph and one additional postero-anterior view of the mandible, usually an open-mouth Towne's radiograph.²¹ A panoramic radiograph is the single most comprehensive image and usually allows for satisfactory visualization of all regions of the mandible (condyle, ramus, body, and symphysis) (Fig. 42.6). It is also useful for examining the existing dentition, and showing the presence of impacted teeth with respect to the fracture, the alveolar process, and the position of the mandibular canal.

The Towne's view adds another anatomic dimension and is especially useful in ruling out subcondylar

Table 42.2 Signs and symptoms of mandibular fractures.

Occlusal changes
Deviation on opening
Altered range of motion
Localized pain
Lacerations, ecchymosis, or hematoma
Neurosensory deficits of the inferior alveolar nerve
Changes in facial contour or mandibular arch form
Blood in external auditory canal
Mobility of bone segments/palpable step-offs

fractures. Fig. 42.7 clearly depicts a left subcondylar fracture, which is not as apparent in the panoramic radiograph of the same patient in Fig. 42.6. Some X-ray machines are capable of taking selective tomograms of the temporomandibular regions. Dedicated tomograms or transcranial views of the TMJ can be obtained as separate studies to further delineate the displacement of fractured segments. In situations where a panoramic view of the mandible is not available, a series of different views of the mandible is required to adequately view all of the anatomic regions of interest. This is more labor intensive, costly, and subjects the patient to a higher dose of radiation. Despite the good visualization of the dentoalveolar structures obtained by a panoramic radiograph, additional periapical or occlusal radiographs are often helpful in viewing specific areas of concern in more detail, especially when dental or alveolar fractures are suspected.

Currently computed tomography (CT) scans offer the most detailed and comprehensive view of the facial skeleton. Current protocols allow for axial, coronal, sagittal, and even reconstructed three-dimensional (3D) images to be formatted, as shown in Figs 42.8–42.11. Despite the superior visualization of the fracture(s), the use of CT scans for the diagnosis of isolated mandibular fractures is uncommon and may be cost-prohibitive. A CT scan is typically reserved for cases involving complex (comminuted, avulsive, etc.) mandibular injuries or concomitant midfacial or orbital injuries. In some cases where a dislocated condylar fracture is suspected, the CT scan will allow for detailed 3D imaging. Figs 42.12–42.14 depict both midfacial and mandibular fractures. Another useful application of the CT scan is in situations where the patient is not able to tolerate routine radiographic positioning and techniques (cervical spine or head injury). Very young patients with limited cooperation may also be candidates for CT scans, but may require sedation during imaging. When a CT scan is used, 2mm incremental axial and coronal views typically allow for sufficiently detailed imaging of the facial skeleton.



Fig. 42.7 Towne's radiograph of patient in Fig. 42.6.

Other studies can be helpful in specific scenarios, but are rarely required. Magnetic resonance imaging (MRI) is of very limited value in evaluating bony injuries. It may be helpful to delineate injuries to associated soft tissues or the intracapsular structures of the TMJ. Ultrasound has occasionally been used to determine condylar position after fractures. Angiography in conjunction with interventional radiology techniques can be used in situations where significant bleeding is associated with facial fractures.^{27–32}

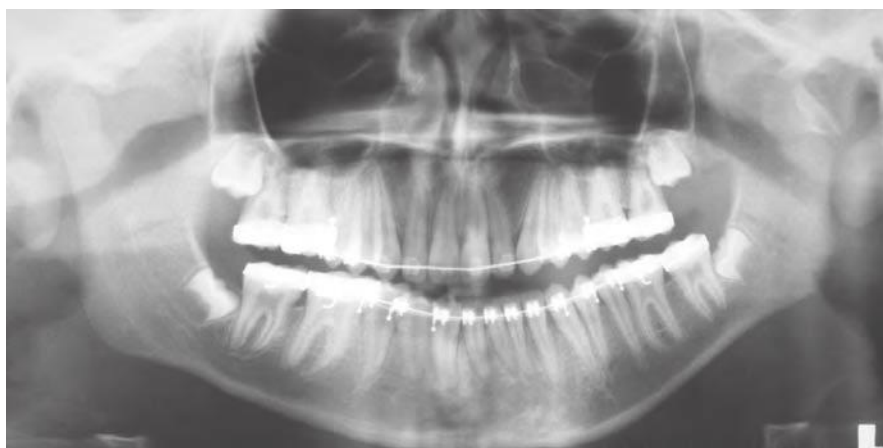


Fig. 42.6 Panoramic radiograph of a patient who sustained a left subcondylar fracture.

Goals of mandibular fracture treatment

The goals of mandibular fracture repair are listed in Table 42.3. The primary goal is re-establishing a stable occlusion. This in turn allows for restoration of a normal mandibular arch form and facial symmetry. Once the occlusion and arch form are restored, emphasis is placed on resuming a normal range of motion, including excursive movements. Lastly, attempts are made to avoid long-term complications that may lead to internal derangement of the TMJ or growth disturbances of the condyle.

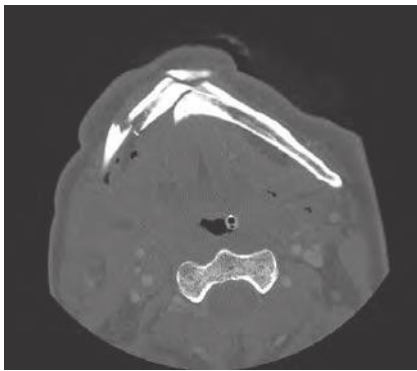


Fig. 42.8 Axial CT scan depicting comminuted symphysis fractures.



Fig. 42.9 Coronal CT scans of patient in Fig. 42.8.

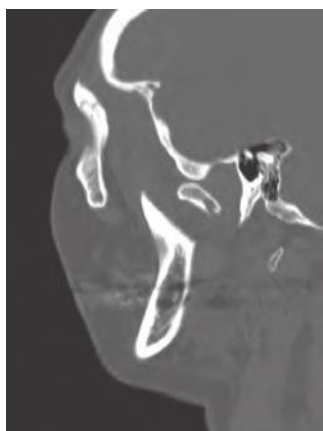


Fig. 42.10 Sagittal CT scan depicting an anteriorly displaced condylar fracture.

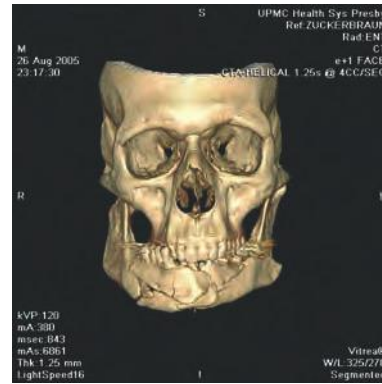


Fig. 42.11 3D reconstructed image of the patient in Figs 42.9 and 42.10. Note additional left angle and right subcondylar fractures.

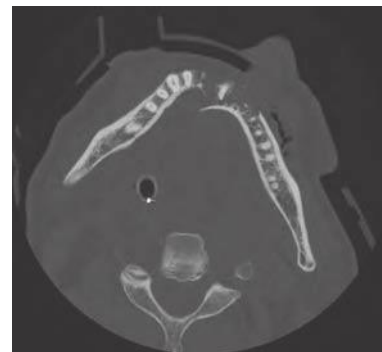


Fig. 42.12 Axial CT scan reveals a comminuted symphysis fracture.

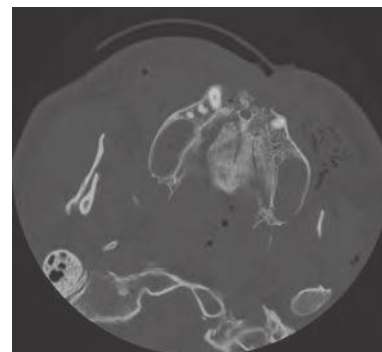


Fig. 42.13 Axial view of concurrent midfacial fractures of patient in previous figure. Note multiple palatal fractures and avulsed maxillary teeth, in addition to right subcondylar fracture.

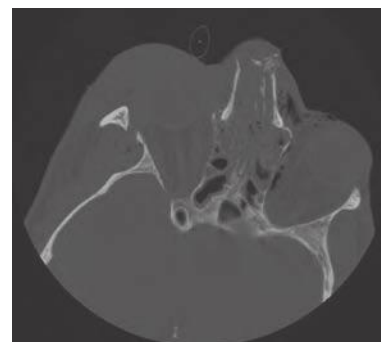


Fig. 42.14 Comminuted nasal complex fractures of patient in Figs 42.12 and 42.13.

Table 42.3 Goals of mandibular fracture repair.

Re-establish a stable occlusion
Re-establish an adequate range of motion
Restore facial and mandibular arch form
Restore pain-free function
Avoid internal derangement of the temporomandibular joint
Avoid growth disturbances of the mandible

Table 42.4 Indications for closed reduction techniques of mandibular fractures.

Non-displaced mandibular fracture
Grossly comminuted mandibular fracture
Atrophic edentulous mandibular fracture
Loss of soft tissue coverage over a fracture
Mandibular fractures in children
Condylar fracture

Treatment options

Closed reduction

Some patients may present with a mandibular fracture yet require no treatment. In these cases the occlusion is stable, the fracture is non-displaced, a favorable fracture pattern exists, and the patient is motivated to be compliant. Management in these rare instances consists of close observation, a liquid diet, and limited physical activity. The surgeon must remain prepared to change the treatment plan should any change in the clinical situation arise. If occlusal discrepancies or other signs of fracture displacement develop, then either closed or open reduction techniques should be implemented early.

Despite the technological advances in maxillofacial plating systems, most mandibular fractures are amenable to closed techniques.^{33–34} Those clinical scenarios well suited for closed reduction are listed in Table 42.4. Most non-displaced stable mandibular fractures lend themselves to closed treatment. Grossly comminuted mandibular fractures are at risk for devitalization and infection of the small bone fragments when open reduction techniques are performed due to the extensive periosteal stripping that is necessary to expose all of the fragments. This is especially relevant in fractures associated with gunshot wounds where additional hard and soft tissue necrosis is possible for several weeks following the initial injury.

A recent systematic review of open vs closed reduction of mandibular fractures showed that there are fewer complications when closed reduction is performed and the study raised doubts regarding the superiority of open reduction and rigid splinting compared to closed reduction and intermaxillary splinting.³⁵ Variables are difficult to control for in these studies, and the concept of what is a preferred outcome is debatable in the current literature. The balance between advantages and disadvantages of a closed vs open technique must be individualized as it should be with the use of all evidence-based decisions.

Atrophic edentulous mandible fractures present a unique challenge from a number of standpoints. These fractures typically occur in the elderly, so healing potential may be suboptimal. Because the blood

supply from the inferior alveolar artery is already compromised, limited osteogenic potential exists. The mandible now relies more heavily on the periosteum for its blood supply. Open techniques require stripping of the periosteum, further prohibiting osteogenesis.

Fractures involving a lack, or loss, of overlying soft tissue are best treated by closed techniques as the presence of hardware may increase the risk of infection. Adequate healing of any fracture is dependent on the blood supply to the area, which is partly contributed to by the quality and quantity of soft tissue coverage over the fracture. A soft tissue flap must be rotated over a fracture if an absolute loss of soft tissue has been determined.

The majority of mandibular fractures in children are best treated with closed techniques (discussed in more detail later in this chapter and elsewhere in this text). Developing tooth buds occupy a significant portion of the mandible and are at risk of injury with the placement of hardware commonly used in open techniques.³⁶ Pediatric patients can be placed into maxillomandibular fixation with circummandibular and piriform rim wires along with interpositional wires placed through the circummandibular wires. Figs 42.15–42.19 show circummandibular and piriform rim wires being placed followed by the maxillo-mandibular fixation wire. Fig. 42.20 shows the post-operative panoramic radiograph of the same patient. Pediatric fractures involving the mandibular arch may also be effectively managed using a lingual splint and circummandibular wires (Figs 42.21 and 42.22).

Most studies also support the treatment of condylar fractures in both children and adults with closed techniques.^{36–42} Regardless of the degree of radiographic displacement of the condylar segment, closed reduction and early postoperative physical therapy achieved excellent clinical results. These same studies also concluded that no correlation exists between radiographic alignment of the fracture and postoperative function.

Closed reduction of fractures is most commonly achieved by applying Erich arch bars to the maxillary and mandibular dentition with 24- or 25-gauge circumdental soft stainless steel wires (Fig. 42.23). The wires are tightened in a clockwise direction while

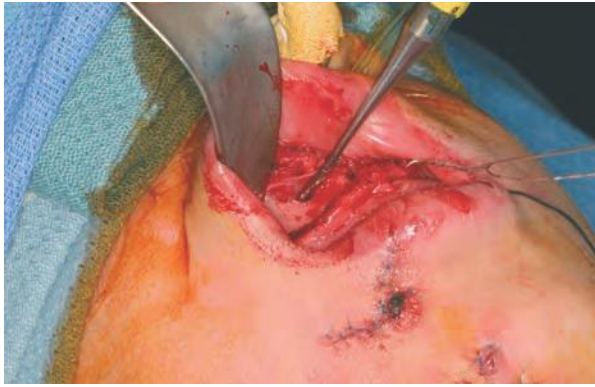


Fig. 42.15 Exposure of a piriform rim for placement of a skeletal fixation wire for a mandibular fracture in a pediatric patient.



Fig. 42.16 Passage of the piriform rim wire.



Fig. 42.17 Sequential passage of a circummandibular wire for skeletal fixation. An awl elevator facilitates passage of the stainless steel 25-gauge wire.



Fig. 42.18 Sequential passage of a circummandibular wire for skeletal fixation. An awl elevator facilitates passage of the stainless steel 25-gauge wire.



Fig. 42.19 Patient from Figs 42.15–42.18 in maxillomandibular fixation via skeletal fixation.



Fig. 42.20 Postoperative panoramic radiograph of the patient in Figs 42.15–42.19.



Fig. 42.21 Lingual splint secured with circummandibular fixation wires.



Fig. 42.22 Patient from Fig. 42.21. The lingual splint covers the occlusal surfaces of both the mandibular and maxillary dentition to allow for indexing of the teeth for ultimate maxillomandibular fixation.



Fig. 42.23 Erich arch bars and interdentally placed stainless steel 25-gauge wires.



Fig. 42.25 Intermaxillary fixation (IMF) bone screws and maxillomandibular fixation wires.



Fig. 42.24 Ivy loops made with 25-gauge stainless steel wires.

applying force in an apical direction. To avoid unnecessarily breaking the interdental wire, the wire is turned until it is lying flush against the arch bar. The end of the wire is then turned on to itself and crimped to prevent irritation or cutting of the gingiva or lip. It is advisable not to use Erich bars for splinting some simultaneously occurring dental injuries. For example repositioned and replanted and root-fractured incisors can be extruded by Erich bars, since the Erich bars are straight and have no occlusal curve built in. In these cases it may better to give the repositioned/replanted tooth a separate splint (see Chapter 39).

Wire loops are then wrapped around the hooks of the arch bars to maintain maxillomandibular fixation. Other closed reduction methods include Ivy loops (Fig. 42.24), Stout wiring, or Ernst and Gilmer ligatures. Some commercially available products have attempted to make the process of applying maxillomandibular fixation faster and easier. Bonded arch bar systems can be technically cumbersome if an absolutely dry field or healthy dentition are not present.

Another system for achieving maxillomandibular fixation involves the use of intermaxillary fixation (IMF) bone screws, which are modified to allow for passage of a wire ligature (Fig. 42.25). One of the potential complications of such a system is that the condyle can be distracted from the fossa in patients who do not have posterior dentition if the fixation

wires are overtightened. Edentulous and partially edentulous patients with severe fractures may benefit from the use of occlusal splints (Gunning-type) to maintain an interocclusal dimension and re-establish arch form. Significant comminution of the mandible with concomitant soft tissue loss may benefit from a period of external pin fixation. External fixators can be easily fabricated from an endotracheal tube and acrylic resin. The reaction is exothermic, so great care should be taken to avoid burning the patient's skin during the bar's fabrication.

Open reduction

Several clinical scenarios support the use of open reduction in the repair of mandibular fractures (Table 42.5). Patients with certain systemic conditions may be more appropriately treated with an open technique. Patients with a severe seizure disorder are unlikely to tolerate maxillomandibular fixation. Patients with other neurologic or psychiatric conditions may also benefit from open techniques as would patients with either decreased pulmonary function or gastrointestinal disorders.

A displaced and unfavorable fracture of the mandible cannot be adequately treated with maxillomandibular fixation alone. The influence of the masticatory muscles on angle fractures or the digastric and suprahyoid muscles on parasymphysal or body fractures causes undesirable rotation of the proximal segments and opening at the inferior border. These changes prevent the lingual cusps of the posterior teeth from

Table 42.5 Indications for open reduction techniques of mandibular fractures.

Systemic disorders
Displaced unfavorable fractures
Multiple facial and/or mandibular fractures
Bilateral condylar fractures and midface fractures
Delayed treatment with soft tissue in between the fracture
Malunion/non-union

occluding and may result in masticatory insufficiency and periodontal changes.⁴³

If a patient sustains multiple fractures of the facial bones and/or the mandible, open reduction of the mandibular fractures allows for a more stable foundation for further reconstruction, especially if other facial bones are involved. When bilateral condylar fractures occur in conjunction with midfacial fractures, at least one of the condylar fractures must be opened and fixated to re-establish the vertical dimension of the face. Further discussion of open techniques for condylar fractures occurs later in this chapter.

Most condylar fractures are best treated with closed reduction for approximately 2 weeks. Then the mandible is actively mobilized to avoid an altered range of motion. When a condylar fracture occurs concurrently with another mandibular fracture, it is favorable to open the non-condylar fracture to allow for the necessary postoperative mobilization of the mandible after the 2-week period of maxillomandibular fixation.

When circumstances such as a closed head injury or other acute illnesses prevent the timely repair of a mandibular fracture, soft tissue may grow in between the fracture segments. The soft tissue within the fracture must be removed and an open approach used to repair the fracture. If a malunion or non-union results from the repair of a mandibular fracture, an open approach is necessary to correct the problem.

With the advent of miniplates and bone screws, it has become widely accepted that most fractures involving the craniomaxillofacial skeleton are now treated with open reduction and internal fixation techniques. An open approach gives the surgeon optimal visualization of the fracture segments, therefore allowing the best possible anatomic reduction. Fractures involving the mandibular symphysis, parasymphysis, body, and occasionally the angle may be successfully approached through a transoral approach. The rich blood supply of the maxillofacial skeleton allows for wide subperiosteal dissection and exposure of the mandible without compromising the vascularity and subsequent healing of the bony segments. A transoral approach begins with a vestibular incision at least 1 cm from the mucogingival junction to avoid damage to the attached periodontal tissues and to allow for greater ease of closure of the incision. In the anterior mandible, the mentalis muscles are initially divided during the dissection. During closure, these paired muscles must be reapproximated using interrupted sutures as a separate layer to avoid either a dehiscence of the hardware or the "witch's chin" deformity. Care should be taken to avoid the mental nerves as they exit the mental foramen near the bicuspid roots. Fig. 42.26 depicts a skeletonized mental nerve carefully positioned over the fixation hardware placed through a transoral incision. Full exposure of the buccal surface of the inferior border is required to place adequate fixation. Fractures of the mandibular angle or ramus can be approached through a poste-

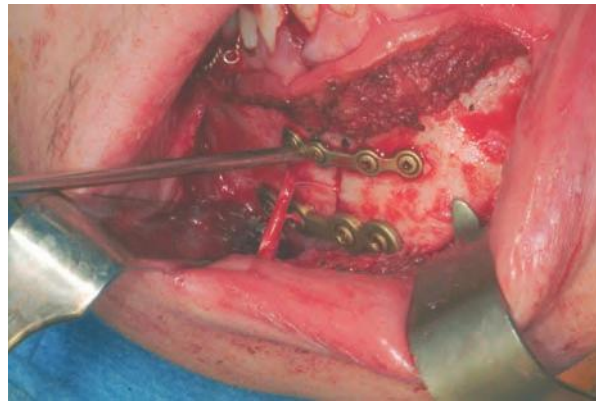


Fig. 42.26 Skeletonized mental nerve positioned over the tension band and fracture plate.

rior vestibular incision. This incision starts just below the external oblique ridge and courses anteriorly approximately 5–7 mm below and parallel to the mucogingival junction. A full-thickness mucoperiosteal flap is elevated. However, a transoral approach may still not allow for direct placement of screws across the fracture site. In such instances the use of a percutaneous trocar may become necessary to allow for the perpendicular placement of the bone screws.

There are also several techniques used to approach the mandible using an extraoral incision, which are reviewed in the Surgical approaches section of this chapter. While it is possible to approach the entire mandible from an external approach, approaches that require skin incisions are usually reserved for fractures involving the posterior body, angle, ramus, or subcondylar regions. Indications for the use of an extraoral approach include the presence of pre-existing lacerations over the fracture, complex fractures, or an edentulous mandible fracture. In edentulous or partially edentulous patients, a vestibular incision may cause scarring and obliteration of vestibular depth, complicating subsequent prosthetic use. Fractures in the anterior region can be approached through a submental incision, but most are dealt with more effectively through an intraoral approach without the need for external scarring.

Surgical approaches

Several factors are considered in the selection of an incision: the degree of exposure required; fracture location; and existing skin crease(s). First described by Risdon⁴⁴ in 1934, a submandibular approach can be used for fractures involving the mandibular angle, ramus, or subcondylar region (Fig. 42.27). The incision is approximately 4 cm long and is placed 1.5–2 cm below the angle and inferior border of the mandible. Placing the incision in an existing skin crease allows the scar to be hidden. The skin and subcutaneous fat are incised until the platysma muscle layer is identified. Once the platysma muscle layer is reached, a nerve stimulator is used to help identify the marginal

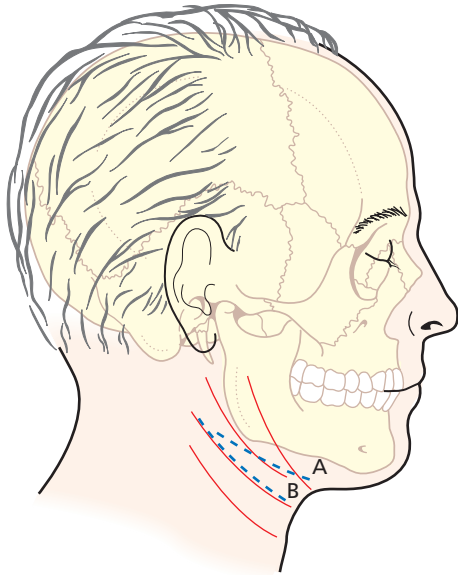


Fig. 42.27 Two locations for a submandibular incision. Incision **A** parallels the inferior border of the mandible. Incision **B** lies within a resting skin tension line. **B** may leave a less conspicuous scar in patients. (Figure 9.3, p. 156 from Ellis E, Zide MF. *Surgical Approaches to the Facial Skeleton*, 2nd edn. Philadelphia, PA: Lippincott Williams & Wilkins, 2006. Reproduced with permission. Copyright © 2006 Lippincott Williams & Wilkins.)

mandibular branch(es) during the remainder of the dissection. The marginal mandibular nerve is located just deep to the superficial layer of the deep cervical fascia. The dissection continues to follow a plane superficial to the capsule of the submandibular gland, proceeding superiorly towards the inferior border of the mandible. The marginal mandibular nerve usually has two branches and typically passes above the inferior border of the mandible proximal to where the facial artery crosses the mandible. If necessary the facial artery and vein can be divided and ligated. Lastly, the pterygomasseteric sling and periosteum are incised to access the fracture segments.

The retromandibular incision also accesses fractures of the mandibular ramus or subcondylar region, as shown in Fig. 42.28. Initially described by Hinds and Girotti,⁴⁵ this incision is similar to the submandibular incision, but it curves upward and behind the angle of the mandible. This approach involves the parotid and masseteric fascia in the course of the dissection. When incising through the masseter muscle, the marginal mandibular and buccal branches must be identified and protected. The primary advantage of the retromandibular incision over the submandibular incision is its greater exposure of the ramus and subcondylar regions.

The preauricular incision is a modification of an incision initially designed by Blair.⁴⁶ The incision provides maximal lateral and anterior access to the joint space. The incision is approximately 3 cm long and has a superior and inferior component, which meet at a point level with the superior aspect of the tragus (Fig. 42.29). The incision is placed within a preauricular skin crease that lies posterior to the superfi-

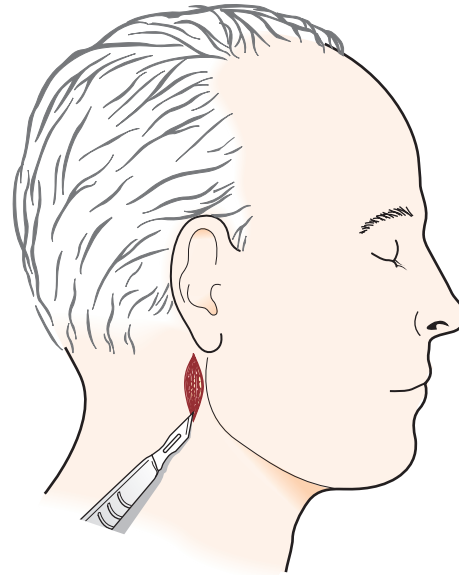


Fig. 42.28 Retromandibular incision. Vertical incision just posterior to mandible. (Figure 10.5 – A only, p. 172 from Ellis E, Zide MF. *Surgical Approaches to the Facial Skeleton*, 2nd edn. Philadelphia, PA: Lippincott Williams & Wilkins, 2006. Reproduced with permission. Copyright © 2006 Lippincott Williams & Wilkins.)

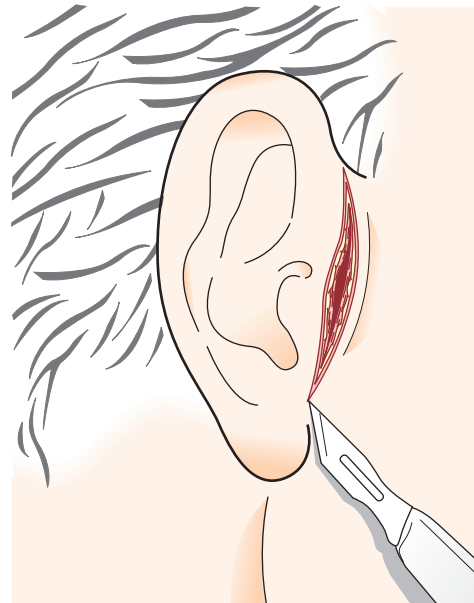


Fig. 42.29 Preauricular incision. (Figure 12.7, p. 199 from Ellis E, Zide MF. *Surgical Approaches to the Facial Skeleton*, 2nd edn. Philadelphia, PA: Lippincott Williams & Wilkins, 2006. Reproduced with permission. Copyright © 2006 Lippincott Williams & Wilkins.)

cial temporal vessels and the auriculotemporal nerve. The vessels can be avoided by following the preauricular cartilage during the dissection. Sufficient surgical access in the inferior portion of the incision can be easily achieved by carrying the incision to a point just below the tragus, but the incision can be extended inferiorly to the level of the lobule.

After sharply incising the skin and subcuticular tissues the superior portion of the incision is progressively dissected with mosquito hemostats to the intensely white temporalis fascia. The inferior por-

tion of the incision is dissected down to the lateral TMJ ligament. Because the external auditory canal is angled nearly 45° anteromedially to the skin surface, the inferior portion of the dissection follows the pretragal pocket to avoid perforating into the external auditory canal. A curved Metzenbaum scissors is placed in the superior aspect of the incision on top of the temporalis fascia and directed inferiorly toward the TMJ ligament. The overlying tissues are sharply incised and a flap is reflected anteriorly and superiorly exposing the temporalis fascia. The flap is also retracted anteriorly away from the tragus. A scalpel is used to make a vertical incision through the temporalis fascia and the dissection proceeds down to the zygomatic arch. Then a full-thickness flap is elevated anteriorly off the arch until the articular eminence is visualized. The lateral TMJ capsule is identified and incised off the lateral lip of the zygomatic arch. Fig. 42.30 depicts the lateral capsular attachment excised off the arch to expose the superior joint space.

Basic principles of internal fixation

The basic principles of mandibular fracture repair using internal fixation can be summarized as reduction, stabilization, and fixation. Open reduction and application of rigid internal fixation is ideally carried out under general anesthesia with a contoured nasal endotracheal (RAE) tube. If a nasal tube cannot be used because of a concomitant midfacial injury preventing safe passage of a nasal tube, then a tracheostomy may have to be considered. If an edentulous space exists in the dentition, then an oral endotracheal tube can be placed in this space.

After an airway is established, arch bars are secured to the maxillary and mandibular dentition using 24 or 25 gauge stainless steel circumdental wires. When possible, a stable and reproducible occlusion is verified. Then the patient is secured into the stable occlusion with ligature wires wrapped around the

hooks of the arch bars. Next, if an extraoral incision is planned, it is demarcated with a sterile surgical marking pen and the soft tissues are injected with a local anesthetic solution containing a vasoconstrictor. Exposure of all fractures is completed using a broad periosteal elevator to allow direct visualization of the fracture segments. When multiple fractures are present, the longest, most stable segments are rigidly fixated first.

Once the bone plates and screws have been placed, the wound is packed and the wire maxillomandibular fixation is removed. Removal of the wires allows for evaluation of not only the restored mandibular arch form but also of the occlusion by moving the mandible. Releasing the maxillomandibular fixation also facilitates closure of extraoral incisions by relaxing the soft tissues in the neck. If a malocclusion develops during the early postoperative period, then training elastics can be placed on the arch bars to assist in the re-establishment of a proper occlusion. The use of elastics limits the degree of mandibular movement, but allows for better oral intake, hygiene, and is often better tolerated by patients. The elastics are also beneficial for neuromuscular training while the patient attempts to return to a normal range of motion. The arch bars and interdental ligature wires are usually removed 4–6 weeks postoperatively. If an ideal anatomic reduction and stable occlusion are achieved intraoperatively, the arch bars and interdental ligature wires may be removed at the end of the surgical procedure.

The primary advantage of rigid internal fixation is that it usually obviates the need for postoperative maxillomandibular fixation and allows for a faster return to function. The application of low-profile titanium alloy plates with self-tapping screws requires less soft tissue stripping than was required in procedures for open reduction with the direct application of transosseous wires. Other advantages of rigid internal fixation include better anatomic reduction, greater stability across the fractured segments, improved healing, and decreased mobility, which further reduce the incidence of infection.

While the details of rigid and semi-rigid fixation are well beyond the scope of this chapter, some basic concepts merit discussion. The introduction of fixation plates and screws has revolutionized the treatment of mandibular fractures. Systems currently used for rigid internal fixation of mandibular fractures include plates and screws of a variety of sizes and configurations. Tension band plates or Champy-style plates generally use 2.0mm screws, while inferior border plates use 2.3–2.7mm screws. At least two screws on either side of a fracture are required for 3D stability, and care is taken to avoid placing screws into the mandibular canal. Plates placed at the inferior border (compression zone) are generally placed through both the outer and inner cortices of the mandible, while superior border (tension band) plates are placed through only the outer cortex.

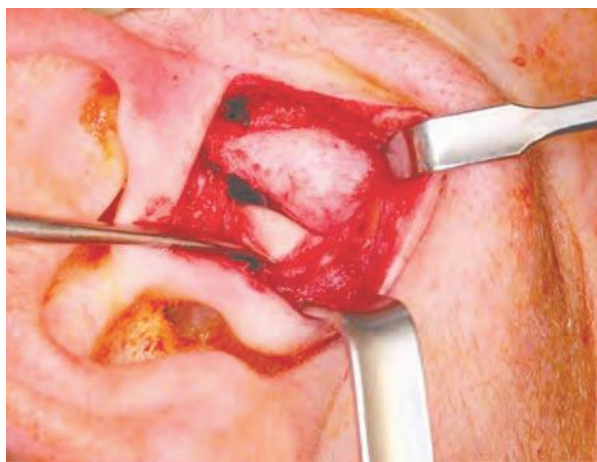


Fig. 42.30 Exposure of the superior joint space via a preauricular incision. Note the superior surface of the TMJ meniscus.

The use of a locking plate and screw has also been advocated in the repair of mandibular fractures. The locking screw has a second set of threads under the head of the screw that engages reciprocal threads on the inner aspect of the hole within the plate. One technical advantage of this design is that the plate does not have to be perfectly adapted against the lateral aspect of the mandible because the screw head “locks” into the hole of the plate. Thus, the locking plate and screw does not require the fracture segments to be compressed against the plate.

More recently, bone plating systems made from resorbable polymers have been used in clinical practice. Early resorbable plating systems were thick and reported to experience cracking under stress, possibly preventing osteosynthesis at the fracture site.⁴⁷ As the systems became more refined, the hardware became thinner and could be placed through transoral incisions. Kim *et al.* studied the use of resorbable plates for mandibular fractures and reported consolidation in all of the patients and an 8% complication rate.⁴⁸ Despite encouraging clinical reports and growing enthusiasm among surgeons, a careful clinical outcome measure analysis on the use of resorbable plating systems must be completed before widespread use is advocated for mandibular fractures. The resorbable materials themselves and the techniques used in their application continue to be redefined.^{49, 50} The topic of resorbable plating systems in the treatment of craniofacial and mandibular fractures is also discussed in Chapters 40 and 41.

The fundamental techniques of rigid fixation include: placement of a tension band, where necessary; passive adaptation of the plate to the properly reduced fracture segments; appropriate positioning of screws in relation to adjacent anatomic structures; placement of screws in accordance with the principles of dynamic compression, where appropriate; and the use of plates and screws of adequate strength and durability. Some surgeons advocate the use of larger screws (2.3 or 2.7 mm) with a fracture plate versus two smaller miniplates. The belief is that the larger fracture plate is necessary to adequately stabilize the segments and neutralize all forces that may occur during function.⁵¹

In 1999, Potter and Ellis examined 46 patients with 51 fractures involving the mandibular angle that were treated using a malleable non-compression plate placed at the superior border of the mandible.⁵² All of the patients had uncomplicated healing of the fractures, suggesting that the required level of fixation for this type of injury is less than previously thought. The advantages of treating fractures with smaller bone plates include less dissection and morbidity. Some of the data showed a decreased incidence of infection in patients who underwent limited dissection and had a less rigid plate placed.⁵³ Insufficient prospective data currently exists to determine which technique is best suited for each fracture of the mandible.

An alternative technique for fixation of mandibular fractures is the lag screw technique. This technique compresses the fracture segments by placing screws through two intact and stable cortices. The hole in the outer cortex is the same diameter as the outer diameter of the screw threads and is also countersunk to receive the head of the screw. This hole is referred to as the gliding hole. The hole in the inner cortex is the same diameter as the internal diameter of the screw. Thus, proper placement of a lag screw requires drilling the hole in the outer cortex slightly larger than the diameter of the inner cortex. When the screw is tightened, the segments are compressed because the head of the screw compresses the outer cortex against the inner cortex while the threads of the screw engage the inner hole (Figs 42.31 and 42.32). A lag screw is placed at a 90° angle to the fracture line to prevent displacement of the fracture segments. Placement of a second lag screw is required to resist rotational forces around the long axis of the first screw.

The lag screw technique offers several advantages over bone plates. First, less hardware is involved. Second, this technique requires less time. Third, the anatomic reduction of the fracture is often better with lag screws than with bone plates. However, the lag screw technique should not be used in either comminuted fractures or fractures involving loss of bone. Lag screw fixation involving the ramus or subcondy-



Fig. 42.31 Axial CT scan of a symphysis fracture that will be reduced and fixated with the lag screw technique.



Fig. 42.32 Placement of a lag screw through the symphysis fracture.

lar region is more technically demanding. Often, a trocar may be necessary to place the lag screw in the posterior mandible without damaging adjacent structures.

Complications of mandibular fracture repair

Complications following mandibular fracture repair are uncommon; infection is the most common. Complications create unique challenges for even the most experienced surgeon and may result in cosmetic and/or functional disturbances. Complication rates have improved since the use of wire fixation, but even the most sound fixation techniques can yield undesirable results.

Infection

Infection is the most common complication of surgical intervention. Lister introduced the concept of asepsis prior to and during an operation.⁵⁴ His concept of decreasing microbial numbers to allow the host to overcome the virulence of the bacteria dramatically reduced infection rates. The potential for infection is always a consideration when treating mandibular fractures, especially when fractures communicate with the oral cavity, as in compound fractures.

Numerous reports have reported various postoperative infection rates associated with mandibular fracture repair, varying from less than 1% to 32%.⁵⁵⁻⁵⁹ A prospective randomized study was performed by Zallen and Curry evaluating the role of antibiotics on dentate patients who sustained compound mandible fractures.⁶⁰ The study revealed an infection rate of 50% at the fracture site for patients who did not receive antibiotic therapy compared to an infection rate of only 6% of those who received antibiotics. The rate of infection did not appear to be related to either an open or closed technique. Abubaker *et al.* performed a prospective, randomized, double-blinded study evaluating the role of postoperative oral antibiotics in uncomplicated mandibular fractures.⁶¹ The study reported no advantage in treating uncomplicated fractures with antibiotics.

One of the many risk factors associated with infection following repair of a mandibular fracture is active substance abuse or patient non-compliance with postoperative regimens.⁶² A significant delay in treatment has also been associated with an increased rate of infection.⁶³ A treatment time between 3 and 5 days after trauma has been shown to be optimal with the lowest rate of infection, in a recent literature review.⁶⁴ Other risk factors include gross contamination of the fracture, pre-existing dentoalveolar disease, teeth within the line of fracture, poor host healing potential, and severe bone resorption patterns associated with edentulous mandibles.

Most infections of the oral cavity are polymicrobial. Both anaerobes and aerobes have been routinely cultured from infections related to mandibular fracture repair. The most common organisms cultured are *Staphylococcus*, α -hemolytic *Streptococcus*, and *Bacteriodes*. Manifestations of an infection include abscess formation, cellulitis, formation of a cutaneous fistula, osteomyelitis, and, rarely, necrotizing fasciitis. Figs 42.33 and 42.34 depict a cutaneous fistula from infected hardware placed for a fracture. Early in the course of the infection, the bacterial population is largely aerobic and Gram positive. As the infection progresses and becomes more chronic, the composition of the involved organisms changes so that anaerobic, Gram-negative organisms predominate. For more detailed information regarding infection in oral maxillofacial surgery see Chapter 29.

Management of these infections begins with a detailed clinical examination followed by appropriate imaging to assess the status of the fractured segments and the hardware. Appropriate laboratory studies, including a complete blood count, should be obtained when clinical indications are present. The use of CT scans or an MRI is appropriate when there is concern that the infection involves the surrounding soft tissues of the neck. Specimens for bacterial culture and sensitivity studies should be sent as early as possible in the patient's clinical course.

Penicillin G, with its excellent Gram-positive coverage, is appropriate for early infections, while clindamycin, with its broader Gram-negative coverage, may become necessary for chronic infections. Antibiotic therapy should begin preoperatively, but the specific length of postoperative antibiotic therapy varies with each circumstance.

It is well documented that the elimination of some of the causative organisms will successfully change the complex microenvironment enough to resolve infections in and around the oral cavity.⁶⁵ Use of broad-spectrum antibiotics in the initial management of an infected fracture is usually reserved for severe infections or when the host's immune responses are



Fig. 42.33 Cutaneous fistula in a patient who had hardware placed for a symphysis fracture.

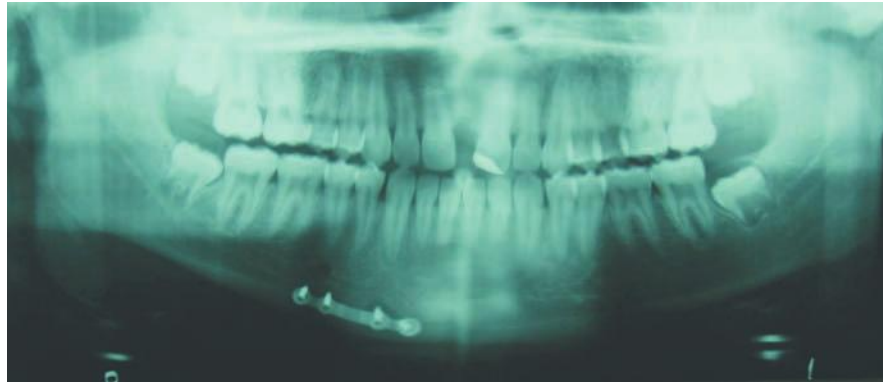


Fig. 42.34 Panoramic radiograph of patient in Fig. 42.33. Note the radiolucency associated with the innermost right fixation screw.

compromised. When initial empiric antibiotic therapy fails to resolve the infection, bacterial culture and sensitivity studies help redirect the surgeon's selection of antibiotics.

Successful resolution of most surgical infections requires three steps: (1) the development of adequate drainage; (2) removal of the source; and (3) appropriate antibiotic coverage. If the infection is the result of a non-restorable tooth or sequestrum, removal of the etiologic agent is required. When fluctuance is present, incision and drainage are necessary to establish prompt resolution. Patients with an infected non-union fracture require debridement and definitive stabilization using one of several methods previously described.

The type of fracture immobilization has been shown to be a factor in the incidence of complications associated with mandibular fractures. Passeri and colleagues reported a 14% rate of infection in mandibular fractures treated with closed reduction.⁶² Infections treated with closed reduction are often associated with either carious or periodontally compromised teeth or teeth within the line of fracture. Luhr examined mandibular fractures treated with closed reduction, dynamic compression plating, and wire fixation.⁶⁶ He found a similar incidence of infection associated with closed reduction and open reduction using dynamic compression plating, but an increased incidence of infection when wire fixation was used. This finding was thought to be due to the mobility of the segments associated with wire fixation. Ellis and colleagues have evaluated multiple types of fixation methods for mandibular fractures.^{53,58,67,68} Ellis observed the highest rates of infection in patients who received two dynamic compression plates. Wire fixation was associated with an infection rate approaching 25%. Champy originally reported an infection rate of only 3.8% associated with his technique of using a 2.0 mm tension band plate via a transoral approach.¹⁶ These data suggest that the use of larger plates may not necessarily be the best treatment for all fracture scenarios. Prospective outcome trials comparing different techniques for similar fracture patterns are needed to properly evaluate these techniques.

Malunion

A malunion is defined as a fracture that has formed a bony union but is misaligned. A malunion usually becomes clinically relevant when it results in a malocclusion. A significant malunion can also result in facial asymmetry and can only be corrected through a carefully planned osteotomy. Malunions are initially treated with conservative therapies such as occlusal equilibration, extended maxillomandibular fixation, or orthodontic mechanics. When the degree of displacement of the segments has significantly altered the occlusion, orthognathic surgery may be necessary to restore normal form and function.

A thorough understanding of dental anatomy and masticatory dynamics is essential in the comprehensive treatment of mandibular fractures to avoid creating a postoperative mandibular arch deformity. Preoperative study models and splint fabrication may aid in fracture reduction in complex cases. Failure to re-establish an anatomic arch form may result in a malocclusion, functional disorders, and facial asymmetry.

Non-union

A non-union exists when bone healing does not occur at a fracture site. A patient experiences pain and clinical mobility over the fracture site in a non-union. A malocclusion is also likely if the non-union occurs in the tooth-bearing portion of the mandible. A radiograph of a non-union often reveals a persistent non-healed fracture with rounded edges adjacent to the fracture segments. The most common cause of a non-union is inadequate reduction and immobilization of the fracture.⁶⁹ In his study of 577 patients, Mathog reported an incidence of non-union fractures of 2.4%.⁷⁰ Other contributing factors of a non-union are infection, severity of the fracture(s),⁷¹ poor blood supply to the mandible, the presence of teeth in the line of fracture,⁷²⁻⁷⁵ metabolic disorders and poor nutritional status,⁷⁶ and patient non-compliance.⁷⁷ Regardless of the surgical technique used, the incidence of a non-union was no greater than 4% in multiple studies.⁷⁸

Treating a non-union begins with identifying and removing the cause of the non-union. If a fracture was inadequately reduced and immobilized, the existing hardware must be removed then properly replaced. All sources of infection must be completely identified. Once the necessary steps have been taken to remove these sources (failed hardware, sequestrum, fistulous tracts, etc.) then appropriate fixation techniques may be used to refixate the fracture. Bone grafting procedures may become necessary when a continuity defect results from the removal of non-viable bone along the edges of the fracture segments.

Nerve injury

The inferior alveolar nerve is the most commonly injured nerve resulting from a mandibular fracture.⁷⁹ Fractures that involve the mandibular body or angle will frequently result in some degree of neurosensory disturbance depending on the degree of fracture displacement. Other branches of the mandibular nerve (auriculotemporal, masseteric, buccal) are at risk of injury usually from a laceration rather than an isolated fracture. The presence of any sensory disturbance following a mandibular fracture should be documented both pre- and postoperatively. A detailed objective neurosensory examination should be performed when the patient reports no improvement in the level of sensation after 6–8 weeks. Most sensory dysfunction improves with time unless the initial injury resulted in a severely displaced fracture and gross disruption of the nerve.

Few studies have focused on sensory deficits associated with mandibular fractures. One particular study by Moore and colleagues reported an incidence of paresthesia of the mental nerve of less than 2%.⁸⁰ They also reported a rate of facial nerve injury of 1.8%. Permanent sensory dysfunction of the mental nerve following mandibular fractures was reported by Larsen and Nielsen.⁸¹ Branches of the facial nerve are also at risk of injury as a result of mandibular fractures. Several studies reported paralysis of the facial musculature following condylar fractures.^{82–84}

Temporomandibular joint dysfunction and ankylosis

Condylar fractures can result in internal derangement of the TMJ. De Riu *et al.*⁴⁰ reported a higher incidence of TMJ pain, joint noises, and deviation on opening in patients who sustained fractures of the condyle. Internal derangement can develop both on the side of the fracture and in the contralateral TMJ. Certain condylar fractures may create a unilateral hinge motion on the affected side. This scenario has resulted in overfunction in the contralateral joint and subsequent hyperactivity and disc dislocation.⁸⁵ Chuong and Piper described using an open technique with direct repair of the altered soft tissues to avoid internal

derangement, but no long-term data exist to support their technique.⁸⁶

Ankylosis is a rare complication of condylar fractures and usually occurs with either an intracapsular fracture in children or prolonged immobilization for the treatment of a mandibular fracture. Ankylosis is thought to develop from intracapsular hemorrhage with subsequent pathologic fibrosis.⁶⁹ If the ankylosis is untreated, it results in underdevelopment of the mandible on the fractured side. Ankylosis can be treated by a gap arthroplasty and any number of adjunctive procedures such as a coronoidectomy and replacement of the condyle with a costochondral rib graft in children or a prosthetic joint in adults. Shorter periods of maxillomandibular fixation, aggressive postoperative physiotherapy, and compliance with follow-up visits are all essential in avoiding ankylosis of the TMJ.

Growth disturbances

Condylar fractures may result in growth disturbances of the mandible. It was previously thought that the cartilage associated with the condyle served as a growth center. However, Durkin *et al.*⁸⁷ believe that the condylar cartilage behaves as a remodeling center after a traumatic injury. They describe a process where the condyle can resorb and a new condylar apparatus subsequently develops. However, this new condyle can grow to larger proportions, especially if the joint space on the affected side is not appropriately maintained and the occlusion is not properly re-established.

Complete condylar regeneration can occur in children, especially in children less than 12 years of age and in those who are still actively growing.^{38,88} This finding was supported by studies that surgically induced fracture dislocations of the condyle in monkeys.⁸⁹ No growth disturbances were noted in any of these monkeys. Thus, the ability of young children to resume normal growth of the mandible contradicts the previously held belief that condylar fractures produced a much greater risk of growth disturbances in younger children.⁹⁰

Facial widening

Widening or flaring at the mandibular angles often occurs after a mandibular symphysis fracture, bilateral condyle fractures, or both. The action of the tongue and suprahyoid musculature causes flaring and lingual tipping of the buccal segments. The fracture line on the outer cortex at the symphysis appears non-displaced. However, direct visualization of the inner cortex reveals separation at the fracture. Ellis describes putting pressure in an inward or medial direction at the gonial angles during the reduction of the fracture(s) while placing the fixation, to avoid this complication.⁹¹

Special considerations

Teeth in the line of fracture

In years past any tooth adjacent to a fracture line was removed because the tooth was considered a direct communication between the fracture and the oral cavity by way of the periodontal ligament. However, several studies concluded that not all teeth required removal when antibiotic therapy and rigid fixation were appropriately implemented.^{74,92,93} Shetty and Freymiller discussed the clinical indications for removal of teeth in the line of fracture (Table 42.6).⁹⁴ These indications hold particularly true for an impacted mandibular third molar associated with an angle fracture. When the third molar does not prevent the fracture from being reduced or is not in direct communication with the oral cavity, the tooth does not need to be removed.

Condylar fractures

The treatment of condylar fractures remains a highly controversial topic in oral and maxillofacial surgery. Each case must be individually evaluated before a particular treatment plan is initiated. Early classification systems of condylar fractures, such as those by Lindahl⁹⁵ and Thoma,⁹⁶ were based on anatomic location of the fracture and the relationship of the condyle to the remaining mandible and the glenoid fossa. Although comprehensive, these particular classification systems did not assist in directing treatment option(s) or affecting the clinical outcome.

MacLennan⁹⁷ developed a classification system that described the degree of fracture displacement, making his scheme more useful in selecting a surgical treatment plan. A type I fracture is non-displaced, whereas a type II fracture involves deviation of the proximal or condylar segment with a simple angulation but no overlap or separation of the fracture segments. In a type III fracture, the condyle is displaced and the fracture segments are overlapped. Lastly, type IV fractures are fracture dislocations where the condyle is completely dislocated out of the confines of the fossa and capsule.

Table 42.6 Indications for removal of teeth in the line of fracture.

Grossly mobile teeth with periapical pathology or significant periodontal disease
Partially erupted third molars with pericoronitis or an associated cyst
Teeth that prevent adequate reduction of the fracture
Teeth with fractured roots
Teeth with exposed root surface from the apex to gingival margin
Excessive delay from the time of fracture to definitive treatment

The goals of condylar fracture treatment include the re-establishment of form and function, which include restoring the occlusion, range of motion, and facial symmetry. Table 42.7 lists both early and late complications from either untreated, or improperly treated, condylar fractures. Currently insufficient clinical data exist to support the use of open reduction for condylar fractures in children or adults. Most condylar fractures are best treated with closed reduction. Contemporary management of condylar fractures now includes endoscopic techniques, which are discussed in greater detail elsewhere in this textbook.

Several absolute indications exist to support the use of open reduction techniques for certain condylar fractures (Table 42.8). Open reduction is performed in situations where closed reduction is not possible or mandibular dysfunction is likely to result if an open reduction is not performed. Mandibular dysfunction may be associated with the following: a fracture dislocation that creates a mechanical stop; displacement of the condyle into the middle cranial fossa; a foreign body in the joint space; or a lateral extracapsular dislocation of the condyle.

Relative indications also exist to support the use of open techniques when treating condylar fractures, as listed in Table 42.9. Certain systemic conditions favor the use of an open technique, such as poorly controlled seizures, severe mental retardation, or psychiatric disorders. Patients with closed head injuries may not tolerate maxillomandibular fixation unless a concurrent tracheostomy is planned. If a tracheostomy

Table 42.7 Complications of condylar fractures.

Early complications
Fractured tympanic plate
Fractured glenoid fossa with or without displacement of the condylar head into middle cranial fossa
Injury to cranial nerves V or VII
Vascular injury
Late complications
Malocclusion
Growth disturbances
Internal derangement of TMJ
Ankylosis

Table 42.8 Absolute indications for open reduction of condylar fractures.

Fracture that prevents placement of the teeth into a stable and reproducible occlusion
Fracture dislocation that creates a mechanical stop and prevents mandibular function
Dislocation of condylar head into middle cranial fossa
Foreign body in the joint space
Lateral extracapsular dislocation of the condyle

my is not desired, then an open reduction may be indicated.

Patients who sustain bilateral condylar fractures and concomitant comminuted midface fractures require re-establishment of the vertical dimension of their midface. An open reduction of at least one of the condylar fractures allows the the midfacial height to be restored, especially if the comminuted midface fractures cannot be rigidly fixated. An open reduction is also favorable when bilateral condylar fractures prevent a stable and reproducible occlusion from being achieved. Another scenario amenable to open reduction is in the presence of a displaced condylar fracture and an inability to perform maxillomandibular fixation due to a lack of pre-existing dentures or severe alveolar bone resorption preventing the fabrication of a lingual splint.

Reports by both Ellis and Dean⁹⁸ and Mikkonen *et al.*⁹⁹ describe using an extraoral vertical ramus osteotomy with removal of the posterior ramus for access to severe medial dislocations of high condylar fractures. This technique allows for improved visualization of and access to the proximal segment for adequate reduction. Figs 42.35–42.38 show a CT scan of a patient with bilateral dislocation fractures of the condyles and a concurrent symphysis fracture. The only major concern with this technique is the theoretical risk of avascular necrosis of the condylar head. However, both studies reported minimal dysfunction and no radiographic evidence of irreversible changes of the condyle.

A technical point of interest involves “preplating” the posterior ramus at the osteotomy site. A reciprocating saw is used to demarcate the desired location of the vertical osteotomy on the posterior ramus. Rigid fixation hardware is placed over the osteotomy, then removed in order to complete the cut. Once the posterior ramus and condyle are removed from the surgical field, the two segments are anatomically reduced and rigidly fixated, as shown in Figs 42.39–42.42. Preplating of the posterior ramus before the osteotomy is completed allows the posterior ramus–condylar unit to be reinserted *in situ* in the intended position without any subtle, unwanted positional changes of the two segments. Figs 42.43 and 42.44 depict the fixation of the symphysis fracture and the postoperative panoramic radiograph, respectively.

Table 42.9 Relative indications for open reduction of condylar fractures.

Medical conditions that are not compatible with maxillomandibular fixation
Bilateral condylar fractures and comminuted midface fractures
Bilateral condylar fractures prevent a stable and reproducible occlusion
Displaced condylar fractures and an inability to use maxillomandibular fixation because of an absence of pre-existing dentures or severe alveolar resorption preventing splint fabrication



Fig. 42.35 Axial CT scan depicting bilateral dislocation fractures of the condyles.

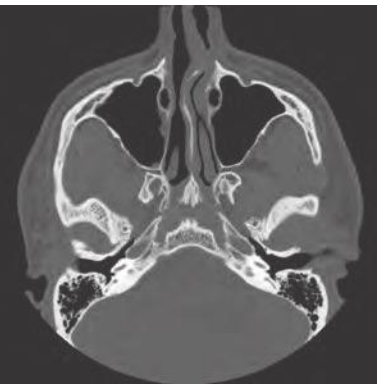


Fig. 42.36 Axial CT scan of patient in Fig. 42.35. Note the lack of the condyles in the glenoid fossae.



Fig. 42.37 Coronal CT scan of patient in Figs 42.35 and 42.36. Both condyles remain vertically upright but anteriorly dislocated.



Fig. 42.38 Axial CT scan of same patient. Note concurrent symphysis fracture

The undersurface of the TMJ meniscus can be directly examined, and if a repair is necessary it can be performed after the posterior ramus and condyle have been removed. Fig. 42.45 shows the undersurface of a TMJ meniscus after removal of the dislocated condyle. Placement of a moist gauze sponge in the undersurface of the meniscus while the ramus and condyle are being reduced and fixated prevents collapse of the joint space from a clot or surrounding soft tissue edema.

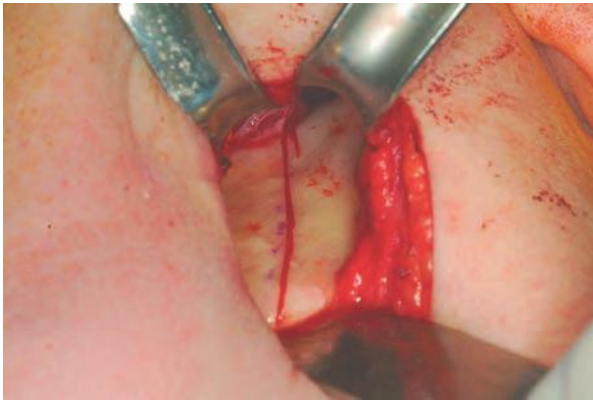


Fig. 42.39 Exposure of left lateral ramus to access dislocated condyle. Osteotomy is started with a reciprocating saw.

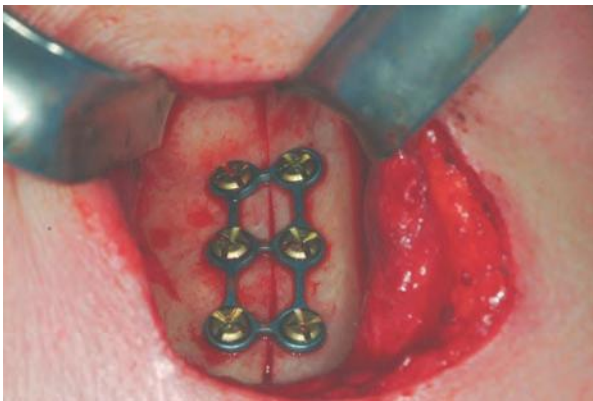


Fig. 42.40 "Preplating" of lateral surface of ramus prior to completion of the osteotomy. An identical procedure was performed on the right side.



Fig. 42.41 Posterior view of reduced and fixated left condylar fracture. An identical procedure was performed on the right side.



Fig. 42.42 Medial view of reduced and fixated left condylar fracture.



Fig. 42.43 Rigid fixation of symphysis fracture via a transoral approach.



Fig. 42.44 Postoperative panoramic radiograph of repaired bilateral condyle and symphysis fractures.

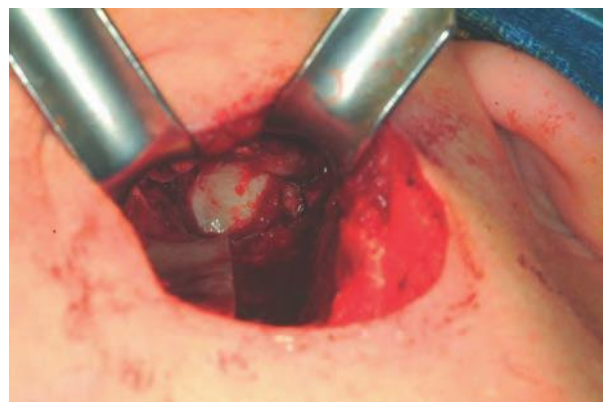


Fig. 42.45 Inferior view of left TMJ meniscus after removal of the dislocated condyle.

Comminuted fractures

The treatment of comminuted mandibular fractures merits special consideration because these fractures are technically more difficult to repair and are associated with a higher rate of complications. Reasons for higher complication rates include: (1) injury to the surrounding tissues from the increased force necessary to create this type of fracture; (2) greater technical difficulty with reduction and stabilization of the fractured segments; and (3) a higher risk for ischemia or avascular necrosis of the bony segments. Despite the rich vascular supply of the maxillofacial skeleton, the smaller bone fragments involved in comminuted fractures may be stripped of their blood supply and become non-viable. The resultant necrosis may lead to either an infection or a non-union of the fracture, which could evolve into a continuity defect. Open treatment of severely comminuted fractures requires enough soft tissue dissection for adequate exposure and visualization, while preserving as much periosteal attachment to the segments as possible so as not to compromise blood supply. In some situations, bone fragments may be replaced as free bone grafts with the application of fixation screws. The use of a locking reconstruction plate is well suited for repair of large comminuted fractures of the mandible. This type of fixation allows the surgeon to bridge the area of comminution and fixate the plate to the proximal and distal intact bone segments. The comminuted fragments can then be reduced and stabilized using positional screws.

Edentulous mandible fractures

Edentulous mandible fractures present a unique and difficult challenge. The mandibular atrophy is both an etiologic factor of the fracture itself and a problematic consideration in the repair of the injury. Previous methods of treatment have included closed reduction of the fracture using the patient's existing dentures with circumskeletal wires, external fixators, internal wires, small plates and screws (both sub- and supra-periosteal), reconstruction plates and screws, and lag screws. As our understanding of fixation methods improves, the ability to manage these clinical challenges has become more predictable.

Several factors may contribute to an unfavorable result in the repair of edentulous mandible fractures. Bradley reported that elderly patients have decreased vascularity to the mandible secondary to the decrease in flow from the inferior alveolar artery.¹⁰⁰ Unlike younger individuals, the blood supply to the edentulous mandible is primarily from the periosteal envelope. An edentulous mandible also has dense, sclerotic bone and decreased osteoblastic activity along the fracture line compared to the dentate mandible. The elderly may also have any number of systemic conditions that affect fracture healing such as diabetes, osteoporosis, vascular disease, renal insufficiency, or

calcium wasting disorders. All of these factors contribute to poor healing potential at the fracture site and an increased rate of a non-union, malunion, or infection.^{101,102}

Most edentulous mandible fractures occur at the body and condyle rather than the angle or parasymphysis regions. The body of the mandible is generally weakest at the mid-body, or saddle, as this is the most significant site for bone resorption. Without dentition to stabilize the segments, the majority of edentulous segments will be significantly displaced from the resultant muscular pull on the smaller and weaker bones.^{101,102}

The degree of atrophy is a factor when choosing a particular surgical option. Closed reduction for edentulous mandible fractures is an effective treatment option, but multiple favorable conditions must exist.¹⁰³ Patients should exhibit adequate bone quantity and quality, minimal swelling, good ridge form, adequate vestibular depth, favorable fracture angulations, good healing potential, and compliance. Immobilizing the segments can be difficult even when the prosthesis fits extremely well. Closed reduction is a reasonable option in the patient who is either unable or unwilling to undergo a more extensive procedure. Longer periods of maxillomandibular fixation are recommended in the elderly patient to ensure adequate healing. Contraindications to maxillomandibular fixation include seizure disorders, psychiatric disorders, significantly altered neurologic states, and significantly compromised pulmonary or nutritional states.

An external fixation device can be used in patients who have grossly infected and/or massively comminuted mandible fractures. It is also useful in those patients with pathologic fractures due to osteoradionecrosis (ORN) or other bony lesions. External fixation is useful in patients with ORN while they receive hyperbaric oxygen therapy in anticipation of surgery that would require periosteal stripping and possible devitalization of bony fragments. External fixation is a very effective therapy despite its limited indications. However, patients will frequently not readily accept the external fixation device due to its unsightliness. The device is often made by securing an endotracheal tube to the transcutaneous fixation pins then injecting an acrylic mixture into the endotracheal tube and allowing it to set. Caution must be used due to the exothermic reaction associated with the acrylic resin. Diligent wound care of the pin sites is critical to prevent an infection, and there must be regular follow-up to evaluate healing and assess the need for debridement.

Open reduction techniques for edentulous mandible fractures must be tailored to the pre-existing morphology of the mandible and severity of the fracture. Transosseous wiring is fraught with complications because of the lack of fracture immobilization and the degree of periosteal stripping required. The use of arch bars with maxillomandibular fixation to reduce

the functional forces on the mandible is not possible, so the wired segments must withstand displacement. Depending on the quality and quantity of bone present and the degree of bone-to-bone contact at the fracture site, the same complications for transosseous wiring may result with the use of small bone plates for fixation of an edentulous fracture.

Open reduction with application of adequate sized rigid internal fixation provides stability and the most predictable healing for edentulous fractures.^{104,105} Typically, this requires the use of at least 2.0 mm bone screws and plates with at least three screws on each side of the fracture. Larger reconstruction plate and screw systems have been used over large areas of the edentulous mandible to ensure rigid fixation of each segment. The disadvantages of this technique include a higher chance for nerve impingement, the need for excessive amounts of periosteal stripping (compromising the blood supply), and the use of larger screws, which can fracture the severely atrophic mandible during placement. Smaller plates and screw systems can be used with less periosteal stripping and more precise screw placement.

In addition to adequate reduction and stabilization of the fractured segments, the successful management of edentulous fractures requires that consideration be given to the amount of bone present. When the mandible is severely atrophic, it is possible that healing will not occur even if open reduction and internal fixation principles are properly applied. In these rare circumstances, the treatment consists of concurrent bone graft reconstruction at the time of fracture repair. This methodology is also appropriate for patients presenting with a non-union of an edentulous fracture. In most cases definitive prosthetic reconstruction is delayed until complete bony healing has occurred. Some authors advocate early reconstruction with bone grafts and osseointegrated implants.¹⁰⁶

Pediatric mandible fractures

The bones of the midface are relatively protected early in life. Thus, midfacial fractures are rare in infants and young children.¹⁰⁷ In contrast, the mandible is more exposed and prone to injury in all age groups. Fracture patterns depend on the level of mandibular development. Compression fractures of the condyle are common in early childhood due to a short thick condylar neck and high cancellous to cortical bone ratio. The risk of condylar neck fractures increases as the child grows and the condylar neck elongates.

The vascular supply of the mandible arises from the periosteum and an endosteal supply, i.e. the inferior alveolar artery. The blood supply from the endosteal route is more prominent in children, while the blood supply from the periosteum is more important in adults.¹⁰⁸ For these reasons the healing potential of pediatric bone is greater, affording flexibility when

using closed techniques to repair mandibular fractures that might result in a non-union in an elderly patient.

Children comprise approximately 5% of all mandibular fractures. Anatomic differences between the pediatric and adult mandible influence the occurrence and pattern of fractures. Pediatric patients often sustain either a minimally displaced or greenstick fracture of the mandible because of the greater elasticity of the mandible and the presence of developing tooth buds. Mandibular fractures in children should raise the clinician's suspicion for concomitant injuries. Kaban *et al.*¹⁰⁹ reported that 76% of pediatric patients with a mandibular fracture also had other associated injuries.

Physical findings on pediatric trauma patients are similar to those of adults: malocclusion, limited opening, deviation on opening, tooth-related injuries, intraoral ecchymosis or lacerations. Chin lacerations often accompany condylar fractures from a fall. The imaging study of choice in the evaluation of pediatric mandibular fractures is the panoramic tomogram. When circumstances do not allow for such an image, then either a mandibular plain film series or a CT scan of the maxillofacial bones can be obtained.

Non-displaced fractures in a pediatric patient who is able to understand and comply with specific instructions may be managed conservatively with a liquid diet and close observation. However, most other fractures, especially those that occur closer to adolescence, are treated with a 2-week period of immobilization.¹¹⁰ Placement of interdental wires and arch bars on primary teeth or newly erupting permanent teeth is difficult and often not feasible because these teeth do not have a sufficient cervical height of contour. Thus, immobilization must take place in the form of either an acrylic lingual splint and circummandibular wires or maxillomandibular fixation with circumsketal wires. Postoperative physiotherapy is essential to regain a complete range of motion. If early mobilization is not undertaken, then the patient may experience muscle atrophy, joint hypomobility, or, in extreme cases, ankylosis. Most pediatric patients avoid mandibular growth disturbances with conservative therapy alone following condylar fractures.^{33,34} If a future mandibular growth disturbance is of concern, the prevailing opinion is to allow remodeling to occur then reassess mandibular growth after the facial skeleton has matured.

Conclusions

The successful management of any mandibular fracture requires a thorough understanding of dental and mandibular anatomy, occlusion, related biomechanical forces, and articulations. Most mandibular fractures can be treated adequately with closed techniques.¹¹¹ More contemporary management relies on open techniques that are based on a proper

diagnosis and classification, anatomic reduction, stabilization with respect to pre-existing occlusal relationships, and appropriate rigid internal fixation techniques. Although the subject of mandibular fractures has been one of the most studied areas in oral and maxillofacial surgery, few prospective data exist regarding the outcome of the various treatment modalities. Retrospective studies offer some evidence that certain open techniques have independently done better than others, but more continued research in materials and outcomes will further refine the treatment of mandible fractures and improve the quality of life for these patients.

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Chapter 43

Transoral Endoscope-assisted Treatment of Displaced Condylar Mandible Fractures

Ralf Schön and Rainer Schmelzeisen

Transoral endoscope-assisted treatment, using an angulated drill and screwdriver, is an alternative to extraoral approaches for the surgical management of displaced condylar fractures; it has been developed in some centers over recent years. Facial nerve injury and visible scars are avoided using the transoral endoscope-assisted approach. Today's indications for the transoral endoscope-assisted surgical approach are presented as well as recent refinements of this surgical technique for the treatment of condylar fractures.

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Fractures of the mandibular condyle are common and account for 9–45% of all mandibular fractures.^{1,2} Shortening of the ascending ramus in displaced condylar fractures causes functional impairment with malocclusion, limited range of motion of the mandible, and development of a contralateral open bite in unilateral and an anterior open bite in bilateral fractures. Surgical reduction with an extraoral approach remains controversial due to possible complications, such as the risk of facial nerve injury and creation of visible scars, and for this reason non-surgical treatment is still most widely employed.^{3–7} However superior functional results are reported after surgical anatomic reduction and fixation for displaced and dislocated condylar fractures, compared to non-surgical treatment.^{5,7–9}

The transoral approach for the reduction of condylar mandible fractures has been demonstrated to reduce the risk of facial nerve injury and does not

leave any visible scars.^{10–13} However, it is a technically demanding method with limited visibility of the fracture site and has not gained wide acceptance.

Due to recent development of endoscopic instruments, especially the chip camera technology, endoscope-assisted techniques have become more widely accepted in oral and maxillofacial surgery, such as in temporomandibular joint (TMJ) arthroscopy and sialoendoscopy. Superior visibility can be achieved through limited incisions with the transoral approach for endoscope-assisted surgical reduction of mandibular condyle fractures.^{13,16,17}

Initially the transoral endoscope-assisted approach was only recommended for laterally displaced subcondylar fractures and satisfying results were obtained.^{13–15} Following the good initial results and after a steep learning curve, the transoral endoscope-assisted approach has also become a routine procedure for the treatment of medially displaced and dislocated condylar fractures over the last decade.^{16,17}

Indications

The indication for surgical treatment of condylar mandible fractures is displacement and dislocation of the fractures causing malocclusion and functional impairment with a limited range of motion. A contralateral open bite is typical for unilateral and an anterior open bite for bilateral condyle fractures, with displacement demonstrating shortening of the height of the ascending mandibular ramus. The size of the fragment is a limiting factor for surgical treatment of condylar neck fractures, and surgical treatment is only performed when at least two screws can be anchored in the condylar fragment. For this reason high condylar fractures, such as intracapsular and condylar head fractures, are treated non-surgically. Edentulous patients are treated with surgical reduction and fixation only when they present with severe pain on motion in displaced subcondylar and low condylar neck fractures.

Contraindications

Non-surgical treatment is recommended in undisplaced condylar fractures without functional disturbances, in high condylar neck fractures, intracapsular and condylar head fractures, in displaced fractures without functional impairment, and also in the majority of pediatric patients and edentulous patients. Non-surgical treatment can be followed by functional treatment with physiotherapy or orthodontic devices, such as an activator, to reduce functional impairment.¹⁸

Pre-, intra-, and postoperative evaluations

The type of fracture, degree of displacement, and result of reduction can be evaluated preoperatively, intraoperatively, and postoperatively with the endoscope and by Townes' and panoramic radiographs, and in selected cases by computed tomography (CT) or cone-beam CT (Figs 43.1–43.6). If intraoperative cone-beam CT scanning is available, control of the reduction can be performed in the operating room. However, due to the precise endoscopic control of the

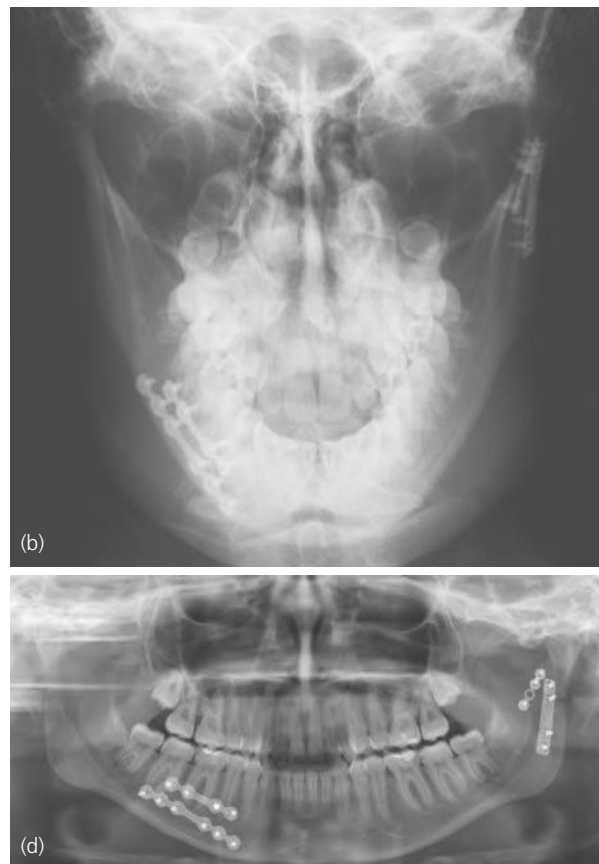
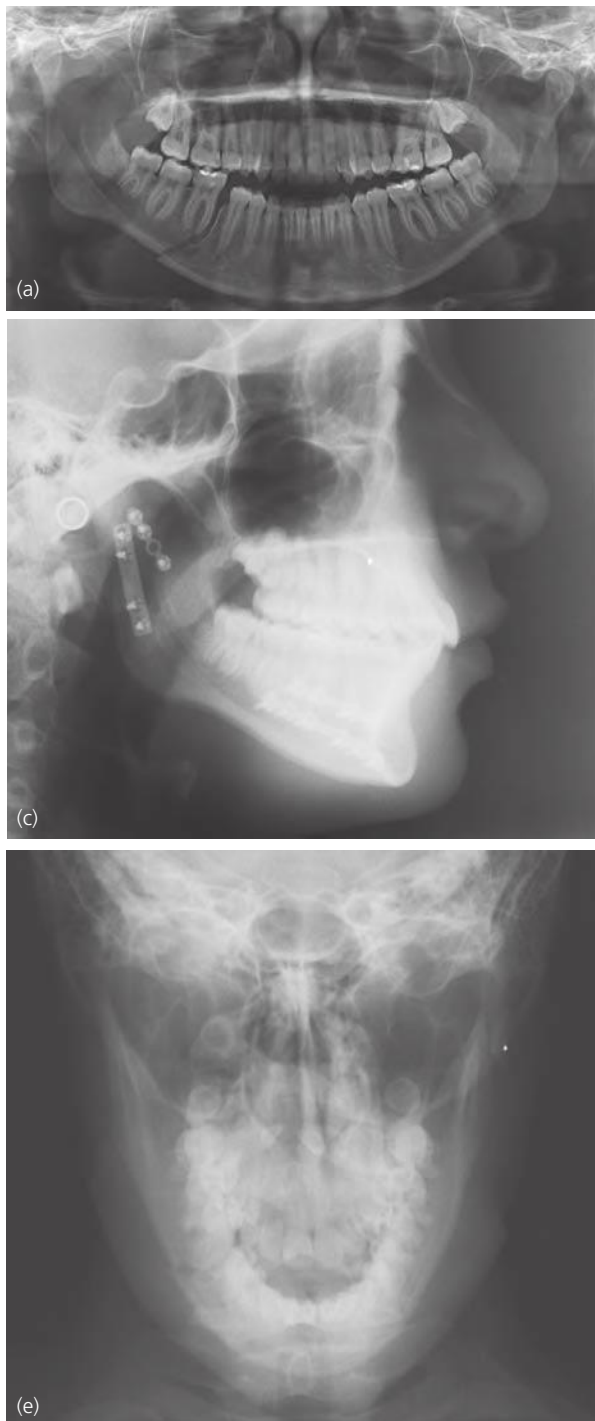


Fig. 43.1 Townes' and panoramic radiographs preoperatively (a, e) and postoperatively (b, c, d) demonstrate a displaced condylar fracture with shortening of the ascending ramus before and after endoscope-assisted transoral anatomic reduction and osteosynthesis. The surgical correction of the shortening of the ascending ramus is demonstrated (b, c, d). Maxillomandibular fixation was used intraoperatively for the osteosynthesis of an additional mandibular fracture.

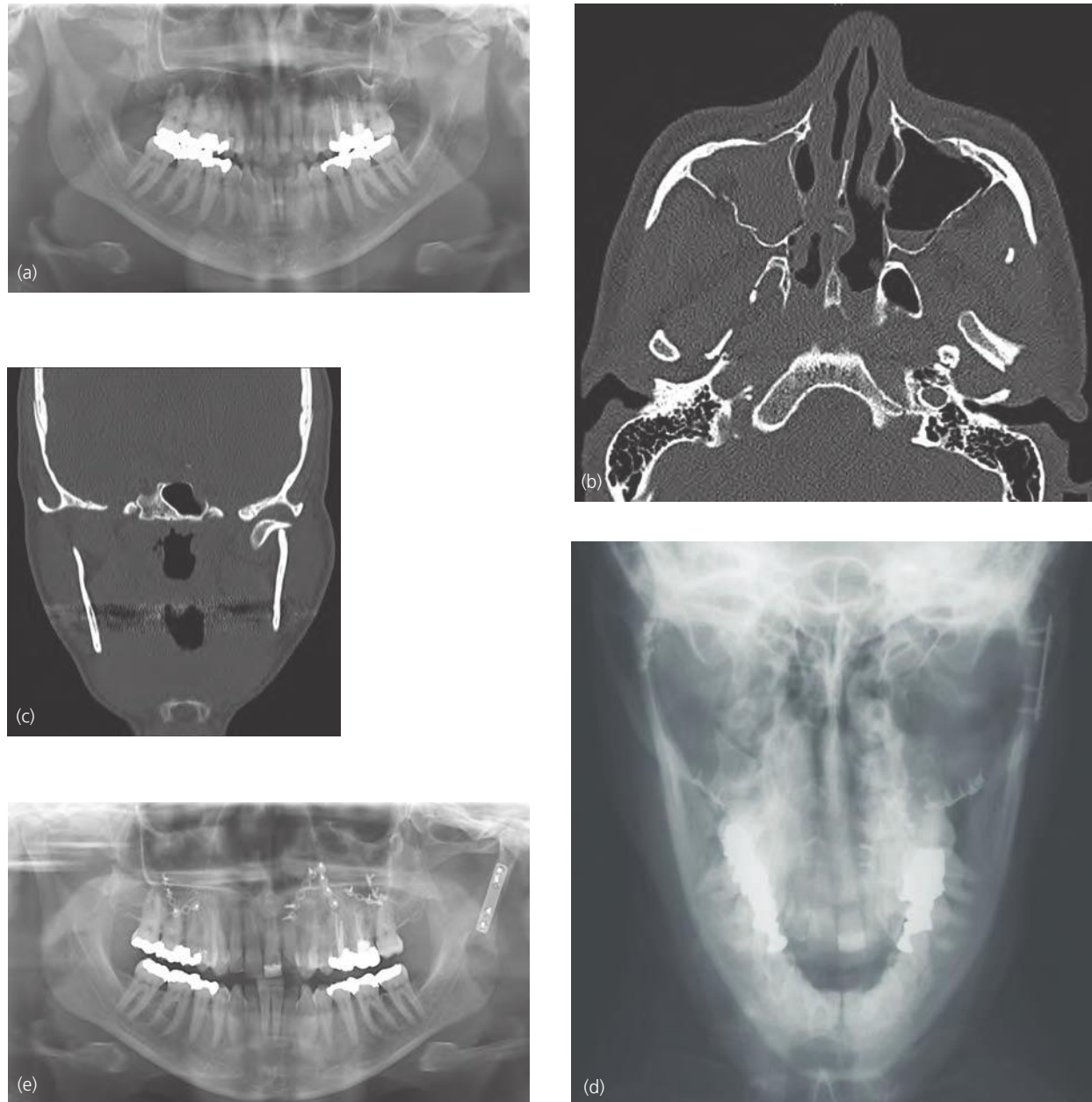


Fig. 43.2 (a–c) Preoperative panoramic radiograph, and an axial and coronal view of a CT scan demonstrate a 90° dislocated fracture of the condylar neck in a patient who also sustained a displaced bilateral midfacial fracture. (d, e) Following the open reduction and fixation (ORIF) of the right zygomatic complex, a Le Fort I fracture, and a left alveolar process fracture, the displaced condylar neck fracture was reduced and fixed by transoral endoscope-assisted approach.

fracture reduction, intraoperative imaging is only indicated in selected cases.

Fig. 43.4 demonstrates the endoscope-assisted reduction and fixation of condylar fractures, performed via limited transoral incision using 30° and 45° angled endoscopes and a Xenon light source (Karl Storz®, Tuttlingen, Germany).

Surgical technique

The transoral approach for endoscope-assisted reduction and osteosynthesis of displaced and dislocated

condylar mandible fractures should be performed using an angulated drill and screwdriver.

Prior to the transoral incision, local anesthesia is infiltrated in the vicinity of the fracture and the area of the surgical approach to obtain a blood-free optical cavity. Bleeding into the optical cavity is usually minimal due to infiltration of local anesthesia. The limited incision is made along the anterior aspect of the ascending mandibular ramus, comparable to the cranial part of the incision for sagittal split osteotomy (Fig. 43.4d).¹⁷ The mucoperiosteum is then elevated subperiosteally and the ascending mandibular ramus is exposed. The masseter muscle is not dissected by

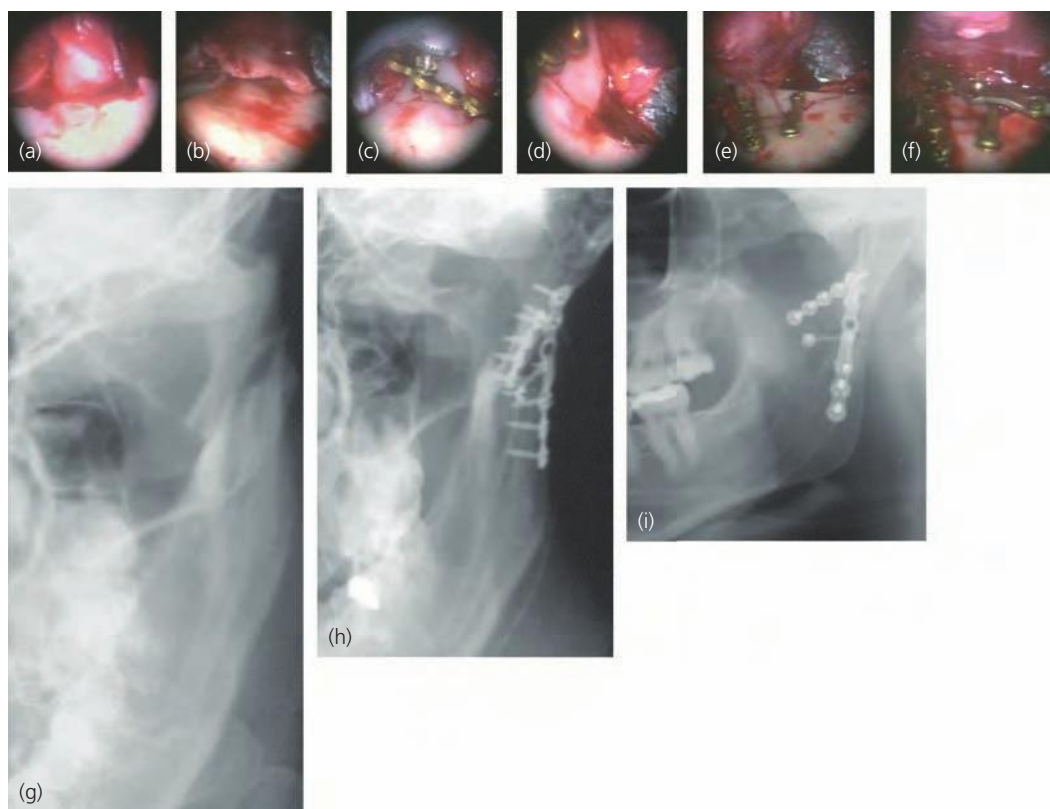


Fig. 43.3 (a) The endoscopic view demonstrates a medially displaced fracture of the mandibular condylar process. (b) By forward and downward rotation of the mandible, the TMJ area is distracted and the fragment is reduced in a lateral displacement before anatomic reduction and fixation. (c) Transoral osteosynthesis is performed using an angulated drill and screwdriver. (d) Note a butterfly fragment is missing at the posterior border. (e, f) The butterfly fragment is fixed with an adaptation plate before a more rigid plate is placed at the posterior aspect of the ascending mandibular ramus. Fracture reduction including the realignment of butterfly fragment is recommended to allow for a load-sharing situation, to reduce the stress of functional loading on the osteosynthesis. Townes' radiographs preoperatively (g), postoperatively (h), and a panoramic radiograph (i) demonstrate the displaced fracture before and after the minimally invasive reduction and fixation.

the subperiosteal approach to avoid bleeding in the optical cavity.^{13,16} Good visibility is obtained during the surgical approach, using a head light with a xenon light source (e.g. Karl Storz®, Tuttlingen, Germany).

Localization of the fracture site and of the displaced fragment is performed using the endoscope (Fig. 43.5d). Muscle relaxation is mandatory to allow reduction of the fracture. To distract the TMJ region, pressure is applied to the posterior teeth of the mandible and the mandible is rotated forward. Maxillomandibular fixation (MMF) is not recommended during the fracture reduction. Due to the manipulation of the mandible to allow for fracture reduction, additional mandibular fractures should be fixed first to prevent further fracture dislocation and injury of the inferior alveolar nerve (Figs 43.1, 43.4, 43.5, and 43.6). MMF is only performed intraoperatively for the fixation of additional mandibular fractures and released before the open reduction and internal fixation (ORIF) of the condyle fracture is performed. In medially displaced fractures, the fragment is reduced in a lateral position to allow miniplate fixation (Fig. 43.3a, b). This procedure may be difficult and can be facilitated in selected cases by inserting an elevator subperiosteally at the lingual aspect of the ascending ramus. The instrument has to be inserted

cranial to the mandibular foramen to push the fragment into a lateral position. Subperiosteal insertion of the elevator is necessary to avoid damage to the maxillary artery.

The first plate is then placed in the cranial aspect of the fracture with visual control of the incisura semilunaris, using an adaptation plate of the 1.5 or 2.0 compact system (Synthes) usually using screws 4 mm in length (Figs 43.3c and 43.5d). Insertion of the first screw together with the plate is performed using an angulated screwdriver with a plate screw holding device (Figs 43.3c and 43.5f).^{13,17} No additional plate-holding instrument is used.

After the first screw is anchored cranial to the fracture line, the fragment is pulled anteriorly and caudally using a nerve hook or a Mitchell's trimmer inserted in an empty inferior hole of the plate to reduce the fracture (Figs 43.3d and 43.5e). When the fracture reduction proves to be satisfactory by endoscopic control, the plate is fixed with a second screw close to the fracture (Figs 43.3d and 43.5e). Care should be taken that no soft tissue is entrapped in the fracture. Then the quality of the fracture reduction is double checked endoscopically along the fracture line and at the posterior aspect of the ascending ramus (Figs 43.3d, e; 43.4c; 43.5e, g; 43.6d). After the fracture

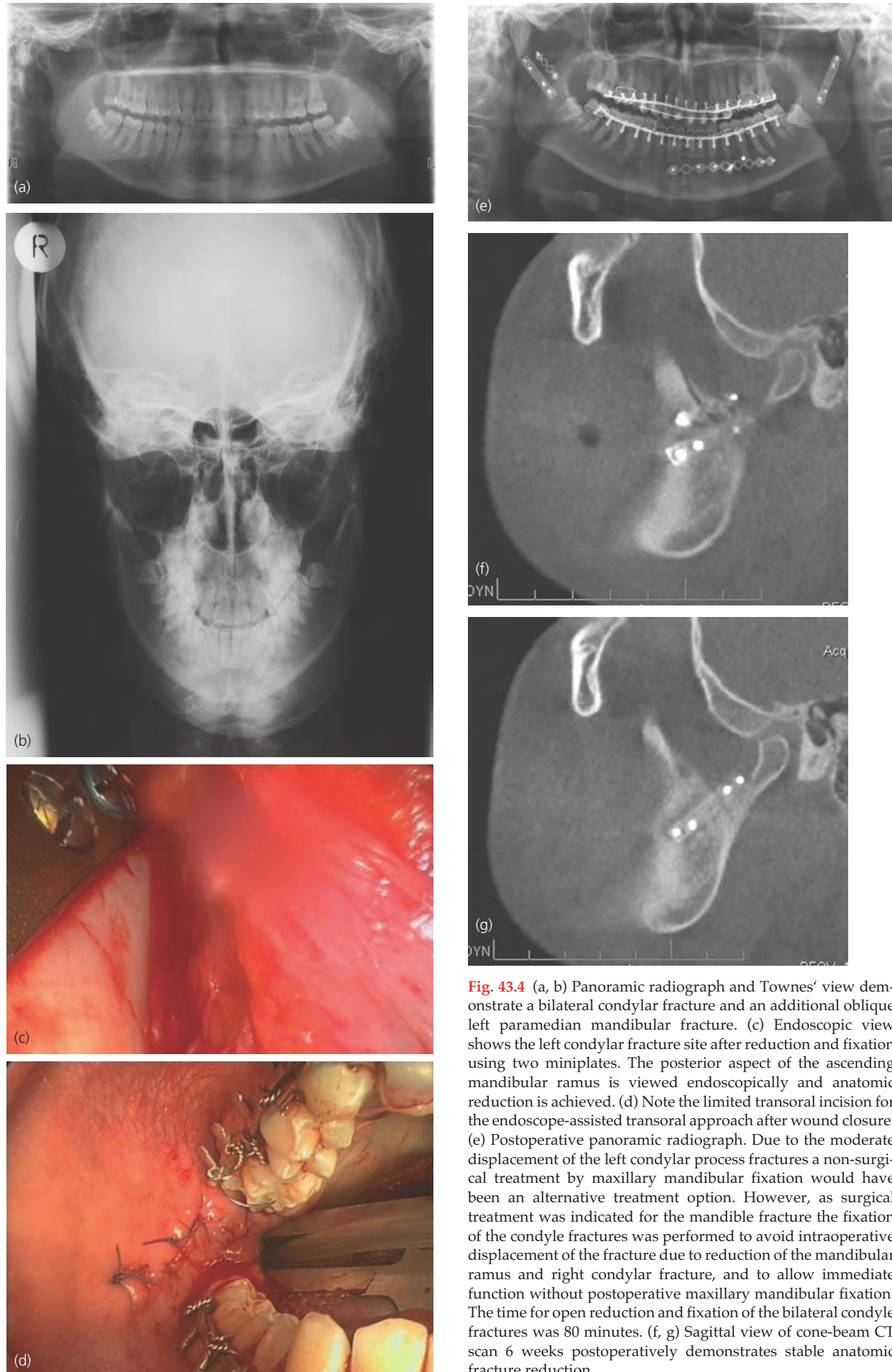


Fig. 43.4 (a, b) Panoramic radiograph and Townes' view demonstrate a bilateral condylar fracture and an additional oblique left paramedian mandibular fracture. (c) Endoscopic view shows the left condylar fracture site after reduction and fixation using two miniplates. The posterior aspect of the ascending mandibular ramus is viewed endoscopically and anatomic reduction is achieved. (d) Note the limited transoral incision for the endoscope-assisted transoral approach after wound closure. (e) Postoperative panoramic radiograph. Due to the moderate displacement of the left condylar process fractures a non-surgical treatment by maxillary-mandibular fixation would have been an alternative treatment option. However, as surgical treatment was indicated for the mandible fracture the fixation of the condyle fractures was performed to avoid intraoperative displacement of the fracture due to reduction of the mandibular ramus and right condylar fracture, and to allow immediate function without postoperative maxillary-mandibular fixation. The time for open reduction and fixation of the bilateral condyle fractures was 80 minutes. (f, g) Sagittal view of cone-beam CT scan 6 weeks postoperatively demonstrates stable anatomic fracture reduction.

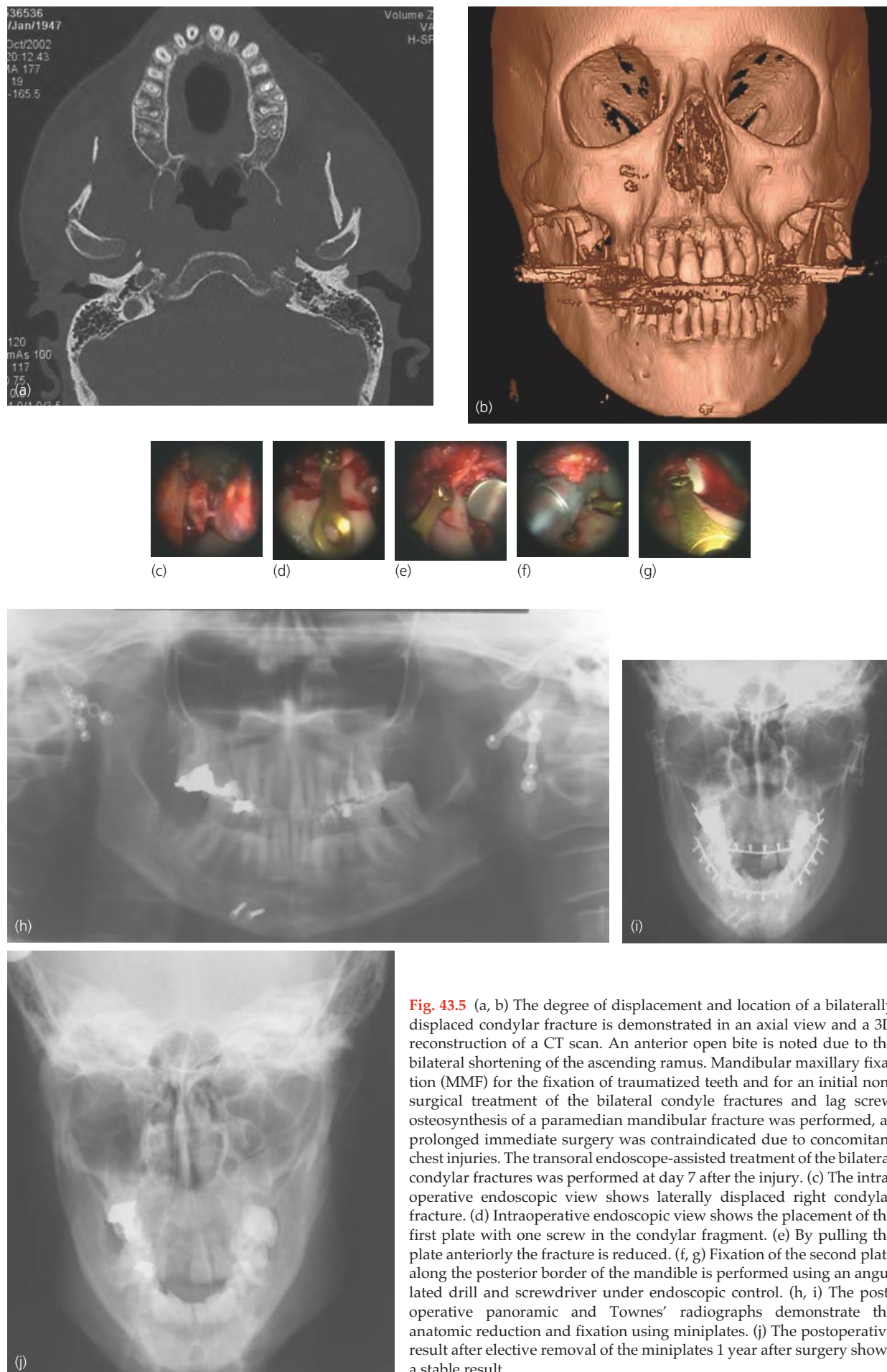


Fig. 43.5 (a, b) The degree of displacement and location of a bilaterally displaced condylar fracture is demonstrated in an axial view and a 3D reconstruction of a CT scan. An anterior open bite is noted due to the bilateral shortening of the ascending ramus. Mandibular maxillary fixation (MMF) for the fixation of traumatized teeth and for an initial non-surgical treatment of the bilateral condyle fractures and lag screw osteosynthesis of a paramedian mandibular fracture was performed, as prolonged immediate surgery was contraindicated due to concomitant chest injuries. The transoral endoscope-assisted treatment of the bilateral condylar fractures was performed at day 7 after the injury. (c) The intraoperative endoscopic view shows laterally displaced right condylar fracture. (d) Intraoperative endoscopic view shows the placement of the first plate with one screw in the condylar fragment. (e) By pulling the plate anteriorly the fracture is reduced. (f, g) Fixation of the second plate along the posterior border of the mandible is performed using an angulated drill and screwdriver under endoscopic control. (h, i) The postoperative panoramic and Townes' radiographs demonstrate the anatomic reduction and fixation using miniplates. (j) The postoperative result after elective removal of the miniplates 1 year after surgery shows a stable result.

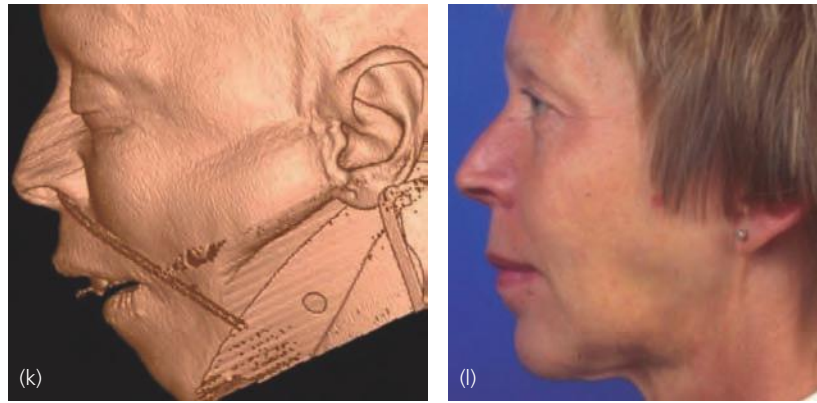


Fig. 43.5 (cont'd) (k) 3D reconstruction of the preoperative CT scan shows a retruding chin point due to backwards rotation of the mandible caused by bilateral shortening of the ascending ramus due to the pull of the temporalis and masseter muscles. (l) The chin projection has been corrected after bilateral anatomic fracture reduction.

has been simplified with the first adaptation plate, the second, more important plate can be precisely placed along the posterior aspect of the ascending ramus, where strong cortical bone allows for anchorage of the plate using screws 6 mm in length. The first screw of the posterior plate is placed in the fragment close to the fracture line. After anchorage of the posterior plate with one screw, the plate is pulled caudally and reduction at the posterior aspect is controlled using an angulated endoscope. If the reduction is not satisfactory, the anterior screw of the first plate may be loosened or removed. The reduction can be improved using a nerve hook or Mitchell's trimmer to pull the posterior plate antero-caudally. The second screw hole of the posterior plate can be drilled away from the fracture site to gain some compression to improve the fracture reduction. When the fracture reduction is satisfactory, osteosynthesis is completed with screws in a neutral position (Figs 43.3f; 43.4c; 43.5f, g; 43.6d).

Occlusion is checked at this stage. In general when precise fracture reduction is demonstrated endoscopically, preinjury occlusion is re-established. The two-plate technique allows for better fracture control and facilitates the precise placement of the most important plate along the posterior mandibular ramus (Figs 43.3e and 43.5f). Placement of the posterior plate as the first plate may limit the visibility of the posterior aspect of the mandibular ramus (Fig. 43.3f) and may cause an inferior anatomic reduction. A dental mirror may also be used to check fracture realignment after placement of the posterior plates (Fig. 43.6d). At the posterior border, preferably a 2.0 AO/ASIF non-compression miniplate (Synthes® Paoli, PA, USA, # 443.451) is used (Figs 43.1, 43.2, 43.4, and 43.6). This plate has been designed for the osteosynthesis of the midface and is less rigid than 2.0 mandible plates. The plate therefore adapts more easily to the bone surface than the rigid mandible plates when the screws are tightened. This proves to be beneficial for exact fracture reduction in the area of the mandibular condyle, as precise bending of the plates may be difficult. Dislocation at the fracture site when tightening the screws is avoided by using the less rigid plates.

In condylar neck fractures, two-plate osteosynthesis with the modified 2.0 mm zygoma AO-ASIF miniplate (Synthes® Paoli, PA, USA, # 443.451) along the posterior border of the ascending ramus is the authors' first choice of treatment.¹⁷ No plate loosening or plate fractures have yet been encountered when this plate has been used. Additional 1.5 or 2.0 AO-ASIF adaptation miniplates (Synthes®, Paoli, PA, USA) are placed in more than 70% of the authors' patients to counteract the demanding mechanical forces in the area of the mandibular condyle when immediate loading is allowed. In some cases the placement of a modified zygoma plate is not possible because of the limited bone surface (Fig. 43.5). The 2.0 compact (Synthes®, Paoli, PA, USA) four-hole plates with space have been used as the authors' second choice (Fig. 43.5). Good visualization for control of reduction and fixation can be achieved endoscopically. An angulated drill and screwdriver facilitate osteosynthesis; the insertion of the first screw together with the plate has been especially helpful (Figs 43.3, 43.5).^{13,16,17} Osteosynthesis was performed in all patients by a transoral approach only, without transfacial incisions for screw insertion. Angled elevators and reduction-manipulation forceps for condylar fractures, along with long retractors and periosteal elevators (Synthes® Paoli, PA, USA, AO Development Institute, Davos, Switzerland), have proved to be useful for the reduction of displaced fractures.¹⁵⁻¹⁷

Postoperative course and long-term results

Immediate postoperative movement is allowed in all patients and a soft diet is recommended for the first 10 days after surgery. Results show precise anatomic reduction and restoration of the vertical height of the ascending mandibular ramus are achieved, without malocclusion, in all patients. After 3 months and after 1 year of follow-up, mouth opening in all patients is generally more than 40 mm without deviation on opening and lateral excursion is not limited.¹⁵ Good TMJ function and an inconspicuous intraoral scar

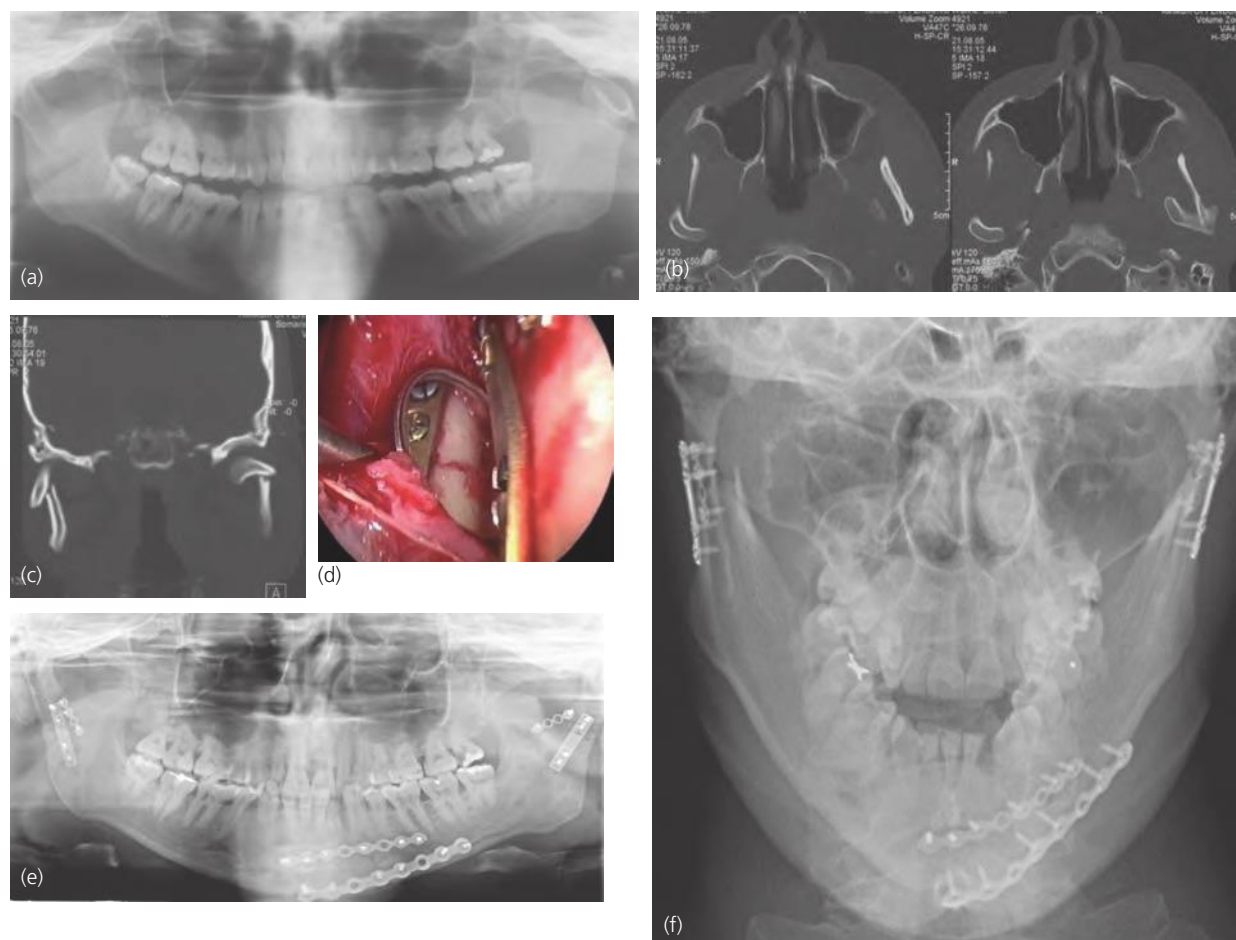


Fig. 43.6 (a–c) A severely dislocated mandibular ramus and bilateral condyle fracture is demonstrated in the preoperative imaging by panoramic radiograph and CT scan. Lateral displacement of the right subcondylar fracture and medial displacement of the left condylar fracture is noted in the axial and coronal CT scan view. (d) The endoscopic view shows the anatomic reduction of the left condylar fracture. Using a dental mirror posterior to the posterior border of the ascending ramus, the fracture site is visualized after ORIF. (e, f) Postoperative panoramic and Townes' radiographs demonstrate the postoperative result.

without signs of facial nerve damage are seen. There have been no signs of TMJ dysfunction or pain in the TMJ.¹⁵

Removal of osteosynthesis material is rarely performed. In selected cases when a limitation of the range of motion was noted 1 and 3 months after surgery, functional treatment using physiotherapy and an orthodontic appliance has been recommended.¹⁸ Good TMJ function with mouth opening of more than 40mm without deviation, and with preinjury occlusion and TMJ motion can be expected after 12 months.

Surgical vs non-surgical treatment of mandibular condyle fractures

Non-surgical treatment of condylar mandible fractures is most widely performed even in displaced and dislocated fractures of the condylar mandibular process. Open bite deformity or malocclusion may occur following non-surgical treatment due to shortening of the ascending ramus.^{8,16,17,19} Functional treatment may lead to satisfying results. However the patient must be compliant and a treatment period of 6–

12 months has to be taken into account to achieve satisfying functional results when physiotherapy and/or activator treatment is performed.¹⁸ To avoid the long treatment period and complications noted in patients with displaced fractures, open anatomic reduction may be beneficial and allow the patient to achieve early function.^{4,5,7–9} Even in moderately displaced condylar fractures, ORIF should be considered when additional mandibular fractures or bilateral condyle fractures with only moderate displacement on one site and displacement or dislocation on the other side may be present. Due to the manipulation for MMF and ORIF of the mandibular fracture, further displacement of the condyle fracture may occur intraoperatively. In such cases, intraoperative imaging by cone-beam CT scan is helpful to decide if ORIF is indicated in the moderately dislocated condyle region. To avoid displacement and to allow for early function, ORIF should be considered, even for moderately displaced condyle fractures, although non-surgical treatment may be possible to avoid MMF and to allow for early function (Fig. 43.4).

However, the indication for surgical reduction vs non-surgical treatment of displaced condylar fractures remains controversial in maxillofacial surgery

because of possible surgical complications, such as damage of the facial nerve and creation of visible scars when surgical treatment is performed.^{4,5-8}

The role of endoscope-assisted transoral techniques

To minimize the risk of the described complications, endoscope-assisted techniques using extraoral and transoral incisions have been described for various indications in the craniomaxillofacial area.²⁰⁻²⁴ The extraoral approach for condylar fractures was previously described as the preferred approach for medially displaced and comminuted condylar fractures and the transoral approach for laterally displaced fractures.¹³ Following the first promising results, the authors have also been using the transoral approach for medially displaced and dislocated condylar fractures routinely for the past decade. Condylar neck fractures have been treated by the same approach, when at least two screws could be anchored in the condylar fragment.^{15,16}

The use of the two-plate technique allows for precise fracture reduction under intraoperative endoscopic control. The precision of the fracture reduction in this mechanically demanding area with limited bone surface to realign is most important for achieving stable long-term results, without screw loosening and plate fractures. When the reduction is not adequate, fatigue fractures of the hardware and loosening of the screws may be encountered. Plate fractures following ORIF of condyle fractures have been reported with an incidence of up to 35%.²⁴ Before endoscope-assisted ORIF was performed in the authors' department about 10% of plate fractures were noted following ORIF in condyle fractures. Using the endoscope-assisted technique, less than 2% of failure of osteosynthesis has been noted in the authors' experience over the last 8 years.

Training

Intensive training in endoscope techniques is mandatory before the endoscope-assisted transoral approach for the treatment of condylar fractures can be performed. A steep learning curve with significant reduction of operating time has been noted when using the endoscope-assisted technique.^{13,15-17,25} After such training, the mean operating time for the minimally invasive transoral treatment at the authors' center today is 1 hour 05 minutes.

Summary

The endoscope-assisted transoral approach has developed in recent years and is today a reliable surgical approach for displaced and dislocated condylar

fractures; this is reflected in increasing acceptance of this technique.²⁶⁻³¹ In non-dislocated fractures and fractures of the condylar head, non-surgical treatment still remains the treatment of choice.³²

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Chapter 44

Soft Tissue Trauma

Bethany Serafin, Paul Koshgerian, and Richard H. Haug

A soft tissue injury is one of the most common problems treated in the emergency rooms and the facial region is an esthetically sensitive area with a complex variety of tissues. In this chapter, current principles of treatment for the different types of soft tissue injuries in various locations in the orofacial region are systematically presented. Factors of importance for successful wound healing are thoroughly discussed.

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Introduction

Although the number of lacerations treated in emergency departments has actually decreased over the past decade, traumatic laceration remains one of the most common problems treated in the emergency room, with 28% of those being to the face.¹ According to a 2004 study conducted by the National Center for Health Statistics, there were an estimated 2.5 million facial lacerations treated in United States (US) emergency rooms in that year alone.¹ Singer *et al.* compared demographics and characteristics of lacerations presenting to the emergency department over 10 years from 1992 to 2002.¹ Their findings of lacerations, not limited to the face, show that lacerations present slightly more frequently at weekends and during the spring and summer months. Furthermore, more lacerations present between noon and 10 pm with two thirds of the patients being male, one third of the

patients being under the age of 18, and three quarters being white.

Facial lacerations occur more commonly in children, with boys between the ages of 4 and 7 years, having a disproportionately larger number of lacerations. According to a study by Mack *et al.*, in the US, every 90 seconds a child 1 year of age or younger, is treated in the emergency department for non-fatal, accidental injury.² Contusions and abrasions top the list of injuries treated; and those contusions and abrasions, in addition to laceration, hematoma, puncture and foreign body wounds, occur most frequently to the head or neck.² Soft tissue injuries outnumber fractures in children who sustain facial trauma due to the relative elasticity of bone at a young age. A study by Gassner *et al.* reviewed 3385 cases of pediatric cranio-maxillofacial trauma sustained over a 10-year period.³ Causes of these soft tissue injuries were most frequently traffic accidents followed by sports and vio-

lence, followed by play.³ As in adult soft tissue trauma, the peak of all types of injuries in children occurs during the spring and summer months. Children represent a special population for soft tissue injuries due to their quick healing and tendencies toward hypertrophic scar and keloid formation.

Maxillofacial trauma results in a multitude of consequences for the patient, as well as their family members and society as a whole. While data specific to soft tissue injury are lacking, studies of maxillofacial injury in general have shown that trauma has significant and often unrecognized psychosocial and economic impact.

There exists in maxillofacial injury the potential for great psychological disability. Post-traumatic psychopathology (PTP) is a spectrum of disease, ranging from short-term anxiety to post-traumatic stress disorder at the extreme end. While few studies have examined the extent of psychopathology following facial injury, research has shown that psychological morbidity is common, it is persistent throughout the follow-up period, it can become chronic, and the clinical severity of the injury does not correlate well with the development of psychopathology.^{4,5} When maxillofacial trauma leads to post-traumatic psychological disturbance, a vicious cycle has then been created because the resulting psychopathology also has a significant impact on physical recovery from injury. There is a biological basis for impaired recovery and wound healing through prolonged inflammatory response when PTP complicates recovery. Also, due to its impact on compliance with treatment and follow-up, PTP contributes to poor recovery.^{4,6}

The financial impact of injury is enormous. In addition to the immediate and long-term cost of care, injury also causes a tremendous loss in productivity, a cost that society ultimately bears. The productivity loss due to injury includes lost wages, lost benefits, loss of marketable goods and services, as well as loss of the ability to perform daily responsibilities at home. The dollar value of productivity loss for injuries that occurred in the US in 2000 is an estimated \$326 billion which far exceeds that of the \$80.2 billion in cost for the medical treatment related to these injuries.⁷

General principles of management

Assessment

During examination of any facial injury, several features and characteristics must be noted and made part of the medical record. The mechanism of injury and time of occurrence, as well as witnessed accounts should be recorded. Mechanism of injury will provide information about any potential foreign bodies that need to be removed or contaminants that may affect wound healing. The circumstances of the injury should also be elicited within the history. Knowing

an injury is work-related, an act of domestic violence, or an assault is important for the medico-legal aspect of the patient's care. Any treatment of the wound already rendered prior to formal evaluation should be ascertained.

Knowing the tetanus immunization status of the patient is important. Past medical history including a list of medications and allergies should be reviewed and vital signs should be measured. It is well known that certain comorbidities and medications adversely affect wound healing and may necessitate a change in the normal course of treatment.

A primary survey of the patient will reveal the patient's airway status, neurologic function, and any other serious injury to the chest, abdomen or extremities. Once life-threatening injuries have been ruled out, an assessment of the wounds in question can be performed. Physical examination includes inspection of the area noting the location, size, and shape of the wound. Limitation of function and involvement of nerves, vessels, muscles, or other vital structures should also be noted. Presence of foreign bodies, contamination, associated fractures or hematomas, non-vital tissue or, in the case of an old injury, presence of infection should be determined. Noting all of these characteristics upon examination will allow the practitioner to develop a thorough, well devised plan for repair.

Timing of repair

While not every traumatic wound presents with a critical load of bacteria necessary to cause infection, every traumatic wound should be considered contaminated. This does not mean, however, that traumatic wounds of the face cannot be closed primarily, even after considerable time delay.

With regard to infection rate, timing of repair is critical. Wounds that are cleaned and closed in a timely manner have less chance of developing wound infection. The role of the critical load of bacteria (100 000 bacteria per gram of tissue) is discussed elsewhere in this chapter; however, this number has been established as a threshold for wound infection. Wounds with inoculums above this number have a 95% chance of becoming infected.⁸ Wounds with less than 10^5 bacteria per gram of tissue will heal uneventfully. In a study of wounds presented to the emergency room, Robson *et al.* observed that wounds less than 2.2 hours old had 10^2 bacteria per gram of tissue – well below the critical number for infection.⁸ Wounds 3 hours old had 10^2 – 10^6 bacteria per gram of tissue and wounds more than 5 hours old consistently held more than 10^6 bacteria per gram of tissue – well above the threshold for infection.

Ideally, wounds of the head and neck are repaired in order for healing to occur by primary intention. This type of healing takes place quickly and with minimal scar formation. Some arbitrary guidelines have been developed and the consensus is that uncompli-

cated clean lacerations of the face can be closed primarily up to 24 hours after injury.⁹ Trott recommends that if the wound, irrespective of time after injury, can be debrided and cleaned such that the wound appears fresh and bleeds slightly, it can be closed primarily.¹⁰

A more prolonged healing process, healing by secondary intention, occurs when a wound is left open and allowed to heal from the inside out by way of a granulation tissue base with subsequent epithelialization and contraction. It is rare to have this type of healing from traumatic wounds on the face.

Delayed primary closure is a method of management of dirty or infected traumatic wounds or wounds that have gone unrepaired for a considerable amount of time. These wounds are converted to fresh wounds through debridement and removal of tissue edges and then covered loosely for several days. After this short period of time the wound can once again be debrided and then closed if no infection is evident.

Antibiotic and tetanus prophylaxis

Non-bacterial contributors to infection

There are several non-bacterial contributors to wound infection. Torn and crushed tissue is likely in traumatic injury to the maxillofacial region. Dead, devitalized tissue within a wound contributes to colonization and subsequent infection.

The impairment of vascular supply to tissue is another contributor to wound infection. While the skin of the face receives a disproportionately large amount of blood, slow healing of a wound in this area can still result when conditions that contribute to poor circulation are present. Systemic conditions such as diabetes or arteriosclerosis or local factors such as excessive wound tension or thrombosis cause impairment of local circulation and thus increased risk of infection.

Location of the wound is a factor in wound infection. Injuries of the face are less likely than other areas of the body to become infected; however, the likelihood is increased if that wound involves the oral cavity.

Foreign bodies such as metal, wood, glass, grass, dirt, etc. harbor organisms and contribute to wound infection. Deep sutures also act as foreign bodies and accumulations of blood and fluid within a wound can act to harbor organisms, delay healing, and contribute to infection.

Abnormalities in immune function, both local and systemic, compromise the healing of tissue injury. Leukocyte dysfunction and inflammatory response reduction have various causes, including immunodeficiency diseases, chronic steroid use, malnutrition, radiation therapy, and burns. Heightened immune response in the form of allergy can also add to the development of wound infection.

Bacterial contributors to infection

There is no convincing evidence that antibiotics are useful for preventing infections in simple lacerations.^{11–13} However, there are times when antibiotic administration should be considered and this coverage should be given as soon after injury as possible. Studies have shown a “golden period” for the effective use of antibiotics within about 3 hours after injury.^{8,14} Antibiotics are indicated in certain circumstances, some of which include: patients with lymphedema, patients who are prone to bacterial endocarditis, grossly contaminated wounds, ear lacerations, bite wounds, and in patients with systemic disease that compromises healing, such as diabetes or cirrhosis.

The acutely contaminated wound presents a challenge to wound management and healing. The goal is to decrease the bacterial inoculum to below the critical level. This can be accomplished by sharp debridement of necrotic and devitalized tissue. Intermittent high pressure irrigation with a pulsating jet lavage system can assist in the removal of debris and bacteria. For grossly contaminated wounds, the repairs usually occur in the operating room. Systemic antibiotics are of little value unless given within the 3-hour time window after injury. Unless given immediately upon presentation to the emergency department, it is unlikely that antibiotics will be administered within that “golden time” period. And when in doubt, the wound can be left open, packed, and delayed closure planned.

Tetanus prophylaxis

Tetanus is a disease that occurs when oxygen-poor tissue, such as that of a wound, gets inoculated with *Clostridium tetani* spores which then germinate to bacilli and produce a potent neurotoxin. Tetanus presents as generalized muscle contractions that can involve the respiratory system and ultimately lead to death. The reported cases of tetanus have declined considerably since the use of widespread vaccination; however, during the time between 1990 and 2001, 534 cases were reported in the US, and tetanus has a high morbidity.¹⁵ Older adults have a higher incidence of tetanus, possibly due to the failure of initial immunization.¹⁶

During history taking, the tetanus immune status of the patient should be established. It should be determined whether or not the patient has had an initial tetanus immunization and, if so, when was the date of the last tetanus toxoid booster. The Advisory Committee on Immunization Practices (ACIP) is a committee of the Center for Disease Control and gives the guidelines for the administration of tetanus prophylaxis (Table 44.1). Recently, the preparation of tetanus toxoid, which was combined with diphtheria toxoid (Td), has incorporated a pertussis vaccine and this formulation is designated Tdap. In the USA, one Tdap product, ADACEL® (sanofi pasteur, Toronto,

Table 44.1 Guide to tetanus prophylaxis in routine wound management. From: Broder KR, Cortese MM, Iskander JK, *et al.* Preventing Tetanus, Diphtheria, and Pertussis Among Adolescents: Use of Tetanus Toxoid, Reduced Diphtheria Toxoid and Acellular Pertussis Vaccines. Recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR February 23, 2006/55 (Early Release), 1–34; and Kretsinger KK, Broder KR, Cortese MM, *et al.* Preventing Tetanus, Diphtheria, and Pertussis Among Adults: Use of Tetanus Toxoid, Reduced Diphtheria Toxoid and Acellular Pertussis Vaccines. Recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR December 15, 2006/55(RR17), 1–33.

History of adsorbed tetanus toxoid (doses)	Clean, minor wound		All other wounds ^a	
	Tdap, Td or DTaP ^b	TIG	Tdap, Td or DTaP ^b	TIG
Unknown or <3	Yes	No	Yes	Yes
≥3	No ^c	No	No ^d	No

^a Such as, but not limited to, wounds contaminated with dirt, feces, soil, and saliva; puncture wounds; avulsions; and wounds resulting from missiles, crushing, burns, and frostbite.

^b Tdap is preferred to Td for adolescents and adults aged 11–64 years who have never received Tdap. Td is preferred to TT for adults who received Tdap previously, or when Tdap is not available. DTaP is indicated for children <7 years old.

^c Yes, if >10 years since the last tetanus toxoid-containing vaccine dose.

^d Yes, if >5 years since the last tetanus toxoid-containing vaccine dose.

Tdap, tetanus toxoid combined with diphtheria and a pertussis vaccine; Td, diphtheria toxoid; TT, tetanus toxoid; TIG, tetanus immune globulin; DTaP, diphtheria, tetanus and pertussis vaccine.

Ontario, Canada) is licensed for use in persons aged 11–64 years as a single dose active booster vaccination against tetanus, diphtheria, and pertussis. Another Tdap product, BOOSTRIX[®] (GlaxoSmithKline, Rixensart, Belgium), is licensed for use in adolescents but not for use among persons aged ≥19 years.¹⁵ ACIP recommends that adults receive a tetanus toxoid-containing booster vaccine every 10 years, or Tdap and tetanus immune globulin (TIG) as part of wound management, to maintain protective levels of tetanus antitoxin. For adults who have already been vaccinated with Tdap, Td preparation should be used if a tetanus toxoid-containing vaccine is indicated for wound care. Tdap vaccination should be deferred in the case of acute illness until that illness resolves or in cases of unstable neurologic conditions such as cerebrovascular accident (CVA) or encephalopathies.

Tissue handling

Several principles apply to tissue handling that can affect the healing process. Gentle handling of all tissue and proper use of retractors, tissue forceps, and sutures promote faster healing. Sutures tied too tightly around the edges of the wound increase the risk of infection.¹⁷ The presence of dirt, glass, or other foreign material increases the risk of infection. Removal of all foreign bodies and non-vital tissue is necessary

prior to closure. Necrotic tissue should be removed; however, skin and dermis should be conserved as much as possible. Often, dusky skin which initially looks devitalized may go on to heal. Later revision will benefit by having as much skin as possible preserved at the time of initial repair. Adequate hemostasis within the wound not only makes exploration and assessment easier, but also decreases the rate of wound infection and delayed healing. Single-bleeding vessels should be ligated, but blind clamping within the wound may lead to excessive tissue damage and prolonged healing time. Persistent oozing may be treated with direct gauze pressure held firmly for 5 minutes. Epinephrine-soaked gauze or other hemostatic agents such as synthetic methylcellulose foam or sheets may also help to control bleeding. The wound should be kept moist throughout repair. Long cases of repair without wound irrigation may cause desiccation of the tissue leading to prolonged healing time.

Another principle in wound handling is elimination of wound dead space prior to closure. If layered closure does not eliminate all the potential for air or fluid accumulation between the tissue layers in clean, non-contaminated wounds, drain placement or pressure dressing should be considered. In contaminated wounds, deep closure increases the risk of infection.¹⁷ Finally, when closing the wound, tension on the skin should be eliminated. Deep subcutaneous sutures and slight undermining of wound edges help to accomplish this. When closing tension is high, the likelihood of ischemia, tissue necrosis, and dehiscence is high as well.

Instrumentation

Absorbable sutures

As discussed earlier in this chapter, facial lacerations in the pediatric population are frequent. Due to the difficulty in removing sutures in a young patient and need for an additional visit to the provider, absorbable sutures have become an alternative to the nylon sutures traditionally used in repair of facial lacerations. Cosmetic outcomes, complication rates, and parental satisfaction have been measured in studies of children who have sustained facial lacerations and there was no significant difference between subjects who received standard wound closure with non-absorbable sutures vs absorbable sutures.^{18–21}

Tissue adhesive

Cyanoacrylate-based tissue adhesives have been used for laceration repair outside the US for many years; however, these have only been recently approved for use within the US, with 2-octyl cyanoacrylate (Dermabond, Ethicon Inc., Somerville, NJ) leading the way. Tissue adhesives are quick, easy to use, and painless because they do not require the use of local

anesthesia, and they also eliminate the need for suture removal. The contraindications for their use outnumber the indications; however, for repair of simple traumatic lacerations, tissue adhesive may be a viable option.

Proper case selection is the key to successful outcome. It is important to avoid getting adhesive between the wound edges in order to avoid wound tattooing from the blue pigments within the adhesive. This is most likely to occur in cases where the laceration is stellate or avulsive. A rare complication of the use of Dermabond reported in the literature is a blue discoloration of a scar after the use of Dermabond to repair a stellate laceration where it is presumed that the wounds were inadequately approximated.²²

The rates of wound infection and dehiscence as well as cosmetic score for closure with tissue adhesive are comparable to those of suture closure.²³ While follow-up costs (physician fee and lost wages) are minimized with the use of tissue adhesive, the cost-effectiveness of the use of tissue adhesive compared to traditional suture closure methods is controversial.²³⁻²⁵

Staples

Skin stapling devices have primarily been used in closing surgically created incisions; however, their use in closing traumatic lacerations should not be overlooked. With the right indications, staple closure can be a time-saving and effective method of soft tissue trauma management. Staple closure is ideal for linear lacerations of the extremities, trunk, and scalp. Studies demonstrate that the cosmetic result is as good with staple closures as with traditional suture closure techniques. In a cost analysis study, suturing of a traumatic laceration was shown to be approximately three times more costly than stapling, with the cost differential increasing as the length of the laceration increased.²⁶ Other advantages of staple closure include rapidity, safety to the operator due to less risk of needle-stick injury, good cosmesis, resistance to infection, less wound inflammatory response, and they are well tolerated by patients. One disadvantage is that two people are required for an ideal staple closure technique. Other disadvantages to the use of staples include the need for a special removal device as well as discomfort upon removal.

Staple closure is indicated in wounds of the scalp where hemostasis has been achieved and the galea is not torn. It is also indicated in linear, sharp lacerations of the extremities or trunk but generally not used on the face. Staple closure can also aid in temporary closure of superficial lacerations or for rapid hemostasis when other life-threatening conditions take priority. Metal staples can produce artifact on computed tomography (CT) scans and can move during magnetic resonance images so their use is contraindicated if the need for those studies is anticipated.

Postoperative care

Postoperative wound care should include verbal and written instructions and information about the dressing management, cleansing of the wound, activity limitations, pain management, expectations of the appearance of the wound during the healing course, and follow-up appointment. Patients should be instructed to keep their dressing intact for the first day. On the second postoperative day, the dressing can be removed and the wound cleaned and dried. In a study of 200 head and neck lacerations and surgical incisions, investigators concluded that allowing patients to wash their wounds within as little as 8 hours after closure had no effect on healing or rate of infection.²⁷

Patients should be informed of the activity limitations that are necessary to prevent wound disruption. Pain management strategies should be discussed and patients should be given a realistic expectation of the amount of discomfort they may encounter, as well as the amount of relief they can expect from medication and other pain relief methods. The patient should understand the changes they can expect in the appearance of the wound, and along with the surgeon, develop realistic goals and expectations for the final appearance of the scar. Management of the scar and timing of possible revision should be discussed, with an endpoint clearly established. Written instructions and follow-up visit appointments should be given to the patient upon discharge.

Dressing management

Wound dressing choices depend on the type and location of the wound, as well as the operator preference. Repaired lacerations of the face can generally be left uncovered. Due to the high vascularity of facial skin, these wounds usually heal well and have a low risk of infection. Use of petrolatum-based topical antibiotic over sutured traumatic lacerations has been shown to reduce infection rate²⁸ and encourage epithelialization.²⁹ The use of white petrolatum ointment may be just as effective at preventing crusting around the wound encouraging epithelialization to occur rapidly and allowing easy removal of sutures with less risk of contact sensitivity than antibacterial ointments.³⁰ A thin layer of ointment over the sutured repair will prevent the unbandaged wound from desiccation and an ultimate delay in epithelialization. Occasionally, wounds in the maxillofacial region will require dressing for protection from contamination or for immobilization of the wound. Wound dressings for specific anatomic locations are discussed in the respective wound section.

Suture and staple removal

When non-absorbable sutures or staples are placed for wound repair, removal is necessary as part of the

wound aftercare. In the maxillofacial region, the timing for removal differs from that of the rest of the body and is in general much earlier. For the face or ear, sutures should be removed in 4–6 days, for the scalp 6–8 days, and for the neck 5–7 days. Sutures in the maxillofacial region should be removed as early as possible; however, at this time, the skin has only regained about 5–10% of its tensile strength. Thus, any stress on the skin after suture removal will lead to dehiscence. It is necessary to continue to support the skin while healing continues and this can be accomplished with adhesive tapes, with or without the use of adhesives. The tapes should be allowed to slowly peel off over several days following placement, as the patient repeatedly washes the area.

Suture marks are complications of laceration repair and their development depends on several factors. The tension with which the suture knot was tied can be a cause. Excessive tension on the tissue leads to small areas of local ischemia and the appearance of unsightly suture marks after healing has taken place. The type of skin repaired is also a contributing factor. The skin of the eyelids is unlikely to reveal suture marks; however, other areas such as the lower third of the nose and the nasolabial areas are vulnerable to marks. Skin variation among patients also plays a role in the formation of suture marks. People who form keloids have a greater risk of having suture marks after laceration repair. The development of a suture abscess also lends itself to the development of suture mark formation. Braided sutures and silk are more likely to invoke an inflammatory reaction at the site of the suture and cause stitch abscesses and therefore more likely than nylon or staples to leave marks. Finally, the duration of the suture or staple left in place determines the formation of suture marks. Staples are kept in place the same period of time as sutures would be for that particular anatomic location. After 14 days in place, the suture track has become epithelialized and marks will be inevitable after suture removal. If the suture is removed before 7 days, no suture marks will be evident. The period between 7 and 14 days is a gray zone for the retention of suture marks that persist once the sutures or staples are removed.

Healing adjuncts

Vacuum-assisted closure

While negative pressure dressings have been used by surgeons for promotion of healing of chronic extremity wounds, infected sternotomy wounds, open abdominal wounds, and decubitus ulcers, little use in the maxillofacial region has been described. Vacuum-assisted closure (VAC) devices (Kinetic Concepts Inc., San Antonio, TX) combine occlusive dressings with negative pressure to improve blood circulation in wounds and thus speed healing. Studies to determine mode of action for this process have shown that

negative pressure applied to wounds increases capillary blood flow velocity, increases the caliber of capillaries, stimulates endothelial proliferation and angiogenesis, and restores continuity of the basement membrane by narrowing endothelial spaces.^{31,32} The restored basement membrane decreases the permeability of blood vessels, thus decreasing wound edema. This altered wound physiology results in faster production of granulation tissue and allows for less frequent dressing changes. Its effect on increased bacterial clearance of the wound is controversial.³³ VAC devices are also used as bolsters for split-thickness skin grafts to create uniform pressure over the graft and therefore enhance graft success.

Despite its paucity in the literature, VAC has utility in the head and neck region (Fig. 44.1). When using VAC systems, it is necessary to obtain an airtight seal over the wound. Maxillofacial wounds, with their extreme contours and hair-bearing skin, make the creation of an adequate seal difficult. Situations that preclude the use of VAC are presence of malignancy or eschar tissue in a wound, presence of exposed dura, and presence of a fistula to an organ or cavity. Its use in the presence of osteomyelitis is debatable.

Leech therapy

A common occurrence in partially avulsive soft tissue injury is the disruption of the delicate vascular supply. Injury to veins with maintenance of arteries may result in an imbalance of arterial inflow and venous outflow causing blood stasis within the tissue. This congestion leads to hypoxia of the tissue, thus compromising its viability and setting the stage for an endpoint of necrosis. When surgical repair of the venous system is not possible, medicinal leeches (*Hirudo medicinalis*) may be used to relieve congestion.^{31,35}

Medicinal leeches have been used for centuries to salvage skin grafts and tissue flaps whose viability is threatened by the obstruction of venous outflow.^{34,35} In addition to actively drawing off blood for use as a bloodmeal, the leech injects an anticoagulant contained within its saliva, hirudin, which allows continued bleeding from the site even after the leech is removed.³⁶ Hyaluronidase and antihistamines are also released by the leech causing diffusion of the hirudin into the wound and causing vasodilatation to prolong bleeding at the site.³⁶

Complications of leech therapy include significant blood loss, allergic reaction to the leeches' salivary proteins, scarring, migration of the leech, and psychological distress of the patient.³⁵ Leech-associated infections are also a recognized complication of leech therapy. Leeches contain multiple bacterial flora within their guts, with *Aeromonas* being the predominant genus. Antibiotic prophylaxis with a quinolone or aminoglycoside is necessary throughout the course of leech therapy.³⁶ Care must be taken during site



Fig. 44.1 (a) A female involved in a motor vehicle accident presents with an infected neck wound 5 days after surgical intervention. After debridement, a wound VAC is used to create a granulation tissue bed prior to coverage of the wound with a split-thickness skin graft. (b, c) A polyurethane sponge is cut to the size and shape of the wound and secured into the wound with an occlusive dressing. (d, e) A small opening is created in the occlusive dressing and airtight tubing is connected to the vacuum pump. (f) Continuous negative pressure of 100 mmHg is applied to the sponge and maintained continuously. (g) The wound VAC sponges are changed every other day for a total of 8 days of therapy. (h) The result is a healthy bed of granulation tissue.

preparation to avoid alcohol-based skin cleaning products which may agitate the leech, causing it to regurgitate and potentially infect the site with its own bacterial flora.

Effective arterial perfusion is confirmed by tissue puncture with resultant brisk bleeding. If upon pinprick, the segment has little or no bleeding, the arterial supply, in addition to the venous supply, is compromised and this tissue will most likely not be salvageable. Once true venous congestion is diagnosed, the patient's skin should be cleaned with soap and rinsed thoroughly with water. The head of the leech is guided to the area of maximum congestion until attachment occurs (Fig. 44.2). Once a leech attaches, it will stay in place for about 30 minutes or until it becomes full and falls off. Adequate venous outflow can be produced by adjusting the number of leeches applied. Some type of barrier such as gauze or an Op-Site dressing should be used to prevent migration of the leech (Fig. 44.3). Once the leech detaches, it should be discarded as a biohazard after one use.³⁵ Local clots formed at the bite wounds should be gently removed. While the average 5 ml of bloodmeal removed during feeding may initially help to decongest the tissue, the real rationale behind the use of leeches is to create a puncture wound that bleeds for hours.^{35,36} The bite wound can ooze for up to 10 hours, losing up to 150 ml of blood. Leeches are generally applied to congested tissue for about 3–5 days until new vessel ingrowth develops to adequately sustain venous drainage. Postoperative care of the patient includes

wound inspection for signs of infection and daily hemoglobin checks.

Considerations for the extremes of age

Advanced age is a risk factor for detrimental wound healing. While the mechanism of age-related delay in wound healing is not entirely understood, it is multifactorial in nature and is the subject of a substantial body of research. With the rate of growth of the elderly population in the US exceeding any other segment of the population, it is clear that this group of patients will benefit from the growing interest in research of the physiologic effect of aging on wound healing and methods to promote healing in the aged.

Concomitant disease in the elderly is one factor contributing to delayed wound healing. Diabetes, cardiovascular disease, renal disease, malnutrition, and obesity are among the age-related diseases that contribute to ischemia in traumatized tissues, thus compromising healing. However, research also suggests that the aging repair process is affected at the cellular level.

Skin becomes weaker with age due to the loss of collagen and elastic fibers, and decreased moisture content. A decrease in macrophages results in decreased skin immunity and a decrease in melanocytes results in decreased protection from sunlight. By the eighth decade, epidermal turnover is decreased by 50%.³⁷ With a loss of subcutaneous fat, a loss of



Fig. 44.2 Leeches are applied to tissue segment with the head (or sucking portion) attached to the most congested tissue segment.



Fig. 44.3 (a) Partial ear avulsion results in a compromised venous outflow while arterial supply is maintained within the small pedicle. (b) The partially avulsed ear is repaired with primary closure and true venous congestion is diagnosed. (c) Leeches are placed at the area of maximum congestion for 5 days. (d) Barriers should be used to prevent migration. (e) The ear has good esthetics after 4 weeks.

elasticity, and a relative desiccation of the skin, the elderly are at greater risk of tearing and bruising.

Specific wounds

Abrasion

Abrasive injuries are among the most common injuries in many age groups. Although the general public often dismisses such injuries, abrasions may become serious and should be thoroughly evaluated.

Abrasions are typically characterized as superficial injuries that denude the surface epithelium; however, involvement of deeper cutaneous layers must be ruled out. The large surface areas that these types of injuries produce serve as massive portals of entry for microbes, much like burn injuries. Without the epithelial surface barrier intact, proper management is required to prevent poor outcomes such as infection, scarring, or contamination tattooing.

Abrasive wounds, generally produced by shearing contact with rough surfaces such as roadways or concrete, often present with the impaction of foreign bodies or other environmental relics. Abrasions require thorough lavage with irrigant along with careful inspection and removal of any solid remnants and necrotic epithelium. Remaining irritants may be the source of prolonged inflammation, infection, and eventual discoloration of the wound following heal-

ing. Abrasions are generally more painful than lacerations or puncture wounds and more aggressive forms of pain management should be considered.

Abrasive wounds should be dressed with antibiotic ointment and covered with sterile gauze if necessary to maintain wound moisture until re-epithelialization is complete and final resolution of the wound takes place. Some practitioners elect to discontinue antibiotic ointments after a 72-hour period, dressing the wound with petroleum jelly or Vaseline for the remainder of healing time. Abrasive wounds are commonplace and often go untreated, yet because of the possible deleterious effects of contamination, these superficial wounds require management.

Laceration

Some basic principles apply when considering closure of simple lacerations of the skin. It is important that the layers of the laceration be reapproximated to their appropriate counterpart. That is, dermis should be sutured to dermis. Lacerations should be closed in layers with resorbable sutures used for deep closure of the dermis to reduce tension on the skin edges. Minimizing wound tension prevents necrosis which will ultimately delay healing and cause a cosmetically unacceptable result. Skin should be closed with appropriately sized non-resorbable suture such as 5/0 and 6/0 nylon. Scars contract with time, so

wound-edge eversion is an important principle. Vertical and horizontal mattress suture techniques as well as introducing the suture needle at a 90° angle to the tissue during percutaneous closure help evert wound edges. A technique that slightly raises the wound edges above the skin plane will ultimately result in a cosmetically acceptable closure.

Stellate lacerations result from blunt trauma and are less likely to yield a cosmetic result even when meticulously closed. Often, the edges of the lacerated tissue are contused and beveled, making primary repair difficult. These wounds should not be excessively debrided, which may be the tendency due to the poor appearance of the edges. Judicious debridement should be employed so that tissue removal will not compromise a later revision or reconstructive effort.

Through-and-through lacerations involving the oral cavity are complex lacerations necessitating layered closure (Fig. 44.4). After thorough debridement and cleaning, repair begins with alignment of the vermillion border and white roll if involved. These landmarks can be tacked with nylon sutures which can be replaced after muscular and mucosal repairs. Next, the muscle is closed with deep 5/0 absorbable sutures followed by mucosal closure with 4/0 or 5/0 absorbable suture. The skin is then closed with 5/0 and 6/0 nylon interrupted sutures. Even after good approximation of the layers, mucosal scarring can occur which can limit function (Fig. 44.5). Scar exci-

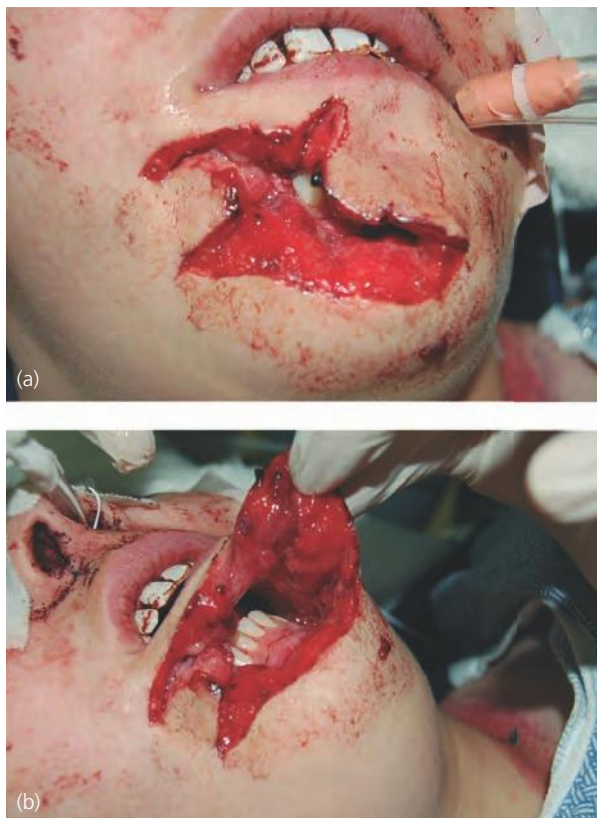


Fig. 44.4 (a, b) A young female patient sustained a complex through-and-through laceration of the chin necessitating a multilayered closure.

sion can help to release the fibrous banding. For skin lacerations, dermabrasion can help improve the look of a cosmetically unacceptable scar (Fig. 44.6).

Hematoma

Blunt trauma and contusion to the soft tissues of the head and neck result in hematoma formation, or a self-limiting bleeding within the subcutaneous tissue from minor vessel disruption. Usually, hematomas resorb spontaneously; however, some may become encapsulated and require evacuation. In early hematoma formation, aspiration with a 14 or 16 gauge needle will usually solve the problem. If the hematoma has matured such that the coagulum is in the jelly stage, incision with removal of the clots is necessary.



Fig. 44.5 Mucosal scarring can result in limitation of opening and lower lip movement.



Fig. 44.6 (a, b) The cutaneous scar can be improved with dermabrasion.

Pressure dressings, drain placement, and meticulous control of bleeding at the time of repair help to prevent hematoma formation.

One area where hematoma formation is particularly detrimental is the auricle. If left untreated, fibrous organization of the clot may take place resulting in “cauliflower ear”. Complete evacuation of the blood and placement of molded pressure dressings which compress the anterior and posterior perichondriums toward each other will minimize the likelihood of recurrence.

Avulsion

When small areas of tissue are lost due to trauma, undermining the adjacent skin will allow the margins to be directly approximated and closed. Larger defects, however, will require skin grafts or flaps for coverage. Direct approximation should be used with caution in areas proximal to certain anatomic features such as the eyelid, eyebrow, lip commissure, or nasal ala. These areas can become distorted if their surrounding tissue is undermined. Full-thickness defects greater than 2cm² are considered to be large defects and may need to be covered with grafts or flaps.³⁸

Mucosa

In general, lacerations, abrasions, and burns of the mucosa heal quickly with little intervention needed; however, if large or gaping, intraoral wounds benefit from approximation and suture closure. For closure of mucosal violations, 4/0 or 5/0 interrupted chromic gut suture will suffice. Failure to repair lacerated mucosa can result in scarring with functional compromise. Contaminated wounds or bite wounds should be debrided, cleaned, and closed loosely with sutures. Patients must maintain good oral hygiene throughout healing and continue with a soft diet until the mucosa is healed.

Tongue

The tongue often receives a portion of the injury secondary to trauma to the orofacial region due to its position and motility. Injury to the tongue ranges from non-operative superficial lacerations to complete avulsion. The tongue is a highly muscular and highly vascular tissue which generally lends itself to speedy recovery times and a high potential for repair. However, this highly vascularized tissue also lends itself to risk of hematoma formation or massive edema that can lead to upper airway obstruction within a short time period after injury or after repair.

Somewhat unique to the tongue, is the fact that many injuries may be self-inflicted. Characteristics of the laceration from a self-inflicted bite may also help in diagnosing a patient whose tongue laceration is secondary to an episodic neurologic disorder. Presentation of the injury may help distinguish

between syncope, stroke, and seizure disorder. Patients whose cause for loss of consciousness is psychogenic, typically do not incur tongue injury; however, when biting does occur in this situation, it is usually the tip that is injured.³⁹ Patients with blackouts due to hypoglycemia or transient ischemic attack are most likely to lose consciousness gradually and therefore uncommonly sustain a tongue laceration. Tongue biting, however, is highly specific for generalized tonic-clonic seizures and usually results in lateral tongue lacerations.⁴⁰

In some age groups, piercing and placement of a solid bar through the dorsal and ventral tongue are considered fashionable. Although the topic is still being debated in the literature in terms of traumatic potential to the dentition, it must be recognized that the site itself is a portal of entry for infection. In a study by Krause *et al.*, 15.4% of patients who had undergone tongue piercing had to seek medical treatment for a complication.⁴¹ Complications such as local infection, deep tongue abscess with subsequent Ludwig’s angina, hemorrhage leading to hypovolemia, and endocarditis have all been reported in the literature.

The largest and most common category of traumatic injury to the tongue falls under laceration. Statistically, children outnumber all other age groups and demographics in number of tongue lacerations. Although most lacerations of the tongue are considered superficial and require no treatment, the depth of the wound can vary and thus mandate different management modalities. These superficial lacerations heal quickly by secondary intention, and should simply be irrigated to remove any foreign bodies potentially involved. Literature suggests that suturing small non-gaping tongue lacerations (less than 1 cm in length) in young children does not affect the quality of the outcome; however, little has been written on the subject.⁴² Often it is the patient’s dentition that pierces the surface of the tongue secondary to strong impact from the inferior of the mandible or from a sympathetic reaction leading to activation of the muscles of mastication without regard for the position of the tongue. Lingual abscesses have been noted following biting of the tongue; however, they generally follow deeper penetrating wounds. Deeper injury into the muscle layers of the tongue requires careful examination of vascular integrity, as well as for the presence of foreign bodies. While tooth fragments are more often found within lip lacerations, there are reports of teeth becoming embedded into the body of the tongue during trauma.^{43,44}

These wounds should again be aggressively irrigated like all other traumatic lacerations, and approximation of the muscle layers with chromic suture should be achieved. Superficial sutures may then be placed to achieve healing by primary intention. Careful inspection of the vasculature is of primary importance to assess any injury to the lingual artery. Finally, prior to closure, all bleeding must be con-

trolled to minimize the chances of hematoma formation.

Hematoma formation in the tongue can be considered an emergency secondary to airway compromise and can lead to death. Hematomas that may result postoperatively from laceration closure most commonly occur in patients with generalized seizure disorders but also have been reported in anticoagulated patients postintubation. The patient can commonly appreciate any swelling around the base of the tongue and airway constriction will often create labored breathing, dyspnea, or wheezing. Ecchymosis is generally evident. Once the airway is secured, aggressive treatment to drain the hematoma and seal the source of the bleed is often initiated. Those areas near the base of the tongue can be especially problematic for the surgeon in terms of access and visualization. Electrocautery or ligature of the vessel is often employed to secure the bleed. Hirudotherapy may also provide benefit to treating venous congestion in the hematoma site; however compliance may be an issue (Fig. 44.7). The literature indicates that cases of macroglossia extending beyond the dentition or alveolar ridges may be managed with high success with leech therapy.⁴⁵ Appreciable resolution may be noticed within the first 6–8 hours and complete resolution often within 24–36 hours. Other treatments of traumatic macroglossia include positive pressure, steroid therapy, and manual reduction of the tongue back into the oral cavity; however each delivers questionable results.

Tongue avulsions or complete amputation are not rare in more serious traumatic injuries. Control of bleeding and placement of sutures through both the muscular and superficial layers may successfully treat some partial avulsions when vasculature was not compromised. However, in the case of complete

avulsion, immediate microsurgical revascularization of the amputated tongue may be the only opportunity for salvage. The literature indicates three successful cases in which near complete transection of the tongue was successfully treated by revascularization.^{46,47,48} The best results should be anticipated if revascularization occurs within the 8-hour threshold commonly accepted for revascularization of muscular tissue. However, one of the three successful cases in the literature chronicled a 16-hour ischemic period before microvascular anastomosis of the lingual artery and vein was completed.

If microscopic revascularization fails or is not attempted for complete amputation of the tongue, successful salvage is rare. And while the tongue plays a critical role in swallowing, speech, and airway protection, some patients are able to maintain a good quality of life and perceive the injury's impact on articulation as minimal should the avulsed segment be lost (Fig. 44.8).

In the more serious cases of severe traumatic injury, it must be appreciated that the patient may have sustained other injuries. A patient should be stabilized prior to any treatment from the surgical specialist. Often the patient may have sustained neurological trauma or be temporarily comatose, and prevention of trauma to the soft tissues of the tongue is the goal. Hanson and Wood lay out methods for fabricating tongue splints that will aid in the prevention of unconscious trauma while the patient is incapacitated.^{49,50}

Traumatic injury to the tongue is often benign and limited to children; however, serious airway consequences may develop in more extreme cases. Control of bleeding and aggressive irrigation prior to closing wounds will generally lead to healthy repair. Postoperative examination of more significant injury



Fig. 44.7 (a) A patient sustained trauma to the tongue with resultant hematoma and significant venous congestion. (b) Leeches can be used to relieve the venous congestion and restore vascular integrity to the tongue.

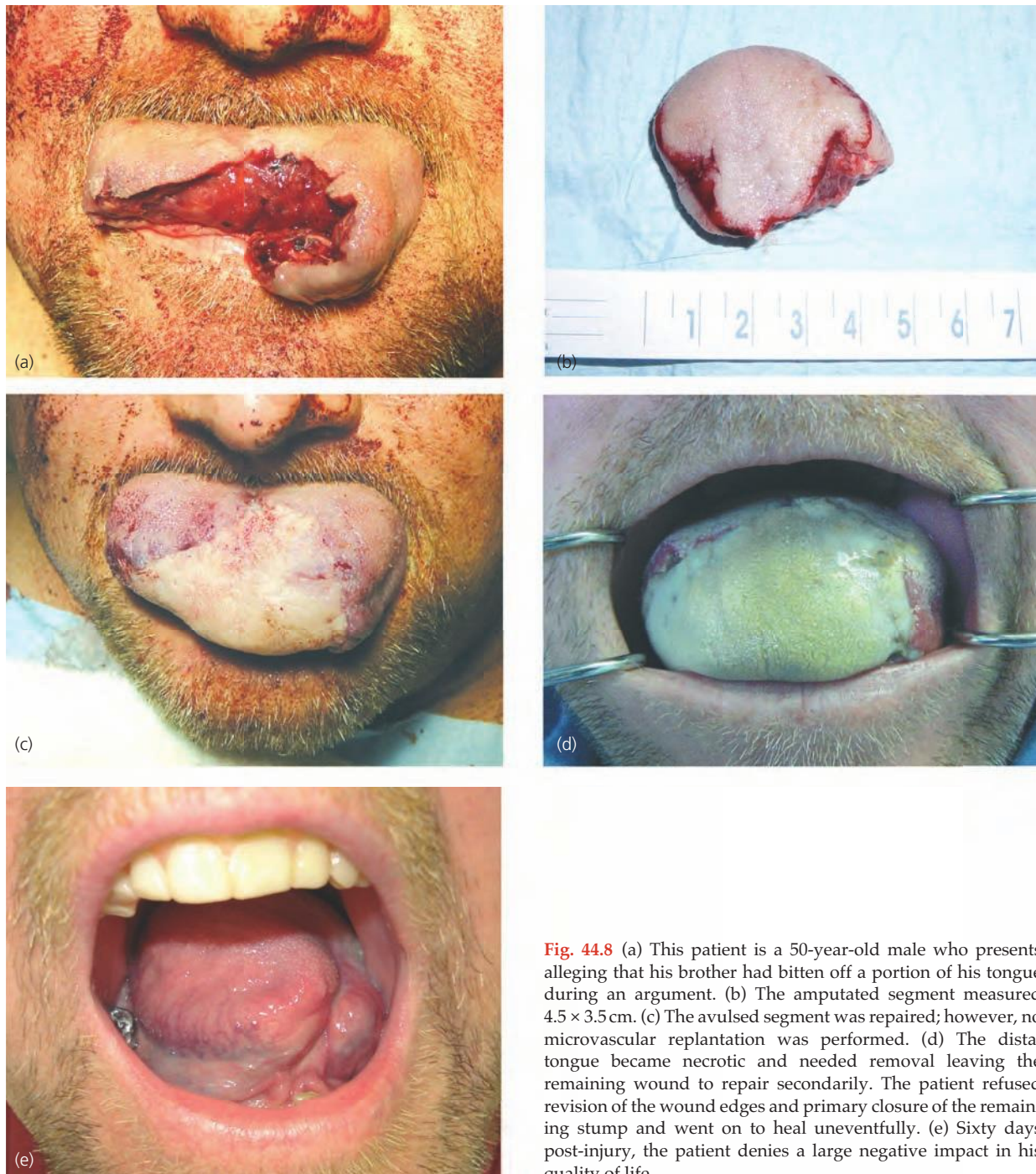


Fig. 44.8 (a) This patient is a 50-year-old male who presents alleging that his brother had bitten off a portion of his tongue during an argument. (b) The amputated segment measured 4.5×3.5 cm. (c) The avulsed segment was repaired; however, no microvascular replantation was performed. (d) The distal tongue became necrotic and needed removal leaving the remaining wound to repair secondarily. The patient refused revision of the wound edges and primary closure of the remaining stump and went on to heal uneventfully. (e) Sixty days post-injury, the patient denies a large negative impact in his quality of life.

should always include examination of lingual nerve function and taste sensation.

Nose

Due to the protrusive position of the nose from the midface, many cases of facial trauma include a nasal component. The nose is a complex structure with an array of tissue components that are dynamic during growth and eventually become central to adult facial esthetics. Nasal trauma may present in combination with other facial injury; however, many affect the nose alone. Knowledge of the nasal anatomy in combination with a basic understanding of the biome-

chanics of the injurious impact is of paramount importance when planning treatment of nasal trauma. Thorough evaluation, diagnosis, and treatment planning of soft tissue injuries to the nose will provide the most acceptable prognosis for repair.

Evaluation of nasal trauma can be a complex task. With the amount of edema associated with nasal or other facial trauma, palpation and visualization may have minimal value if delayed. Ecchymosis is often present around the orbits and significant edema is generally appreciated. Traumatic telecanthis or crepitus around the upper eyelid should always be further investigated to rule out naso-orbital-ethmoid (NOE) fracture or medial wall fracture. An accurate evalua-

tion is generally best obtained immediately upon admission, including CT scan, airway assessment, rhinoscopy, and, most importantly, comprehensive digital palpation.

Evaluation is often divided into three nasal compartments: upper, middle, and lower.⁵¹ These anatomic compartments will aid the surgeon in establishing breadth of injury and extent of surgical repair. Because the nose as a unit consists of bone, cartilage, and skin so tightly connected, a discussion of soft tissue trauma to the nose must include all of the components. Positive upper vault compression evidenced by digital palpation indicates nasal bone fracture and often disunion from the frontal bone. A positive result is evidenced by collapse at sellion and apparent “disappearance” of the nose into the ethmoid sinuses.⁵¹ Upper compartment injury most often indicates need for manual reduction often in combination with bony fixation. Positive middle vault compression results in nasal tip deflection superiorly, as the injured septum gives way below the bony-cartilaginous junction. Pressure applied at columella or the nasal tip resulting in collapse or hyper-rotation indicates injury to the lower vault. This method of classification is consistent with the Brown-Gruss system. The age of the patient will also impact delivery of care, especially when bony or cartilaginous involvement is appreciated. Facial growth centers such as the septo-vomerine suture may be impacted and more serious esthetic sequelae could be expected as the child reaches nasal

maturity at 18–20 years in males and 16–18 years in females. Importantly, disruption of the external branch of the anterior ethmoidal artery is more common in children due to the lack of osseous protection afforded in the mature skeleton; this problem is generally absent in adult populations. Control of hemorrhage, especially in children, with nasal packing is always advisable to prevent hematoma formation. Frequent examination of the septum is critical because septal hematoma is significant and a potentially devastating sequela of nasal trauma. Untreated, this can lead to infection, necrosis, and resorption of septal cartilage. If large enough, septal collapse occurs resulting in saddle-nose deformity. Meticulous evaluation and diagnosis of nasal injury will lead to more predictable repairs.

Soft tissue injuries to the nose are often comprised of skin flaps, cartilage tears, mucosal violations, or a combination. A three-layer closure is necessary to repair full-thickness lacerations. Due to the complex anatomy of the nose and variation in vectors of impact, these flaps are often irregular in shape which may prove challenging to the surgeon. Thorough assessment of the viability of the flaps is often the most critical and unfortunately the least sophisticated and predictable objective. Capillary refill, color, and bleeding are three clinical indicators used to predict viability; however, no one finding should be trusted individually. A bluish, congested flap with thinned borders will often breed a questionable to poor prog-

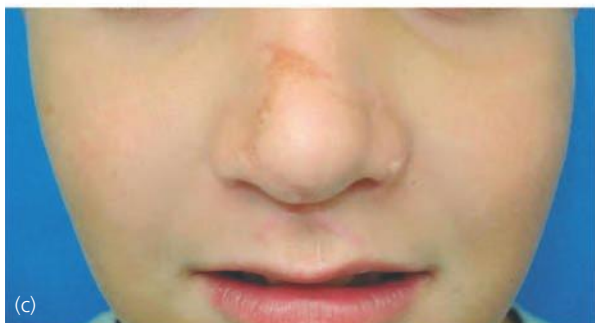


Fig. 44.9 (a) A young patient sustained a partial avulsion of his nasal tip which appears dusky. (b) The small pedicle is apparent after closure of the flap. (c) The patient presents after healing. Some flaps with questionable viability may go on to succeed.

nosis, while more viable flaps will tend to be pink and free of ischemic elements. As discussed elsewhere in this chapter, hirudism (use of leeches) is a viable treatment for more severe flap injuries with signs of moderate to severe venous congestion. It must be remembered that even the most clinically viable appearing flaps may often be lost, and conversely the most questionable may succeed (Fig. 44.9). Avulsive injuries may be treated with regional flaps for primary repair. More progressive therapies including island composite nasal flaps, prefabricated porous alloplastic materials, and free autogenous grafts are often considered when flaps are lost and secondary repair becomes the goal. Avulsive injuries may also be left open, allowed to heal and reconstructed at a later time.

Gentle but thorough irrigation of the wound along with removal of any foreign elements should be undertaken. Failure to do so may lead to infection and jeopardize the surgical repair. Closure of any nasal wound should be directed towards healing by primary intention, along with emphasis on esthetics and function. Repair should start at the ala if involved, maintaining it in proper position. When repair begins cranially and progresses caudally, there is a tendency for the alar rim to be displaced superiorly, leading to an unesthetic result. Deeper lacerations should be closed in individual layers, using fine absorbable sutures for mucosa, and absorbable sutures to approximate separated cartilage, perichondrium, and muscle layers. Failure to reapproximate important muscle layers, including any involved muscles of facial expression, will have severe implications on the patient's esthetic outcome. Furthermore, the cartilaginous components provide the nasal tip with its major support. This support is derived from the size, shape, and resilience of the medial and lateral crura, the attachment of the medial crus to the septal cartilage, and the attachment of the upper and lower lateral cartilages.⁵² Careful attention should be paid to these areas when reconstructing the nose to provide the most esthetic result. Often, more severe trauma will necessitate reapproximation of cartilage to the bone underlying the superficial rhinion, the point where the nasal bone and upper lateral cartilages meet. Functional impairment may result, with nasal passage stenosis, if the upper lateral cartilages are not positioned properly in relationship to the cartilaginous septum. The surgeon must keep both functional and esthetic implications in mind when treating these cases. Intranasal stenting after mucosal repair may help prevent or minimize the development of stenosis or synechiae. Final flap closure should approximate clean healthy borders with fine nylon suture for a better result. More severe injuries may necessitate multiple surgeries, however most superficial injuries may be treated in the clinical environment with local anesthetic. Steri-strips (3M Healthcare, St. Paul, MN), antibiotic ointment, in combination with other protective dressings, are all encouraged.

After treatment, postoperative instructions should be given, steroid or anti-inflammatory medications are often prescribed, and the patient should be followed closely to ensure proper healing. The patient should be evaluated at regular intervals to assess any sensory deficit including anosia or dysgeusia. Any evidence of hematoma formation must be immediately addressed. Hematoma formation is most often diagnosed via anterior rhinoscopy as a bulging of the lateral nasal wall between the caudal edge of the upper lateral nasal cartilage and the cephalic margin of the alar cartilage. Muscles of facial expression should be tested and appropriate sinus drainage should be verified. Nasal injuries are among the most common facial injuries and also are often difficult to treat. Careful closure with the appropriate materials in conjunction with realistic patient expectations will result in the most successful cases.

Ear

Traumatic auricular injuries consist of lacerations ranging from simple to full thickness, avulsions or partial avulsions, and hematomas. The auricle consists only of skin, a small amount of subcutaneous tissue, and cartilage. The key to repairing lacerations on the external ear is maintenance of the cartilaginous framework. If simple, the laceration can be repaired with interrupted non-absorbable sutures such as 5/0 nylon to approximate the skin. If cartilage is disrupted, a minimal number of fine, absorbable intercartilaginous sutures should be placed. This is followed by closure of both skin surfaces and placement of a molded pressure dressing. With avulsions or partial avulsions, even small segments will usually survive due to the abundant blood supply. The first landmark for replacement of the partially avulsed ear is the external auditory canal. Failure to appropriately realign the canal can lead to stenosis – a complication which is difficult to correct later. The cartilage should be sutured in a clockwise fashion around the canal followed by approximation of the helical rim. Once these landmarks have been established and sutured, the remaining cartilage and skin can be closed.

Blunt injury to the ear can cause a hematoma which requires immediate drainage and application of pressure dressing in order to prevent clot organization and fibrous distortion of the auricle, or “cauliflower ear”. If needle aspiration of the hematoma is unsuccessful, a small incision should be made through which to evacuate the blood clots. Recurrence is likely unless pressure is applied from either mattress sutures or a molded pressure dressing which can also be sutured into place (Fig. 44.10).

Complete avulsions of the ear may be treated by either replantation or implantation. Replantation can be considered if the repair occurs soon after injury, if the injury is clean, and there is little damage to the avulsed segment. Once the replantation is performed and vascularity is re-established, it is important to



Fig. 44.10 Pressure is applied to the repaired helix from a pressure dressing held in place with mattress sutures that compress the perichondria to prevent hematoma development.

deal with the resultant venous congestion. This may be done by making multiple skin incisions in the congested area or using leech therapy. Mladick *et al.*⁵³ described a technique which they called the “Pocket Principle” for implantation of the avulsed ear. The avulsed ear is dermabraded and then reattached. The dermabraded reattached ear is then buried into a retroauricular subcutaneous pocket for 3–4 weeks where revascularization and epithelialization occur. The auricle is then bluntly dissected from the pocket and externalized. Many modifications of this technique have been described to overcome problems such as poor vascularization or poor soft tissue coverage upon externalization of the avulsed ear.

Eyelid injury

Traumatic injuries to the eyelids often present in combination with other periorbital injury including, but not limited to, fracture of the orbital rims and medial or lateral wall fractures. Soft tissue injuries involving the periorbital area are of high importance due to the esthetic and functional consequences of disrepair. Injuries to the eyelid may range from simple surface lacerations to complex globe injuries involving muscles for retraction of the eyelid, suspensory ligaments, tarsal plates, and conjunctiva. The implications of improper repair or misdiagnosis of injuries to these structures may lead to esthetic disturbance and loss of symmetry as well as significant dysfunction.

Evaluation is the critical step for injuries involving the periorbital structures. Evaluation should include examination of visual acuity, extraocular muscle mobility, V2 sensory evaluation, as well as mechanical palpation. Consultation with an ophthalmologist is often indicated. The layers of the eyelids incorporate many tissue components that are often difficult to visualize without high-powered magnification. Due to limitations in the acute setting of injury, loupe

magnification is often the preferred method of evaluation of fine structures. Knowledge of the periorbital anatomy is of paramount importance when determining extent of injury and during repair. Evaluation of deep laceration injury necessitates examination of each layer of the eyelid from skin, orbicularis oculi, retractor muscles, ligaments, tarsal plates, and conjunctiva. Each layer will have consequences on outcome and involvement of specific complex structures may impact the final prognosis.

Injuries to the upper eyelids are generally more complex and more difficult to repair than lower eyelid injuries.^{54,55} The upper eyelids are anatomically complex and serve to protect the globe from injury. Upper eyelid injuries can involve the lacrimal gland or drainage apparatus and, with improper repair, lead to epiphora. The lacrimal gland is an almond-shaped gland which is housed in the lacrimal fossa adjacent to the superior and lateral rectus muscles. It is divided into a palpebral and deeper smaller orbital lobe by the aponeurosis of the levator palpebrae superioris. During wound exploration of the upper eyelid, attention must be given to determine lacrimal gland involvement. The more pink, vascular, and firm quality of the lacrimal gland will allow its differentiation from orbital fat. Care must be taken to ensure its reposition into the lacrimal fossa during avulsion or severe laceration in order to restore continuity and function (Fig. 44.11).

Injury to the upper eyelid retractors has serious esthetic and functional repercussions. Levator palpebrae superioris, the levator aponeurosis, and Muller's muscle all act as eyelid retractors for the upper eyelid. Injury to these muscles must be included in the primary repair or eyelid ptosis is often the result. Reattachment of Whitnall's and Lockwood's suspensory ligaments will also minimize globe ptosis. Laceration of the medial palpebral ligament causes the lid to displace laterally. Furthermore, approximation of the conjunctival layer is beneficial to prevent complications including symblepharon and eyelid retraction.⁵⁴

Simple surface lacerations involving the eyelids are often of little consequence. The eyelids employ a thin skin layer with high regenerative capacity that most often leads to quick and esthetic resolution with proper closure.⁵⁴ Fine suture such as 6/0 nylon should be employed to close these surface lacerations. If non-compliance with removal is anticipated due to a patient's age, rapid absorbing sutures can be placed. Studies reveal no difference in cosmetic outcomes in facial lacerations closed with absorbable sutures compared to nylon.^{18,19,56}

Lower eyelid injuries are often less complex anatomically and produce less significant complications in repair. However, the lower eyelid can be the site of very complex injuries which may include different degrees of tissue loss necessitating flap advancement or donor tissue to achieve primary closure (Fig. 44.12).

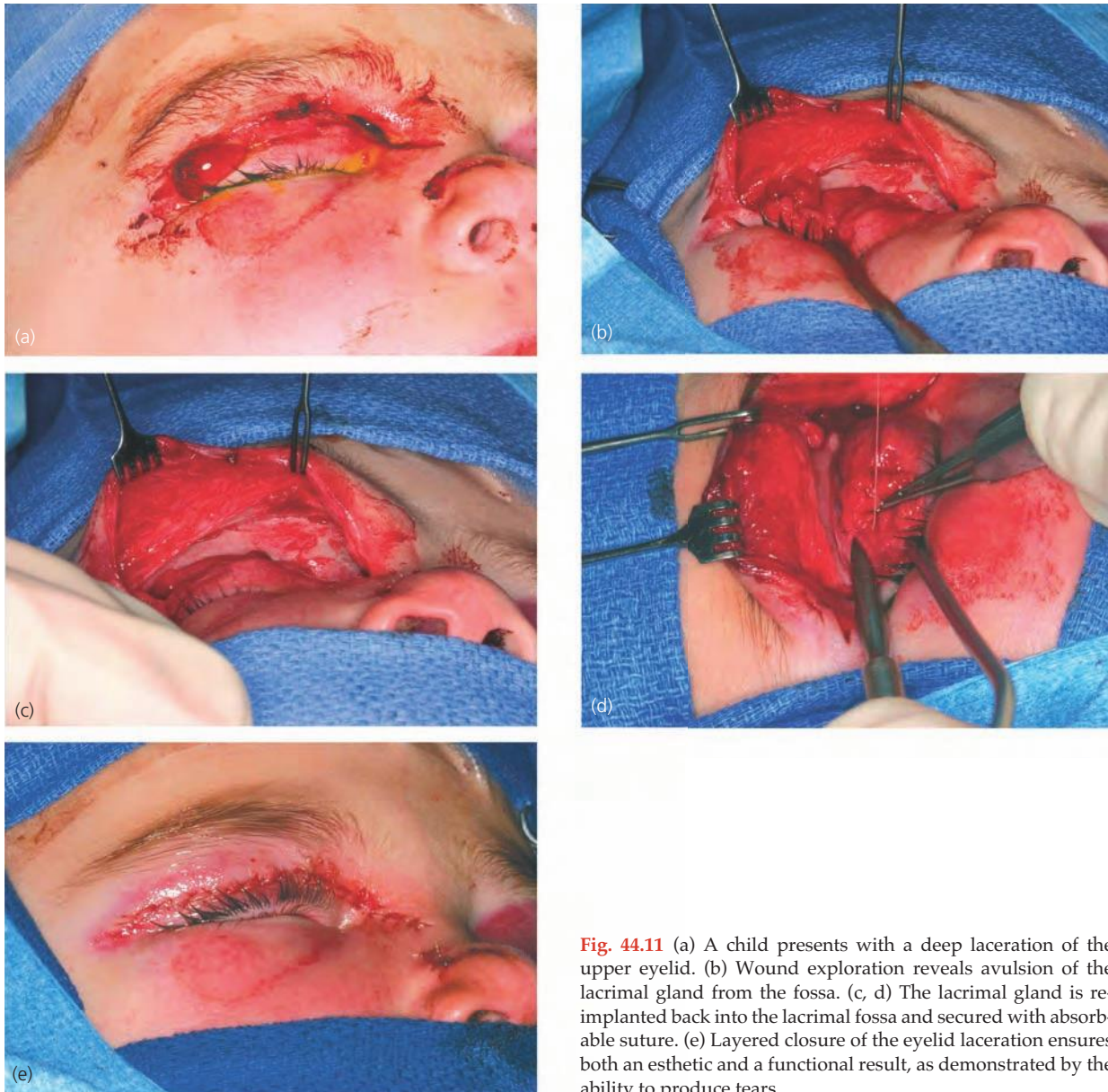


Fig. 44.11 (a) A child presents with a deep laceration of the upper eyelid. (b) Wound exploration reveals avulsion of the lacrimal gland from the fossa. (c, d) The lacrimal gland is reimplanted back into the lacrimal fossa and secured with absorbable suture. (e) Layered closure of the eyelid laceration ensures both an esthetic and a functional result, as demonstrated by the ability to produce tears.

Donor tissue is most often taken from the contralateral eyelid, but has also been harvested from supraclavicular and periauricular areas.^{54,55,57} Full-thickness injuries should be closed in layers, with particular attention paid to the tarsal plate and levator aponeurosis reapproximation. Eyelid margin involvement necessitates marginal reapproximation using fine chromic suture to prevent eyelid notching.⁵⁴ Lacerations involving the medial canthus may present with traumatic telecanthis, medial canthal blunting, and epiphora, and often signify complex injury.⁵⁴ Traumatic telecanthis is often a sign of NOE fracture or medial wall fracture, and is usually defined as any measurement greater than 33mm. Reattachment of the medial canthal tendon will alleviate pressure on the lacrimal apparatus and help avoid chronic epiphora.⁵⁴ Complex eyelid reconstruction often yields poor functional results and revision is almost always necessary.^{55,57}

Eyelid injuries may be very complex. Thorough assessment will allow determination of all of the structures damaged and in need of repair. The upper and lower eyelids house the tear drainage system which may be involved in complex lacerations. The canaliculi and the lacrimal apparatus are at risk of injury when trauma to the eyelids occurs (Fig. 44.13). Thorough evaluation at the time of injury may prevent long-term complications from an undiagnosed injury. The prognosis often depends on the location and depth of injury, associated structures involved, and availability of tissue for repair.

Canaliculi and lacrimal apparatus

Disruption of the nasolacrimal apparatus can occur from shearing forces to the midface which have also involved the bony skeleton. Dysfunction of tear flow can also be caused by a purely soft tissue injury



Fig. 44.12 (a, b) A male patient presents with a soft tissue avulsion of the lower eyelid. (c–g) Adjacent tissue was rotated to provide coverage of exposed conjunctiva and create a new lower lid.

involving laceration of the canaliculi. The nasolacrimal apparatus begins as the eyelid puncta drain to the medial canthus, and continues as the canaliculi, the common canaliculus, the lacrimal sac, and finally the nasolacrimal duct. The nasolacrimal duct courses from inferior to the middle canthus through the nasolacrimal canal to its opening in the inferior nasal meatus. Injury may occur at any point along the

apparatus, most commonly in combination with LeFort II and LeFort III fractures or with penetrating injuries.⁵⁸ NOE fractures also show high association with injury to the lacrimal apparatus, particularly with dacryostenosis in the months following injury.⁵⁸ These injuries may be highlighted on CT scan, or through digital subtraction macrodacryocystography, which incorporates a radioactive dye through



Fig. 44.13 (a) A motor vehicle accident is the cause of these facial lacerations including a laceration of the medial aspect of the upper and lower eyelids. (b, c) Thorough evaluation of the wound reveals lacerated superior and inferior canaliculi which were intubated using a Crawford tube and dacryocystorhinostomy was performed to prevent chronic epiphora.

the nasolacrimal apparatus. Although injury to the lacrimal apparatus is relatively rare in most midfacial trauma, thorough assessment can minimize the likelihood of the chronic complications involved in lacrimal injury.

Any injury to the orbit, especially the medial orbit and bridge of the nose, should increase suspicion of an injury to the lacrimal apparatus. As noted previously, symptoms may present acutely, but most commonly present months following midfacial injury. The most common symptom is epiphora, while others include visual impairment, midfacial anatomical distortion, nasal ecchymosis, and palpable step deformities involving the inferomedial orbital rim.⁵⁹ Although epiphora is the most common symptom involving traumatic injury to the lacrimal apparatus, it is most often appreciated as a chronic and persistent complication following midfacial injury. Further, the most common diagnoses related to symptoms of epiphora are inflammation/infection, obstruction, and tumor, which may further complicate diagnosis in minor injuries.⁵⁹ However, any patient presenting with epiphora should be questioned regarding any past trauma involving the associated structures.

Dacryocystorhinostomy is the standard treatment for diagnosed injury to the nasolacrimal apparatus. Many techniques exist, yet most center around the placement of silicone intubation systems or Crawford tubes while the system re-epithelializes and heals (Fig. 44.14).^{59,60}

This treatment has become well accepted; however, the current debate centers on prophylactic intubation of the system in all midfacial injuries. Traditional

management has emphasized correct fixation and wide open reduction of the fracture segments surrounding the pathway, to ensure osseous patency for the duct.⁵⁹ After proper fracture management, the lacrimal system may be addressed during a second surgery at 6 months postoperatively for those who present with symptoms. If segments surrounding the duct are allowed to heal out of anatomical position the duct could be obliterated, leading to long-term complications. Furthermore, many believe that achieving early and accurate repositioning helps avoid compression of the upper lacrimal pathways and some degree of telecanthis, which may result in decreased need for secondary surgery.⁷ More aggressive management has been predicated upon the high percentages of lacrimal complications associated with certain injuries. Studies cite figures that 20–47% of patients with NOE fractures experience nasolacrimal obstruction following their primary surgery.^{59,61} Spinelli *et al.* concluded that the addition of lacrimal intubation to the complex primary repair of selected midfacial injuries may indeed obviate the need for secondary lacrimal bypass surgery resulting from late cicatricial obstruction of the nasolacrimal duct.⁵⁹ Thorough irrigation and dye testing following intubation will ensure proper positioning and patency, regardless of when the procedure is performed.

Injuries to the lacrimal apparatus may result in serious long-term consequences in the form of persistent obstruction and recurrent epiphoral episodes if not treated. Prophylactic intubation of the nasolacrimal duct in patients experiencing fractures to high-risk areas, should be considered.⁵⁹ Patients with

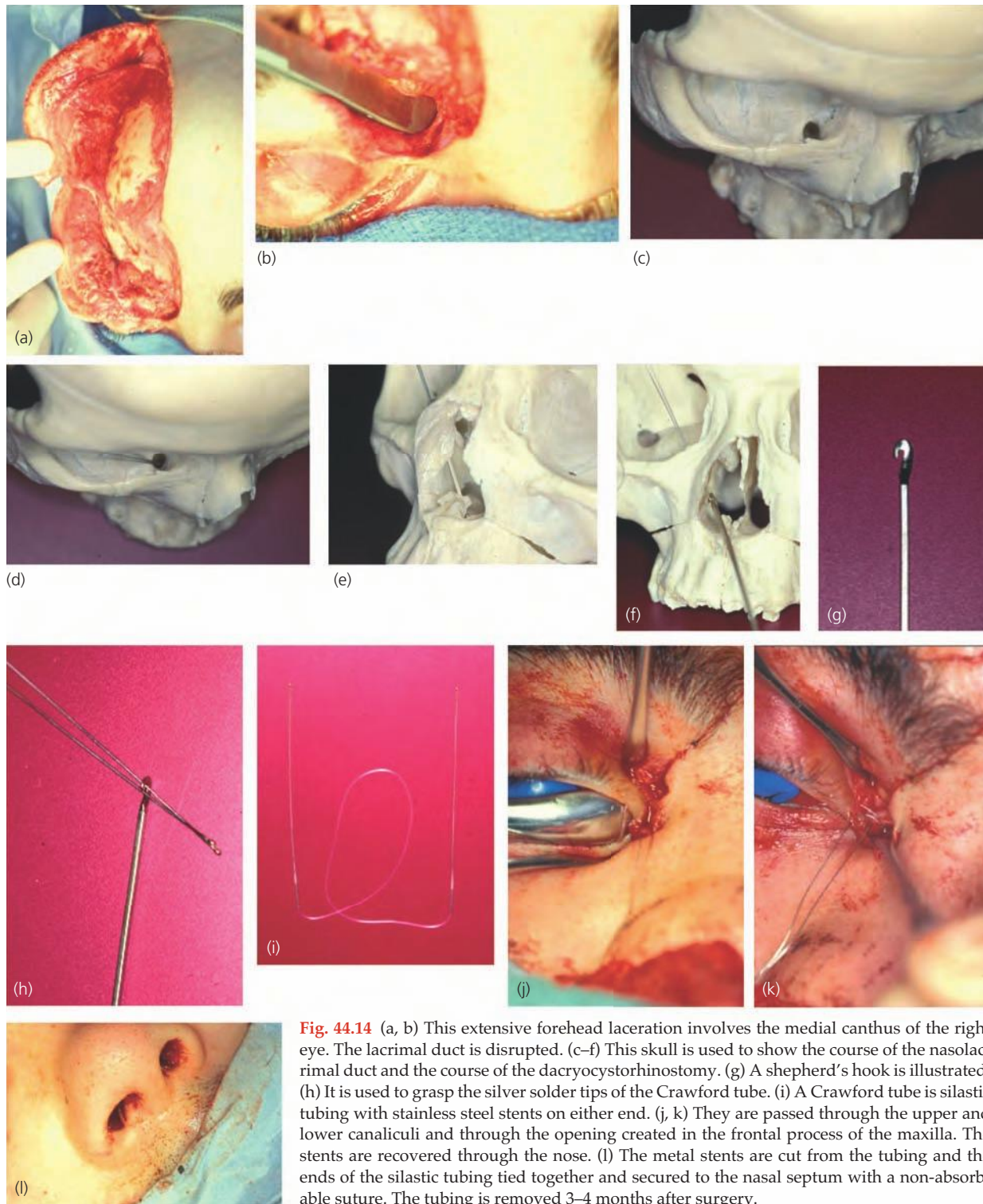


Fig. 44.14 (a, b) This extensive forehead laceration involves the medial canthus of the right eye. The lacrimal duct is disrupted. (c–f) This skull is used to show the course of the nasolacrimal duct and the course of the dacryocystorhinostomy. (g) A shepherd's hook is illustrated. (h) It is used to grasp the silver solder tips of the Crawford tube. (i) A Crawford tube is silastic tubing with stainless steel stents on either end. (j, k) They are passed through the upper and lower canaliculi and through the opening created in the frontal process of the maxilla. The stents are recovered through the nose. (l) The metal stents are cut from the tubing and the ends of the silastic tubing tied together and secured to the nasal septum with a non-absorbable suture. The tubing is removed 3–4 months after surgery.

injury to the nasolacrimal apparatus should be followed closely for signs and symptoms associated with the injury at 1 week, 2 weeks, 1 month, 6 months, and 1 year.

Parotid duct and capsule

While parotid gland and duct injuries are rare, recognition of their occurrence in facial soft tissue trauma is critical. The complications of missed parotid capsule or duct injury are difficult to resolve. Standard

treatment protocols for these complications are controversial and have varying degrees of success.

The primary cause of parotid duct injury is from penetrating trauma, including glass from motor vehicle accidents, stab wounds, and gunshot wounds.⁶² Injuries range from involvement of the gland capsule, parenchyma, and/or duct, to injury of adjacent structures such as branches of the facial nerve. Treatment modalities include surgical repair and non-surgical management dependent on the structures damaged and the extent of their injury as well as the timing of

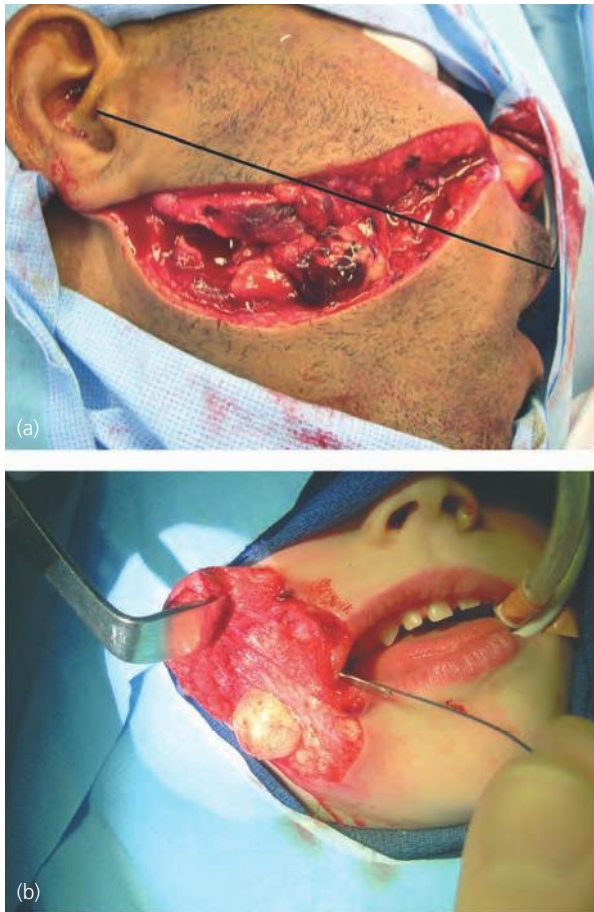


Fig. 44.15 (a) The main parotid duct runs in a trajectory that can be approximated by a line drawn from the tragus of the ear to the middle of the upper lip. (b) Any cheek laceration that involves this area must result in a high degree of suspicion for duct injury.

presentation. Frequently, injuries are not recognized at initial trauma evaluation.

The main parotid duct runs in a trajectory that can be approximated by a line drawn from the tragus of the ear to the middle of the upper lip. Any cheek laceration that involves this area must result in a high degree of suspicion for duct injury (Fig. 44.15). When ductal injury is present, injury to branches of the facial nerve is likely. Nerve injuries are covered elsewhere in this chapter.

Thorough wound inspection must be performed in order to determine the extent of damage to the gland and duct. Wound examination includes milking the parotid gland with intraoral examination of the duct to verify salivary flow. The anatomic location of the injury can also provide guidance for choice of management. Van Sickels and Alexander separated parotid duct injuries into three areas according to anatomic location:⁶³ injury of the duct during its course within the gland, over the masseter muscle, and anterior to the masseter. Lacerations of the gland can be managed with closure of the capsule and placement of a pressure dressing for 1 week. Frequent examination would reveal any salivary accumulation, which should be aspirated and the pressure dressing maintained.

A more frequently occurring injury is laceration of the duct over the masseter muscle. Injuries in this region can be treated by one of several methods including primary repair by microsurgical anastomosis, ductal repair with autogenous graft, ligation of the proximal duct to atrophy the gland, or non-operative management. The duct should be cannulated intraorally with lacrimal probes and if any doubt exists about the presence or quantity of saliva coming from the duct, the duct should be cannulated with a 20-gauge angiocatheter and injected with a small amount (1 ml) of methylene blue. If the duct is intact, this technique can provide information about the integrity of the parotid capsule. If the duct is transected, it will localize the distal cut end. The proximal cut end can then be localized by reapproximating the wound and following the lacrimal probe or catheter. The proximal cut end can also be found by milking the parotid gland and looking for saliva in the wound. If both proximal and distal stumps of the duct are visible, Stenson's duct can be cannulated with a hollow Silastic (Dow Corning, Midland, MI) catheter (16–22 gauge pediatric cardiac catheters are ideal) with a length of about 8–10 cm and the duct ends pulled together over the catheter. The stump ends can then be sutured with 9/0 or 10/0 nylon suture over the catheter, which maintains the integrity of the duct during suturing and prevents the posterior wall of the duct from being captured within the repair. The tube passed through the duct orifice into the mouth is sutured to the buccal mucosa. The catheter is maintained for 14 days along with external pressure dressing for 3 days.

If the cut ends of the duct will not reapproximate without tension, or when a segment of the duct is missing making direct anastomosis impossible, one option is to ligate the proximal duct allowing the gland to undergo atrophy. Drain placement may be necessary to prevent sialocele formation while the gland undergoes atrophy.⁶⁴ While this is the common method of treatment for duct ends which are not able to be anastomosed primarily, Chudakov and Ludchik describe their experimental study in dogs where venous autografts were used to complete the repairs.⁶⁵

When the duct injury occurs anterior to the masseter muscle, creation of an intraoral fistula may be indicated if duct repair is not possible. This is done by suturing a cannulated proximal duct after reimplanting it through the buccinator and buccal mucosa at the level of Stenson's papilla (Fig. 44.16). In cases where the duct is reanastomosed or an intraoral fistula created, the parotid gland is prone to obstructive sialadenitis. A course of antibiotics can be considered to prevent infection, and sugar-free lozenges can be used to stimulate saliva.

Conservative treatment of parotid duct injuries has also been advocated by some. Lewis and Knottenbelt describe a prospective study where they investigated the non-operative management of 19

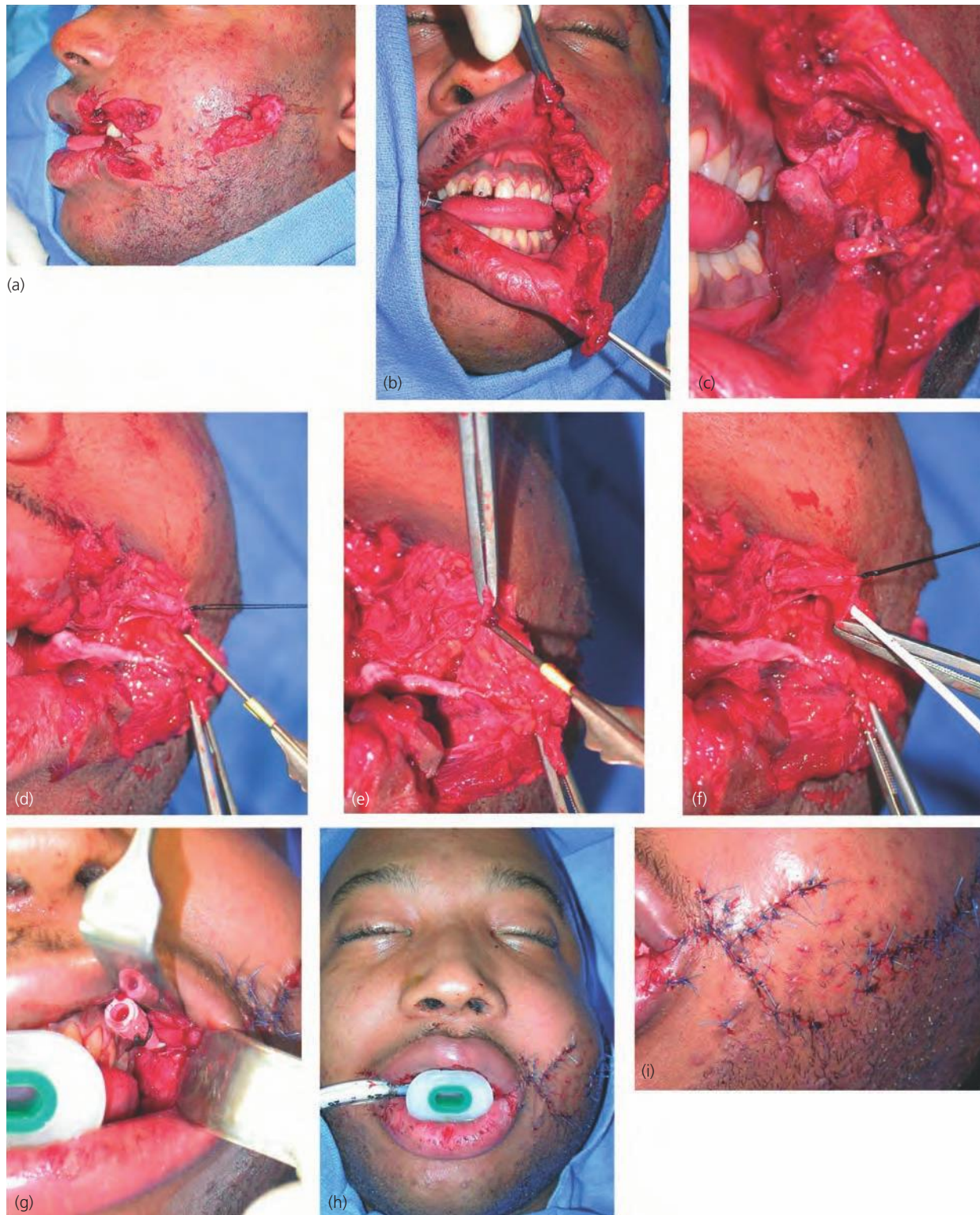


Fig. 44.16 (a, b) A young patient presents with a gunshot wound to the face creating a through-and-through wound at the level of the parotid duct. (c) Examination reveals a parotid duct laceration. (d, e) The proximal duct is localized and cannulated with progressively larger lacrimal probes. (f) It is then cannulated with a hollow silastic tube. (g) The tube is replanted within the buccal mucosa at the level of Stenson's papilla and sutured into place. (h, i) The external wounds are closed in layered fashion. The catheter is maintained for 14 days with an external pressure dressing maintained for 3 days.

patients with confirmed parotid duct injuries.⁶⁶ All patients healed without surgical intervention. The 53% of patients who had minor complications healed with the use of sialogogues for fistulas and weekly aspiration of sialoceles. The remaining 49% healed without complication. The use of Botox has also been shown with good success to resolve parotid fistulas.⁶⁷

Botulism toxin type A inhibits salivary production by its anticholinergic effect on parasympathetic secretory motor fibers. Without constant salivary flow, the fistula will then resolve.

While sialography may be useful in the postoperative period to evaluate response to treatment, or to help identify undiagnosed parotid injuries, it has little

utility in the face of acute injury. That being said, sialography may help diagnose diffuse rupture of the gland parenchyma which can occur from blunt trauma to the parotid region.⁶⁸ Helical CT scan is a more useful diagnostic aid for acute traumatic injury to the salivary glands.⁶⁹ Magnetic resonance imaging (MRI) shows promise as a useful method of evaluation as better techniques are developed. High-resolution MRI studies, like sialography, are not practical tools for use in the acute injury setting; however, they may be useful for visualization of abnormal salivary duct systems following trauma. Using certain reconstructions of this image, fine anatomic details such as the parotid duct and facial nerve can be visualized – even within the gland itself. 3D reformations can also be created to allow visualization of the anatomy in precise planes.⁷⁰

Facial nerve

Injury to the facial nerve can have devastating functional and esthetic consequences. Facial nerve injury is likely to occur in penetrating injuries to the face and gunshot wounds where thermal injury radiates through large portions of tissue. Evaluation of an injury for facial nerve involvement is relatively easy to accomplish in a conscious, cooperative patient. Inability of the patient to move the muscles of facial expression suggests nerve transection. In an unconscious patient, examination will include the use of a nerve stimulator. Like evaluation for parotid duct injuries, anatomic location of the wound guides treatment for facial nerve injury. Nerve lacerations which occur medial to a line drawn from the lateral canthus of the eye to the mental foramen do not need primary repair due to their likelihood of spontaneous repair.⁶³ Wounds proximal to this line need to be explored for possible nerve injury with need for primary repair. Location of the wound with respect to the masseter muscle may also guide treatment, in that injuries which occur within the region of the masseter muscle and have involved the facial nerve are also likely to involve the parotid duct.

Ideally, facial nerve repair should be performed under magnification at the time of primary wound closure. If repair is performed within 48 hours of the injury, the use of a nerve stimulator can help identify the distal cut ends of the nerve; however, after 48–72 hours the nerve undergoes degeneration and the nerve stimulator is less likely to elicit muscle twitching from the distal nerve ends making their identification more difficult. The ends of the nerve should be cut to create a clean edge free from devitalized nerve tissue. The cut ends of the nerve should be mobilized and freed from the surrounding tissue, such that there is approximation without tension. The most important prognostic factor in facial nerve repair is tension-free anastomosis. While perineural repair is discussed throughout the literature as the most ideal, epineural repair may be more realistic. Two to three

sutures (8/0 or 9/0 monofilament) taken through the epineurium in a tension-free manner will approximate the segments and preserve the fascicles. When large nerve defects complicate the wound or tension-free approximation is not attainable, the use of interpositional grafts may be considered. Return of function may be delayed for up to a year after repair. Failure of the repair may be caused by tension or opening at the anastomosis or muscle or nerve fibrosis. Patients whose repair fails may be candidates for facial nerve rehabilitation.

Neck

Debates regarding the management of soft tissue injury to the neck have been documented dating from World War I to the present. After decades of changes in management protocols in efforts to optimize outcomes, a new strategy has emerged. The current strategy has been widely accepted in civilian settings across the US and has become available in large part by advances in technologic and diagnostic screening tools. Selective surgical intervention of traumatic injuries to the soft tissues of the neck in combination with strict observation of immediate and progressive indications for immediate surgical intervention have recently eclipsed the arguments in favor of mandatory surgical exploration for these types of injuries.

The neck serves as the conduit for many of the most fundamental organ systems, including the nervous system, aerodigestive system, and cardiovascular circulatory system. The neck houses the spinal cord, the laryngotracheal apparatus, the esophageal complex, and perhaps most critically the vessels supplying and draining oxygenated and deoxygenated blood to the brain, the carotid arteries, and the jugular veins, respectively. Penetrating neck injuries may cause damage to any of these or other critical structures, providing a challenging diagnosis for the surgeon and often a complex surgical plan. With advances in diagnostic imaging technology including computed tomographic angiography (CTA), multi-detector computed tomography (MDCT), and MRI, the surgeon's role in these cases has become clearer.

Mimicking general traumatic injury, most cases involving the soft tissues of the neck involve males in their 20s and 30s.⁷¹ Causation of injury is generally placed into three categories with few cases falling outside the top three: assault, motor vehicle accident, and self-inflicted injury. Different modalities of traumatic injury include both low and high-velocity bullet wounds, birdshot and buckshot shotgun wounds, and stab wounds to the neck.

Traumatic injuries to the soft tissues of the neck have long been anatomically classified into three zones.^{72,73} The most inferior zone, zone 1, extends from the sternal angle and clavicles to the inferior aspect of the cricoid cartilage. Zone 2 occupies the space from the cricoid cartilage to the angle and inferior border of the body of the mandible, the largest of

the three anatomic zones. The most superior zone, Zone 3 spans from the mandibular angle and body to the base of the skull. Penetrating neck trauma must also be classified in a second dimension, depth. Ordog uses a system of classification establishing depth of injury when categorizing shotgun wounds to the neck. Type 0 refers to the most superficial injury where pellets are in skin only, type 1 penetrating subcutaneous tissue, type 2 penetrating beyond deep fascia, and ultimately type 4 signaling extensive tissue damage.⁷⁴ Establishing a similar system to assess the depth of injury is often useful in determining management (i.e. penetrating injuries superficial to the platysma are often simply debrided and closed). Classification systems provide the structure necessary for proper assessment and management.

Clinical assessment of injury is often a complex task with these injuries. Patient presentation ranges from minimal discomfort to life-threatening airway compromise and profound shock. Presentation often depends on the systems affected, each component having the ability to present at different times post-injury. Notable are esophageal injuries which often go unnoticed upon admission due to their subtlety, yet 24-hour delay in diagnosis is associated with a significant increase in mortality.⁷⁵ Furthermore, an absence of initial signs or symptoms should be received with caution. Bell *et al.* reveal that Fogelman and Stewart's paper on penetrating neck injuries indicated that 70% of their patients with significant vascular injury showed no evidence of bleeding at the time of admission and 43% of their patients displayed no evidence of peripheral vascular collapse.⁷¹ Furthermore, 25% of patients with blunt vascular injuries will not display symptoms for 24 hours, and often a thromboembolic event resulting in acute ischemic stroke is the initial manifestation.⁷¹ Patients with any neck injuries, both penetrating and blunt, should be followed meticulously.

Signs and symptoms of injury differ not only in time of presentation, but also in proportion to which systems are affected. Hard signs of vascular injury, often reliable indicators of severe injury, include a bruit or thrill, expanding pulsatile hematoma, pulsatile hemorrhage, and pulse deficit, while softer vascular signs with less reliability include hypotension, stable hematoma, nervous system ischemia, and proximity to major vascular structure. Retropharyngeal or prevertebral air on cervical radiography should alert the surgeon to esophageal or hypopharyngeal perforation. Symptoms of esophageal injury include dysphagia, odynophagia, drooling, hematemesis, hoarseness, and air bubbling from a wound, while subcutaneous emphysema is often the most common presenting sign in significant injury to the aerodigestive tract.⁷⁵ Attention should be paid to the patient's complaints when conscious and any imaging or diagnostic armamentarium should be used as needed. MDCT, CTA, nasopharyngoscopy, laryngoscopy, barium swallow, bronchoscopy, and

four-vessel cerebral angiogram have all been validated as useful tools with relatively high sensitivity and specificity. The CTA has emerged as the most cost-effective and reliable screening tool to assess penetrating neck injury due to its speed, and ease of use by support staff.⁷¹ Bell cites a significant decrease in the number of neck explorations since screening with CTA, and a "virtual elimination of negative neck explorations".⁷¹

Although in near agreement regarding the selective surgical management of penetrating neck injuries, a clear list of injuries and signs and symptoms requiring mandatory surgical intervention is lacking. Although the lists are separate, most give high priority to airway collapse or compromise and extreme vascular injury. Ordog cites stage II hypovolemic shock not responding to fluid and blood resuscitation, stage III and IV shock, major pathology on physical exam, high-velocity bullet wounds, close range (<5 m) shotguns, and high-velocity shrapnel and explosive injuries all require mandatory surgical therapy.⁷⁴ Bell *et al.* include five indications for immediate surgical intervention regardless of zone, including exsanguinating hemorrhage, expanding hematoma, shock, airway compromise, and massive subcutaneous emphysema.⁷¹ While a shift in the field has been felt regarding selective management, the necessity of immediate surgical intervention for specific cases should be appreciated.

Patients will often present to the surgeon without cervical spine clearance and still in a collar from transport. Often judgment must be made regarding access to the neck wound while the patient is in the C-collar. It is generally felt that immediate urgent treatment of the penetrating neck wound should take precedence over concerns for the cervical spine, including removing a cervical collar to gain access to the injury when dealing with gunshot wounds. Extreme caution and scrupulous judgment should be used when addressing this dilemma.

Treatment of neck injuries is highly surgeon- and center-dependent and the contents of the neck often require a multidisciplinary approach. Differing surgical techniques are available for access, dependent on the zone of injury, balancing esthetics and ease of surgical exploration. Zone 1 injuries often require a combined transcervical and transthoracic approach, while zone 2 injuries are often accessed via a vertical incision anterior to the sternocleidomastoid muscle. Zone 3 injuries often necessitate horizontal incisions to adequately visualize their components. Management of the vascular component may be repair, bypass, ligation, or observation. Primary repair by reanastomosis is generally favorable, although grafting and ligation are sometimes necessary. Not all vascular injuries require surgical intervention and instead may be observed and followed in 1–2-week intervals. Such injuries include small intimal defects and pseudoaneurysms less than 5 mm. Routine repair of all carotid injuries is recommended by Bell in all

but comatose patients.⁷¹ The majority of esophageal injuries resulting in death occur from mediastinitis and sepsis, thus direct esophageal repair utilizing a one- or two-layered technique should be used to prevent abscess formation. Esophageal surgeries secondary to penetrating neck trauma have high rates of mortality. Management of the esophageal, laryngeal, and tracheal components of the injuries should be approached by the appropriate services.

Follow-up on patients with neck trauma should be frequent and thorough. In the first week many patients require additional support. Bullet wounds and other penetrating injuries display high rates of infection, with deeper penetrating wounds being more susceptible. Prophylaxis is recommended in many of these cases and should be contemplated by the surgeon. Vascular injuries have been shown to predispose certain patients, especially those with existing atherosclerosis, to thromboemboli and stroke.⁷⁵ Administration of systemic anticoagulation therapy has decreased this problem significantly. Protracted observation is recommended.

Scalp

The scalp consists of five layers: skin, subcutaneous tissue, the galea aponeurosis, loose areolar tissue, and pericranium. It is thick, inelastic, and has multiple extensive vascular anastomotic complexes which make achieving hemostasis difficult after injury. Severed blood vessels lack the ability to contract due to the inelastic nature of the scalp and can contribute to life-threatening hemorrhage after extensive laceration. It is important to assess the amount of hemorrhage that has occurred from the time of injury to the time of repair in these cases, especially for complex scalp lacerations in the pediatric population.

Treatment of lacerations of the scalp is similar to those of the face. The wound must be adequately cleaned, debrided, and reassessed. Thorough wound exploration can detect skull fractures that are less likely to show up on skull films.⁷⁶ Hemostasis can be achieved with electrocautery, Raney clips, or direct pressure, but control of bleeding prior to closure allows for easier examination and approximation. Direct pressure with gauze and pressure dressing can be applied and left in place for 30–60 minutes to control bleeding.⁷⁷ Local anesthesia with epinephrine also aids hemostasis. Hair removal is not necessary prior to closure and has not been shown to decrease infection rates.⁷⁸ Shaving hair from the skin increases the risk for wound infection; therefore, if it is necessary to remove hair, it should be cut with scissors.⁷⁹

A relatively new method of closing simple scalp lacerations is the hair apposition technique (HAT). Four to five strands of hair are taken in a bundle on either side of the laceration and crossed over the laceration. A mosquito hemostat is used to make one twist of the hairs to approximate the wound. A drop of tissue adhesive is then used to secure the hair twist.

In a randomized controlled multicenter trial, the HAT method was found to be at least as acceptable if not superior to standard suturing methods.⁸⁰ Advantages of the technique include less pain, shorter treatment time, high patient acceptance, no need for suture removal, less scarring, and less wound dehiscence. Contraindications to this method of scalp repair include contaminated, extensive lacerations, lack of hemostasis, and inadequate length of hair.

Simple lacerations of the scalp can also be closed primarily with sutures; 3/0 or 4/0 non-absorbable suture such as nylon or colored polypropylene makes for easy retrieval. Stapling of scalp lacerations has also been shown to be an effective, efficient, and cosmetically acceptable closure technique.⁸¹ When lacerations involve the galea, it needs to be approximated as much as possible and closed with resorbable sutures. This layered closure will prevent cosmetic deformity of the forehead associated with disinsertion of the frontalis muscle. Closure of this layer will also protect the underlying connective tissue, with its rich bed of emissary veins and direct connection to intracranial veins, from bacterial infection.

For large avulsive scalp defects, microsurgical reconstruction is a reliable option for providing coverage. Replantation of an acutely avulsed scalp and free flap coverage may provide better stability than traditional skin grafts.^{82,83} For infected or heavily contaminated wounds delayed closure or closure by secondary intention should be considered (Fig. 44.17). If pericranium is intact, a split-thickness skin graft can be used to cover the defect. If pericranium is missing, the skull must be decorticated with burr holes in order to allow secondary healing or in order for the skin graft to take (Figs 44.18).

After repair, large scalp lacerations may necessitate a pressure dressing in order to prevent hematoma formation. After 48 hours the pressure dressing can be removed and normal wound care can continue.

Postoperative untoward results

Delayed healing

Delayed wound healing is failure of the systematic progression through the phases of wound healing. Three classic phases are generally described in terms of wound healing: inflammation, proliferation, and maturation. Abnormal healing may manifest as a prolongation or failure of completion of any of these phases, resulting in problems such as infection, chronic inflammation, hypo- or hypergranulation, or over-repair.

Infection

Normal wound healing can be inhibited by high levels of bacteria. Bacteria and their by-products maintain a balance with host resistance in tissues.



Fig. 44.17 (a–c) Large contaminated avulsive scalp lacerations may be allowed to heal by secondary intention if pericranium is intact. Wet-to-dry dressings allow for mechanical debridement of the wound during healing.



Fig. 44.18 (a, b) A young female patient presents 5 days postassault with a large infected scalp laceration. The wound was cleaned, debrided, packed, and allowed to heal secondarily. (c–e) After adequate granulation of the wound, the wound was pexed. Monocortical screws were placed at the temporal crest of the skull. Sutures were placed through the temporalis and the muscle was anchored to the bone via the screws. (f) The wound was then able to be closed. (g) The wound is intact 2 weeks after closure.

Once this balance is disrupted by either an impairment of the host defenses or an increase in bacterial load, infection may result. Research has shown us that critical tissue levels of bacteria, a growth of more than 100,000 organisms per gram of tissue is consistent with clinical infection and delayed wound healing.⁸⁴ The face, due to its high vascularity, may be able to tolerate higher bacterial loads before infection ensues.

Diagnosing postoperative wound infection can be difficult due to the effects of cytokine release which look the same in both normal and abnormal healing: erythema, pain, edema, and increased temperature. A wound can be considered infected if purulence is present. Definitive diagnosis may be made from wound tissue biopsy cultures which reveal the presence of beta-hemolytic streptococci, or the presence of a critical level of bacteria (greater than 10^5 per gram of tissue). Due to their time requirement, tissue cultures may not be practical for diagnosis in some cases; however, Gram stain can be performed in 15 minutes and be useful for determination of a critical level of bacteria. If even one organism is visible upon rapid slide Gram stain technique, it is inferred that more than 10^5 organisms are present within the wound.⁸⁴

Unlike medical infections which are amenable to systemic antibiotic therapy, wound infections resolve with operative treatment. Presence of foreign bodies, sutures, necrotic tissue, or hematomas tip the balance in favor of the bacteria. Incision and drainage, debridement of necrotic tissue, excision of foreign bodies, and evacuation of hematomas all work at the site of infection to decrease the bacterial inoculum and prime the tissue for wound healing.

Chronic inflammation

Wounds subject to long-standing levels of bacteria fail to progress through the phases of wound healing. Inflammatory cytokines such as interleukin-1 and tumor necrosis factor- α are sustained at high levels within the wound and inhibit growth factor production.³⁷ Oftentimes, host defenses are impaired contributing to chronicity of the wound. Determining, by clinical evaluation alone, when a chronic wound can be closed – whether by direct closure, graft or flap – is difficult. The appearance of epithelialization is not predictable because this process can continue in sub-optimal wound healing, even when granulation has not progressed as well, thus forming a crater in the skin surface.

Quantitative bacterial testing (i.e. wound tissue culture) is a definitive method to determine when a wound has less than 10^5 bacteria per gram of tissue. When critical colonization is established, the wound cannot be closed and methods to decrease the bacterial contamination of the wound must be employed in order to optimize the wound for successful closure. Surgical debridement, frequent

cleansing, protection from continued trauma, and, if indicated, occlusion of the wound are techniques to optimize the wound. Other, more controversial techniques deserve discussion.

The use of systemic antibiotics is usually not indicated for chronic wounds except when sepsis, osteomyelitis, cellulitis, or other signs of invasive infection are present. If the chronic wound is associated with a mild local infection where the erythema present is within 1 cm around the perimeter of a wound, systemic antibiotics are not indicated.⁸⁵

When local measures can no longer control the infection, systemic antibiotics are indicated. However, many wounds may be amenable to local treatment before infection becomes invasive. Because ischemia may reduce a wound's bioavailability to a systemic antibiotic, topical antibiotics may be a good option for treatment of local infection. Topical antibiotic and topical antiseptic preparations have a lower incidence of systemic effects but the incidence of bacterial resistance with their use is unclear. Some research shows that resistance is decreased, while other studies claim that topical antibiotics are a major cause of antibiotic resistance.^{86,87}

In randomized controlled trials, enzymatic debridement and silver-releasing hydroalginate dressings have shown to be favorable for healing of chronic wounds.^{88–90} Enzymatic debridement is sometimes used after an initial sharp surgical debridement or in place of sharp debridement if the patient's medical history is complicated by a bleeding disorder; however, it is rarely needed in the maxillofacial region.

Over-repair

Keloids and hypertrophic scars are two types of abnormal scarring that develop as a result of soft tissue trauma. Keloids and hypertrophic scars fall into the category of fibroproliferative disease (FPD) of the skin, of which the pathogenesis is unknown. Hypertrophic scars present as raised, reddened, itchy scars that spontaneously flatten and soften, whereas keloids remain raised and thickened and may undergo periods of quiescence alternating with periods of growth. Hypertrophic scars are red in appearance and pruritic due to the increased vascularity. Keloids may be painful.

Keloids and hypertrophic scars both exhibit fibrous outgrowth; however, hypertrophic scars remain within the borders of the wound as opposed to keloids, which enlarge over the wound confines and may continue to grow indefinitely. While clinically and histologically similar, keloids and hypertrophic scars may not represent varying degrees of a single disease entity. Because of such differences in behavior, the pathogenesis may be completely different. Until the pathophysiology becomes clearer, patients with abnormal scarring will have little reassurance that any particular mode of treatment may eliminate their pain, pruritis, or chance of recurrence. Multiple

treatment modalities exist for keloids and hypertrophic scars; however, no single method has demonstrated superiority.

Compression therapy is a method employed to decrease scar formation and is commonly used in burn patients by the use of compression garments. The mechanism is thought to be that the hypoxic effect of the compression on fibroblasts causes fibroblast degeneration and ultimately less collagen formation. Another theory is that the mechanical effect of the compression causes the collagen fibers to be laid down in flatter ribbons rather than whorls. The use of compression is usually not practical for the prevention of keloid recurrence or minimization of scarring in the head and neck with the exception of ear keloids. Soft tissue trauma induced by piercing of the earlobe or auricle may lead to keloid formation in some patients (Fig. 44.19). Specialized pressure earrings or magnets may be worn continuously for 6 months after excision of a keloid or hypertrophic scar in an effort to reduce the chance of recurrence.^{91,92}

Silicone gel sheets which work by hyperhydration of the subcutaneous tissue, have shown some efficacy in reducing the symptoms and appearance of keloids; however, in the head and neck region, this method is not practical.⁹³ The occlusive nature of the sheets prevents water loss which in turn decreases capillary action, resultant inflammation, and subsequent colla-



Fig. 44.19 Soft tissue trauma induced by piercing of the earlobe or auricle may lead to keloid formation in some patients.

gen formation. The sheets should be applied after re-epithelialization and be worn for at least 12 hours a day for 1 month.⁹⁴

Intralesional steroid injection is a common method of treatment for keloids. Steroid injection may help to alleviate some of the pain and pruritis associated with these lesions as well as soften them. The mechanism, like most other therapies targeted at management of fibroproliferative disease, is decrease of production of collagen through fibroblast inhibition. Triamcinolone acetonide (Kenalog; Bristol-Myers Squibb, Princeton, NJ) is often used and injected at a concentration of 40mg/ml.⁹⁵ Intralesional steroid injection is most often used as an adjunct to excision or debulking of the lesion. Side-effects of this treatment include pain from injection, hypo- or hyperpigmentation, skin necrosis and skin atrophy, as well as systemic effects of steroid absorption.

Electron beam radiation after surgical excision of keloids has been used to prevent recurrence; however, irradiation carries the risk of inducing malignant tumors and increasing pigmentation of the lesion, and the method lacks standardization. That is, optimal doses to prevent recurrence vary by site and a standard protocol has yet to be determined. When compared to intralesional steroid injection following keloid excision in a randomized controlled trial, radiotherapy showed better success in the form of decreased recurrence rate and better patient compliance.⁹⁶

Some research has suggested that intralesional injection of interferon- α 2b into hypertrophic scars improves scar quality which is due to a resultant decrease in number and function of fibrocytes.⁹⁷ However, as a method to treat keloids, interferon- α 2b has been less successful. Randomized controlled clinical trials failed to show the benefit of interferon- α 2b either as an adjunct to surgical excision or as a stand-alone therapy.^{98,99}

The morbidity associated with keloids and hypertrophic scars can be significant and range from poor cosmesis to severe pain and pruritis. An emphasis of research on prevention, in addition to treatment, has occurred in an effort to eliminate some of the potentially devastating effects of fibroproliferation disease. Prevention of fibroproliferation disease is the subject of both *in vitro* and *in vivo* studies. The local application or injection of basic fibroblast growth factor (bFGF) immediately after closure of surgical incisions has been shown to reduce scar formation.¹⁰⁰ Fibroblast growth factor has also shown promise in decreasing healing time and promotion of better quality of healing in chronic traumatic wounds.^{101,102} The mechanism by which bFGF promotes healing with a decrease in scar formation is not entirely understood; however, one theory is based on the fact that bFGF is a potent angiogenesis inducer.¹⁰⁰

Other modalities to decrease keloid recurrence and lessen hypertrophic scar symptoms include cryotherapy, laser therapy, 5-fluorouracil (5-FU) or imiquimod application, and intralesional bleomycin.

The limitations of use for these methods vary from hypo- to hyperpigmentation, pain and burning from injection or application, high recurrence rates, and unknown efficacy due to lack of research. Until the pathophysiology of fibroproliferative disease is better understood and a specific therapy developed, combination therapy may be the most effective treatment of keloids and hypertrophic scars.

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Chapter 45

Cleft Lip and Palate: An Overview

Radhika Chigurupati, Andrew Heggie, and Krishnamurthy Bonanthaya

This chapter is a comprehensive overview on management of cleft lip and palate deformities. Epidemiology, etiology, embryology and genetics are discussed in the first half of this chapter. The importance of interdisciplinary team care is emphasized, and the sequence of treatment from birth to adulthood is presented. The surgical principles of correction of both primary and secondary deformities of cleft lip and palate are discussed in the second half of this chapter.

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Global burden of birth defects: cleft lip and palate

More than four million children are born with birth defects worldwide every year. Craniofacial anomalies comprise a large fraction of all human birth defects, less frequent only than congenital heart disorders and clubfoot. Cleft lip with or without palate (CL/P) is the most common craniofacial birth defect with an estimated quarter of a million affected babies born each year in the world. This malformation shows considerable variation across geographic regions and ethnic groups and has significant medical, psychological, social, and economic ramifications.¹ It is a costly public health problem with an average lifetime treatment cost per child in the US estimated to be roughly \$101 000.²

The World Health Organization (WHO) and most cleft organizations across the globe recommend interdisciplinary care by a team of specialists. In reality, however, surgical and non-surgical treatment is often fragmented and dictated by socio-economic factors and access to medical facilities. In developing countries, particularly in rural areas, care is often neglected due to social beliefs and lack of awareness, or initiated late due to restricted resources and inadequate access. The delay in treatment and intermittent care by local or overseas cleft mission surgeons, combined with incomplete follow-up, results in poor outcomes with unnecessary complications. More recently, some humanitarian non-profit organizations supporting cleft care have changed their aid philosophy. They are identifying centers with a potential to deliver quality care in low- and middle-income countries and

providing support for the physicians, staff, and hospital infrastructure, helping to establish parameters of care, as well as monitoring treatment outcomes at these centers.³

Birth defects are emerging as a cause of neonatal mortality in countries that have made progress in controlling infectious diseases and malnutrition. The strategies proposed to reduce the global impact of birth defects include: (1) effective family planning, genetic counseling, and prenatal diagnosis; (2) education for couples to decrease maternal exposure to avoidable environmental risk factors such as tobacco, alcohol, and teratogenic medications; (3) improving periconception maternal intake of micronutrients such as folic acid (400µg); and (4) improving the availability of medical and surgical care locally for the affected infants. National leadership and commitment are essential for proper surveillance of birth defects, infant mortality, and to monitor the clinical and cost effectiveness of various interventions.^{4,5}

The WHO initiative for collaborative craniofacial anomalies research has identified areas of uncertainty in clinical care and efforts are being made to conduct trials with sufficiently large samples of patients to provide evidence for treatment strategies. Initial research efforts have focused on addressing surgical, anesthetic, and nursing care for patients with craniofacial anomalies in developing countries. Surgical techniques for repair of various cleft sub-types and correction of velopharyngeal insufficiency are being evaluated. Adjunctive services such as use of prophylactic ventilation tubes, presurgical orthopedics, psychological counseling, speech therapy, and feeding interventions before and after surgery are also being monitored and assessed. An international database of craniofacial anomalies has been established to improve answers to questions relevant to individuals with cleft and craniofacial anomalies, their families, and health care providers.⁶

Epidemiology

Cleft lip with or without palate (CL/P) has an average birth prevalence of 1:700 ranging from 1:500 to 1:2000, depending on the race (Table 45.1). There are wide ethnic variations with highest occurrence in Native Americans (3.6:1000), followed by Asians (2.1:1000 Japanese births and 1.7:1000 Chinese births), Caucasians (1:1000), and lowest in those of African descent (0.3:1000). Cleft of palate only (CP), which differs genetically from CL/P, has a birth prevalence rate of 1:2000 and is more similar across all populations.⁷⁻¹³ About half of the oral clefts involve lip and palate (46%), a third of the clefts involve only the palate (33%), and clefts of lip alone account for 21%. CL/P is more often unilateral than bilateral and more common in males than females. The unilateral defects occur more often on the left side than the right side. Clefts of lip occur in the ratio of 6:3:1 for unilateral

Table 45.1 Epidemiology of oral clefts.

Distribution of oral clefts
Cleft lip and palate 46%
Cleft palate only 33%
Cleft lip only 21%
Cleft lip with or without palate
Average birth prevalence 1:700
More common in males
Unilateral > bilateral
Left side > right side
Association with other anomalies 10%
Cleft palate only
Average birth prevalence 1:2000
More common in females
Association with other anomalies 50–60%

left, unilateral right, and bilateral.¹⁴ CP is more common in females and more often associated with other developmental anomalies.

Clefts are referred to as non-syndromic and syndromic, based on their association with other anomalies. About 50% of CP and 10% of CL/P are associated with a syndrome.¹⁵⁻¹⁷ Some common syndromes associated with cleft lip and palate include Van der Woude, Treacher Collins syndrome, Down syndrome, oro-facial digital syndrome, Opitz syndrome, craniofacial microsomia, and fetal alcohol syndrome.¹⁸ Nearly half of the syndromic cleft palate presentations are associated with the triad of micrognathia, glossoptosis, and airway obstruction (Pierre Robin sequence). The most common syndromic presentations of this triad are Stickler's syndrome, accounting for 25%, and velo-cardio-facial (VCF) syndrome, accounting for 15% of all syndromic cleft palate individuals.¹⁹

Etiology and genetics

Non-syndromic CL/P is a complex trait with multifactorial etiology, resulting from gene–gene and gene–environmental interactions. Identification of key genes contributing to the genesis of orofacial clefts will help in early diagnosis, disease prevention, or possibly developing adjunctive therapies. The most recent estimates suggest that anywhere from 3–14 genes contribute to cleft lip and palate.^{20,21} Candidate genes and loci responsible for non-syndromic CL/P have been identified on chromosomes 1, 2, 4, 6, 11, 14, 17, and 19.²²⁻²⁴ Two genes IRF6 and MSX-1 now seem to explain about 15% of non-syndromic CL/P. Mutations in IRF6 lead to Van der Woude and popliteal pterygium syndromes. Mutations in other genes, TBX22, FGFR1, and P63, also contribute to syndromic clefts.^{25,26} Aberrant transforming growth factor beta-3 (TGF-β3) signaling plays a role in the pathogenesis of cleft palate.

Environmental factors that contribute to the etiology of facial clefting disorders include cigarette smoking,^{27–30} folic acid deficiency during the periconceptional period,^{31,32} and maternal exposure to alcohol and teratogenic medications such as retinoids, corticosteroids, and anticonvulsants (phenytoin and valproic acid).²³ Co-sanguineous marriages, maternal diabetes, and obesity have also been linked to an increased risk of orofacial clefts. Less consistent associations have been found between clefts and maternal viral infections such as rubella and varicella.^{6,19}

Studies conducted to determine the risk of having a child with CL/P show that every parent has about a 0.14% (1:700) chance of having a child with a cleft. The risk of recurrence of a cleft condition is determined by a number of factors, including the number of family members with clefts, their relationship to family members with clefts, race and sex of the affect-

ed individuals, and the type of cleft. Studies show that the recurrence risk for first-degree relatives is about 3.3% for CL/P and for isolated CP it is 2%.³³ Once parents have a child with a cleft the risk of having a second child with a cleft is about 2–5%, and after two affected children that risk rises to 9–12%.^{34,35} In twins with CL/P and those with isolated CP, the concordance is far greater for monozygotic twins than for dizygotic twins.¹⁹ Parents and young adults should be counseled appropriately by a geneticist so that they are in a better position to make decisions about future pregnancies.

Embryology

The embryologic development of the face begins at 4 weeks after conception from the neural crest ectomesenchyme that forms five prominences; the fronto-nasal process, and paired maxillary and mandibular processes surrounding a central depression. During the fifth and sixth weeks of embryonic development, bilateral maxillary processes derived from first brachial arch fuse with the medial nasal process to form the upper lip, alveolus, and the primary palate (Fig. 45.1). The lateral nasal process forms the alar structures of the nose. The lower lip and jaw are formed by the mandibular processes.

This process of formation of the face is the consequence of a cascade of processes that involve cell proliferation, cell differentiation, cell adhesion, and apoptosis. Failure or error in any of these cellular processes that lead to fusion of the medial nasal process with the lateral nasal and maxillary process can cause orofacial clefts (Fig. 45.2). The molecular events that underlie these cellular processes are under the control of a strict array of genes that include fibroblast growth factors (FGFs), sonic hedgehog (SHH),

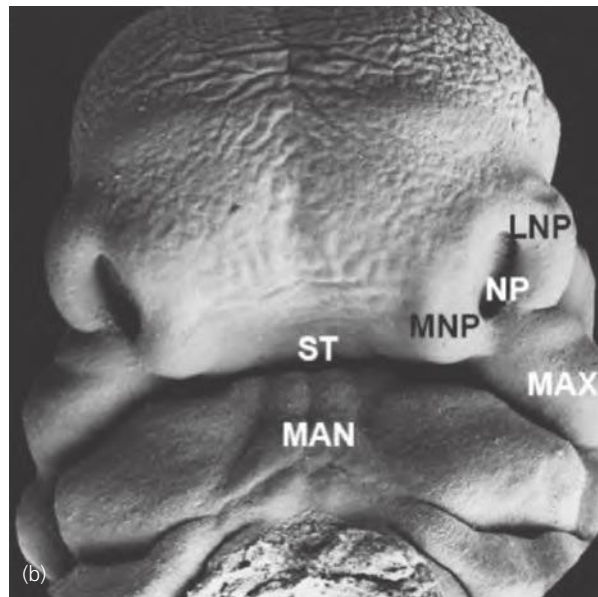
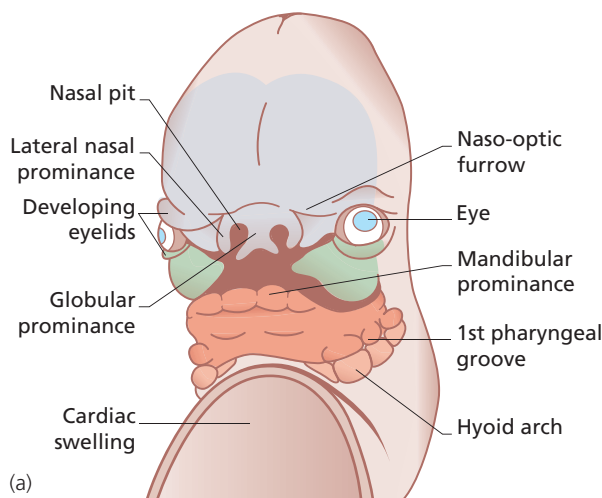


Fig. 45.1 (a) Schematic diagram showing embryonic development of face at 6 weeks. (b) Electron microscopy showing the development of face of a 37-day-old human embryo. The nasal pit (NP) is surrounded by the medial nasal process (MNP) and lateral nasal process (LNP) and maxillary process (MAX). (Adapted and redrawn from Sperber, GH. *Craniofacial Development*. Hamilton: BC Decker Inc, 2001. Reproduced with permission.)

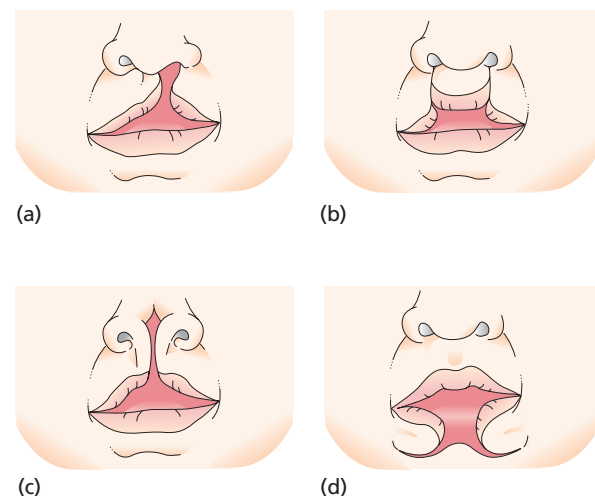


Fig. 45.2 Orofacial clefts resulting from errors during embryonic development of face. (a) Unilateral cleft of upper lip. (b) Bilateral cleft of the upper lip. (c) Midline cleft of the upper lip and nose. (d) Median mandibular cleft. (Adapted and redrawn from Sperber, GH. *Craniofacial Development*. Hamilton: BC Decker Inc, 2001. Reproduced with permission.)

bone morphogenic proteins (BMPs), and members of the transforming growth factor beta (TGF- β) superfamily and other transcription factors.¹⁰ The formation of the secondary palate begins during the sixth week after conception from the two palatal shelves, which extend from the internal aspect of the maxillary processes. During the eighth week, these bilateral maxillary palatal shelves after ascending to an appropriate position above the tongue, fuse with each other and the primary palate. A disruption in the fusion of these embryonic components can occur due to delay in elevation of the palatal shelves from vertical to horizontal, defective shelf fusion, or post-fusion rupture resulting in a cleft of the secondary palate (Fig. 45.3).³⁶

Classification

In order to standardize documentation and communicate effectively, various types of classification systems have been described. The early Veau classification included groups 1–4 with increasing severity of clefting:

- group 1 – cleft of the soft palate;
- group 2 – cleft of the hard and soft palate up to incisive foramen;
- group 3 – complete unilateral cleft lip and palate;
- group 4 – complete bilateral cleft lip and palate.

However, this classification is not always adequate to document the variations. The more sophisticated schematic diagrams, such as the one described by Kernahan and Stark have been used recently (Fig. 45.4).³⁷ Berkowitz used a simple classification for labio-palatine clefts:

1. Clefts of lip and alveolus.
2. Clefts of primary (including lip) and secondary palate.
3. Clefts of secondary palate only.
4. Submucous cleft.

Interdisciplinary management of the cleft individual

The idea of interdisciplinary care is to coordinate treatment by multiple specialists in a timely fashion with an aim of achieving normality in all aspects, including feeding, breathing, speech, hearing, alignment of teeth, appearance, and overall psychological and physical development. The timing of surgical and non-surgical interventions should coincide with the physical, cognitive, and social development of the child (Table 45.2). Cleft teams generally include a craniomaxillofacial surgeon, pediatrician, nurse practitioner, speech pathologist, orthodontist, social worker, and geneticist. Experience in Scandinavian countries and a multicenter Euro-cleft study have

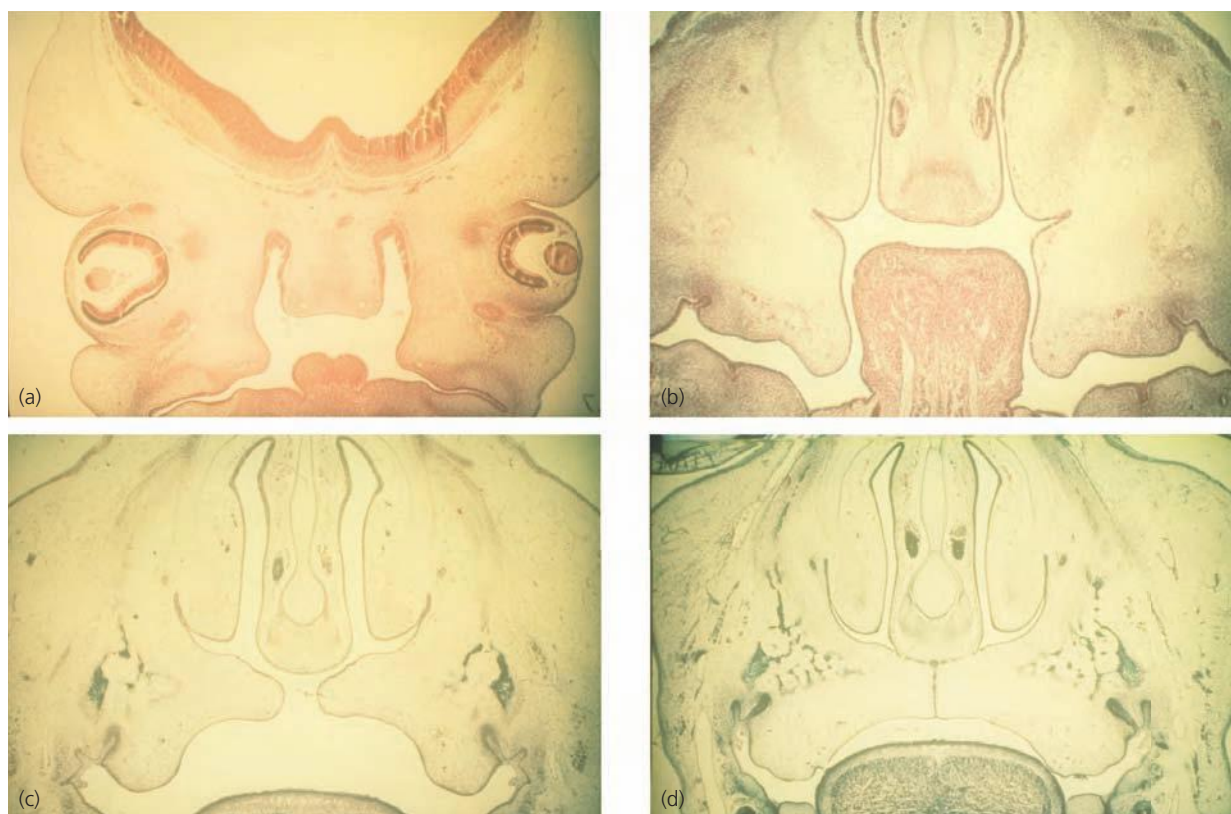


Fig. 45.3 Coronal scanning electron microscopy photos of human embryo showing the stages of formation of the palate at 8–9 weeks. (a) Development of palate showing palatal shelves and tongue position. (b) The vertical orientation of the palatal shelves on either side of tongue. (c) Palatal shelves elevate and (d) fuse with each other and the nasal septum in the midline.

demonstrated that standardization, centralization, and participation of surgeons who perform a large number of cleft procedures produce better surgical results in terms of speech, appearance, and facial growth.³⁸ However, this cannot be applied to all countries, particularly those with a high volume of cleft individuals and a limited number of care facilities.

Prenatal diagnosis

Interdisciplinary team care begins with prenatal diagnosis and parental counseling. With the advent of sophisticated high resolution three-dimensional (3D) ultrasonography and genetic tests for screening of birth defects, intrauterine diagnosis of cleft lip is possible. Early diagnosis of a cleft of the lip should alert the obstetrician to the possibility of other malformations that may require further investigations. While early diagnosis may help parents to be better prepared, the advent of such a capability raises both ethical and psychological issues, such as dilemma of termination of birth. Physicians and surgeons have to inform parents that CLP in the absence of other major systemic anomalies is a treatable non-life-

threatening condition. The cleft team also can discuss feeding issues and timing of lip and palate surgery, and can help establish contact with support groups for the family.³⁹

Transvaginal ultrasonography may reveal a cleft of lip as early as 11 weeks whereas 16–20 weeks is ideal for transabdominal ultrasonography. Several factors may influence the accuracy of ultrasound studies: sophistication of the scanning equipment; experience and skill of the sonographer; number of weeks into pregnancy; position of the baby while scanning; amount of amniotic fluid; maternal body structure; and severity of cleft.

Premaxillary protrusion is an important clue to the presence of cleft lip and cleft palate and may be more conspicuous than the cleft itself. The presence of a paranasal echogenic mass favors the presence of bilateral cleft lip and cleft palate. Clefts of palate alone are rarely visualized on ultrasound.⁴⁰ The majority of the cases of orofacial clefts are detected antenatally. A recent study by Johnson *et al.* showed that the frequencies of prenatal diagnosis for cleft lip and palate, cleft lip only, and CP only were 33.3%, 20.3%, and 0.3%, respectively.⁴¹ Although the benefits of fetal healing have been well documented, at this time there is no indication for intrauterine repair of the cleft lip deformity as the risk of fetal surgery is far too

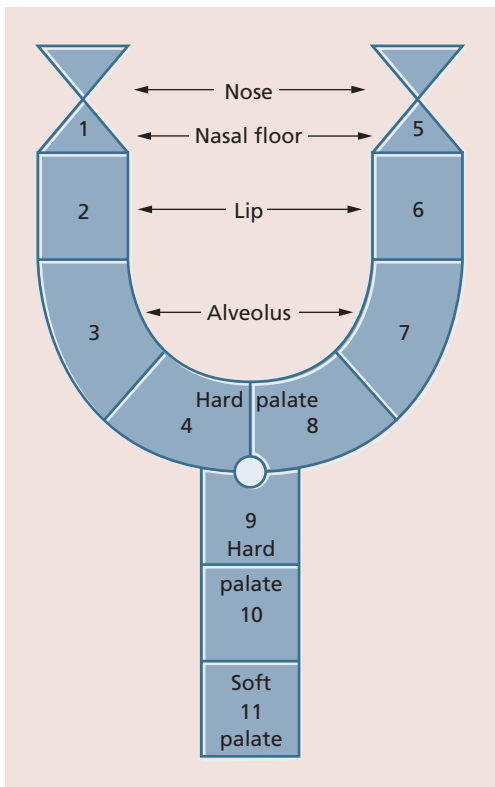


Fig. 45.4 Kernahan and Stark classification of clefts. Kernahan’s striped-Y classification: areas 1 and 5 represent the right and left sides of nasal floor, 2 and 6 represent the right and left sides of the lip respectively. The alveolus is represented by areas 3 and 7, the hard palate anterior to the incisive foramen by areas 4 and 8, the hard palate posterior to the incisive foramen by areas 9 and 10, and the soft palate by area 11. (From Millard Jr DR. The unilateral deformity. In: *Cleft Craft: The Evolution of its Surgery*, Vol 1. Boston, MA: Little, Brown, 1977; 52. Reproduced with permission. Copyright © D. Ralph Millard, Jr.)

Table 45.2 Timing of treatment in the cleft patient.

Prenatal
Diagnosis and parental counseling
0–6 months
General assessment for associated anomalies ENT evaluation – breathing, feeding, swallowing, and hearing Presurgical orthopedics (0–3 months) Primary lip repair (3–4 months)
6 months – 2 years
Speech and oral sensory motor assessment Grommets/ear tubes (as needed) Primary palate repair (9–12 months)
Preschool: 3–5 years
Dental care Speech assessment and therapy (continue as needed) Assess need for lip revision
Childhood: 6–12 years
Correction of velo-pharyngeal dysfunction (as needed) Orthodontic treatment – phase I Alveolar cleft repair (8–11 years)
Adolescence: 13–18 years
Orthodontic treatment – phase II Orthognathic surgery (if needed) – 14–16 years (female), 16–18 years (male) Revision chielo-rhinoplasty Replacement of missing teeth (as needed)

high both for the fetus and mother for correction of this non-life-threatening condition.

General assessment

Every child born with a CL/P should be thoroughly assessed by complete physical examination and necessary diagnostic tests to check for associated systemic abnormalities, including congenital heart, renal, or airway anomalies. If the child is delivered in a non-medical facility or a small hospital they should be referred to a tertiary hospital with specialists or a craniofacial team for further evaluation. A proper airway assessment, and counseling for nutrition and feeding should be initiated immediately.

Feeding and nutrition

Feeding is one of the first concerns in a child born with a CL/P. Parents should be taught how to feed the baby and informed about various feeding nipples that can deliver more milk under less pressure. The goal is to provide adequate nutrition to satisfy the caloric requirements and avoid failure to thrive. The team nurse generally provides the parents with information on feeding before birth or immediately after birth. Children with cleft lip only without a cleft palate may have some difficulty in creating a seal around the nipple but generally can be breast fed before and after lip surgery. However, the presence of a cleft palate makes it difficult to create a negative pressure that is necessary to feed. Four randomized clinical trials conducted with a total of 232 babies showed that squeezable bottles may be easier to use than rigid bottles for children with CL/P. There was no statistical difference found in growth outcomes of children with CL/P who were fitted with a passive palatal appliance to help with feeding and those without an appliance.^{42,43} A feeding tube is rarely necessary except in infants with other associated anomalies, particularly airway anomalies.

Ear, nose, and throat evaluation

A proper airway assessment should be priority for a newborn with congenital craniofacial anomalies. Infants are obligate nasal breathers. It is important to check if there is obstruction at any level in the upper or lower airway, including the nares and choanae. Children born with a cleft of the palate may have associated micrognathia, glossoptosis, and airway obstruction (Fig. 45.5). In these children, one should look for signs of increased effort while breathing, stridor, weight loss, and failure to thrive. Parents should be informed to watch for an abnormal breathing pattern or respiratory distress, particularly during upper respiratory tract infection. If there are signs of airway obstruction, a pediatric otolaryngologist should be consulted to perform an endoscopic evalu-

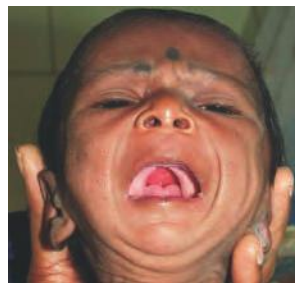


Fig. 45.5 Child with Pierre Robin sequence (PRS) showing a wide cleft of palate, small lower jaw, and a retropositioned tongue in the cleft obstructing the airway.

ation of upper and lower airway to look for possible cause of obstruction.

An audiology assessment is recommended soon after birth to check for hearing abnormalities. Children with cleft palate exhibit a higher frequency of otitis media prior to palate repair than those without clefts. Middle ear ventilation disorders due to eustachian tube dysfunction can cause conductive hearing loss. This can also contribute to speech and language delay in these children. Although not as common as conductive hearing loss, sensorineural hearing deficits exist within the cleft population, and it has an effect on speech perception and clarity, as well as auditory comprehension skills.^{44,45} Early speech and language stimulation and an initial speech evaluation no later than 6 months after birth are recommended for children with clefts of palate.

Presurgical orthopedics

The benefits of presurgical orthopedics include better alignment of the alveolar segments and premaxilla, tension-free approximation of the cleft lip edges, and improvement of nostril symmetry and shape. Presurgical orthopedics was introduced in the management of clefts by McNeill and Burston. They initiated the use of a palatal acrylic plate in order to bring the collapsed maxillary alveolar segments into proper alignment prior to lip surgery. Latham described use of an active expansion device to align the collapsed lateral maxillary segments and retract the premaxilla in complete bilateral clefts, and to achieve symmetry of the alveolar arch in complete unilateral clefts.^{46,47}

Grayson and others have shown that gentle application of presurgical orthopedic forces to mold the alveolar segments and the nostrils within 0–3 months of birth has shown some benefits in correction of the nasal deformity in children with complete bilateral CLP and wide unilateral clefts. Nasoalveolar molding increases the surface area of the nasal mucosal lining, and also helps with elongation of the columella and making the columella upright. This preoperative expansion of the nasal lining allows suturing of interdomal cartilages without tension and decreases widening of the nose (Fig. 45.6).^{48–51}

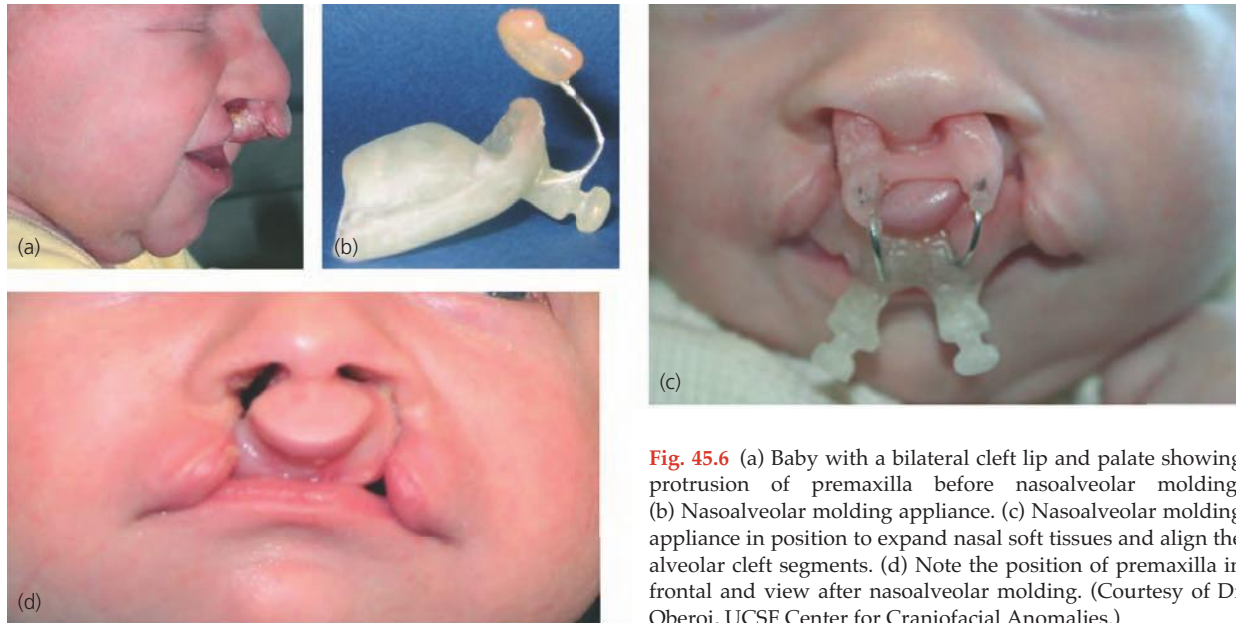


Fig. 45.6 (a) Baby with a bilateral cleft lip and palate showing protrusion of premaxilla before nasoalveolar molding. (b) Nasoalveolar molding appliance. (c) Nasoalveolar molding appliance in position to expand nasal soft tissues and align the alveolar cleft segments. (d) Note the position of premaxilla in frontal view after nasoalveolar molding. (Courtesy of Dr Oberoi, UCSF Center for Craniofacial Anomalies.)

There is a wide variation in the availability of expertise and cost of treatment when it comes to infant presurgical orthopedics or nasoalveolar molding. In recent years, several centers have reported the adoption of this technique to improve the outcomes of lip and nose repair, especially in complete bilateral CLP.^{52,53} It is important to evaluate these recent studies critically for their overall clinical and cost effectiveness. Non-surgical lip adhesion with tape is a cost-effective and simple technique that can bring the alveolar segments closer to facilitate cheiloplasty in infants with wide clefts of the lip and palate (Fig. 45.7).

Cleft lip repair

The goal of primary lip repair is to reconstruct a functional lip with minimal scarring and normal appearance. The timing for primary lip repair is usually between 3 and 6 months after birth. Most craniofacial centers follow the rule of ten's to ensure that the

infant is fit for the surgical procedure. This rule implies that the infant should be at least 10 weeks of age, weigh at least 10 lbs, and have a hemoglobin level of at least 10 g/100 ml. Some centers have reported lip repair in children with lower hemoglobin levels (8 g/100 ml) with no deleterious effects. In low- and middle-income countries, infants are often malnourished and proper feeding and nutrition counseling is essential to prepare them for lip surgery by 3 months.⁵⁴ Lip repair is performed under general anesthesia with an Oral RAE[®] endotracheal tube taped to the midline of lower lip without distorting the commissure. Postoperative care includes keeping the wound clean by preventing crusting and using antibiotic cream; in some centers arm restraints are also used for 7–10 days.

A surgical lip adhesion may be preferred as an initial surgical procedure within 6–8 weeks after birth in some centers. Lip adhesion helps to align the maxillary alveolar segments and achieve a tension-free definitive lip repair at a later date. Good approximation of the alveolar segments also allows the surgeon

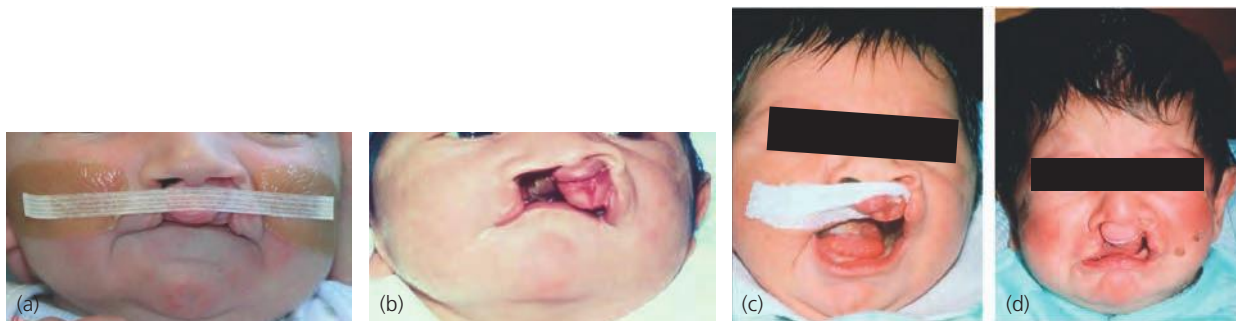


Fig. 45.7 (a–d) These photos illustrate that simple presurgical orthopedic techniques, such as lip taping, can be used to align the cleft alveolar segments prior to lip repair.

to perform a gingivoperiosteoplasty at the time of definitive cheiloplasty. The disadvantages of converting the complete cleft lip to an incomplete one by lip adhesion are: the need for an extra operation and the possibility of excising more tissue at the time of definitive lip repair. Non-surgical orthopedic techniques during the first 6–8 weeks after birth, as described earlier in this chapter, can produce good alignment of the alveolar segments.

Unilateral cleft lip

Surgical anatomy

Unilateral cleft lip is an asymmetric deformity that presents with a multitude of inherent anatomic variations (Fig. 45.8). The most visible anatomic abnormalities of the complete unilateral cleft lip and nose deformity are due to the abnormal position of the orbicularis oris muscle. After fetal dissections, Fara noted that in a complete cleft lip, the fibers of the orbicularis oris muscle instead of proceeding horizontally from the commissure towards the midline turn upward along the margins of the cleft. The fibers of the orbicularis terminate medially beneath the base of the columella and laterally beneath the alar base and periosteum of the piriform rim.⁵⁵ It is these abnormal muscle attachments and pull, that cause the typical bulge on the unrepaired cleft lip, distortion of the ala of the nose, and deflection of anterior nasal spine and septum of the nose. The nasal deformity is proportionate to the severity of clefting. In the complete unilateral cleft lip and palate deformity, there is slumping of the alar cartilage on the cleft side resulting in an asymmetric nasal tip. The alar base is displaced laterally, inferiorly, and posteriorly leading to a widened nasal aperture. There is shortening of the medial crus of the alar cartilage and lack of over-

lap of upper and lower lateral cartilages. The columella and the caudal edge of the septum and anterior nasal spine are deviated to the non-cleft side.⁵⁶

Evolution of unilateral cleft lip repair

Several surgeons, including Rose (1891), Thompson (1912), Blair (1930), Le Mesurier (1949), Tennison and Randall (1952), and Skoog (1974), have contributed to the evolution of cleft lip repair, but the most popular technique was introduced by Millard (1955) who described the rotation–advancement concept. Today various modifications of the rotation–advancement technique by Millard are used to repair the unilateral cleft lip deformity.⁵⁷ In Millard's technique the medial flap is rotated downward to achieve length, while the lateral flap is advanced (Fig. 45.9). It is an extremely versatile procedure that the surgeon can modify or adjust while operating. The advantage of this technique is that the suture line lies on the recreated philtral column and incision allows easy access for primary rhinoplasty to reposition the nasal septum, lower lateral cartilage, and alar base. The main disadvantage is that the inexperienced surgeon requires good surgical judgment during the operation as it is not based on exact measurements. The triangular flap technique, described by Tennison and Randall, is based on exact measurements, can be reproduced well, and used more easily in wide clefts of the lip.⁵⁸

Principles of repair of unilateral cleft lip and nose

An adequate repair of the unilateral lip deformity should correct the alignment of the orbicularis oris muscle, and create a cupid's bow and philtral column on the affected side. In the unilateral defect the normal side can be used as a guide to identify the key

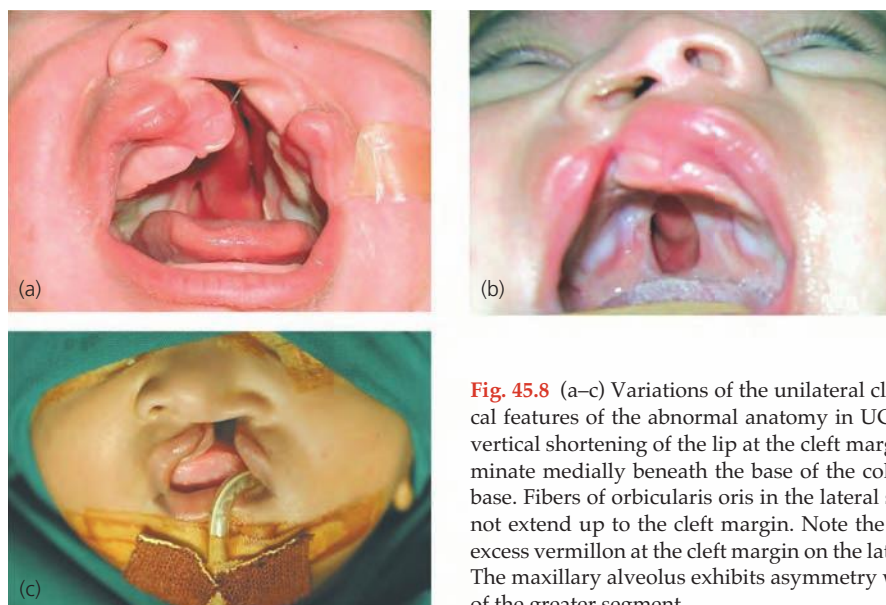


Fig. 45.8 (a–c) Variations of the unilateral cleft lip and palate deformity. The typical features of the abnormal anatomy in UCLP are seen in these photos. There is vertical shortening of the lip at the cleft margins, orbicularis oris muscle fibers terminate medially beneath the base of the columella and laterally beneath the alar base. Fibers of orbicularis oris in the lateral segment are more hypoplastic and do not extend up to the cleft margin. Note the discrepancy of vermillion width with excess vermillion at the cleft margin on the lateral side compared to the medial side. The maxillary alveolus exhibits asymmetry with an upward and outward rotation of the greater segment.

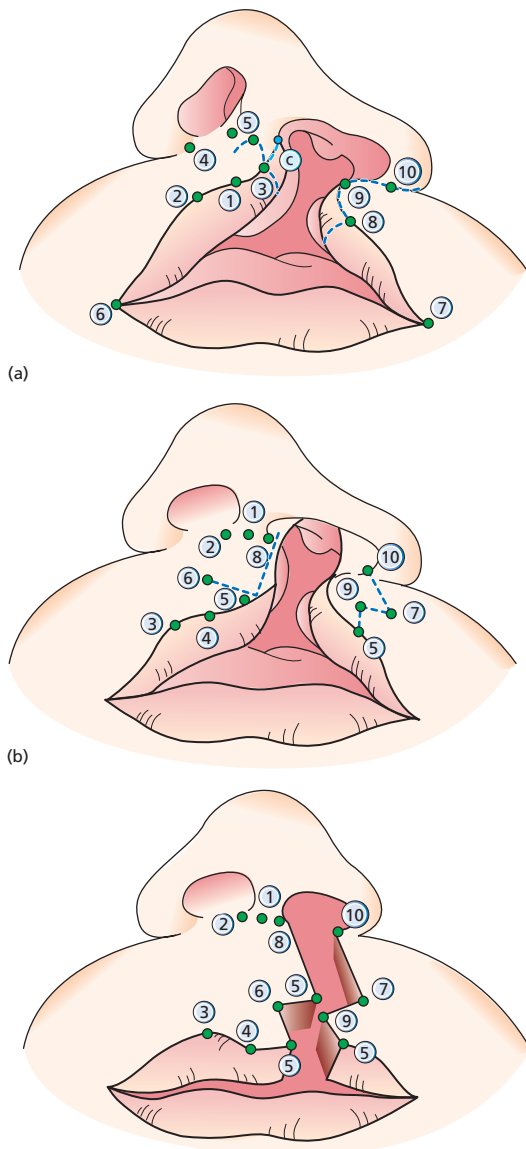


Fig. 45.9 (a) Markings of the key points and incisions for repair of the unilateral cleft lip using the Millard rotation-advancement principle: lengthening the shortened vertical height of the lip on the medial side to match the lateral, and advancing the flap of tissue from lateral to medial. (b) Markings of the key points and incisions for repair of the unilateral cleft lip using the triangular flap technique.

points and to plan the incisions on the cleft side. Despite inherent variations there are some similarities that form the basis of the guiding principles in surgical repair of this deformity (Fig. 45.10).⁵⁷

1. *Rotation or lengthening of shortened vertical height of lip.* The difference in the vertical length of the lip from the height of the cupid's bow to the base of the columella between the non-cleft side and the cleft side indicates the amount of rotation and back cut necessary. The medial lip element (non-cleft side) is rotated inferiorly to achieve adequate length and symmetry of the cupid's bow.
2. *Advancement of flap of tissue from lateral to medial.* The flap of tissue from lateral should be advanced

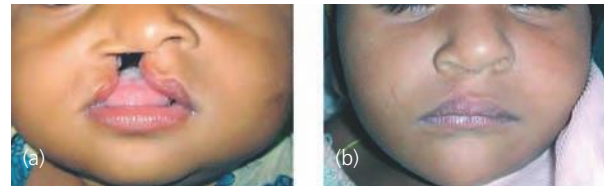


Fig. 45.10 Unilateral cleft lip repair. (a) Preoperative and (b) postoperative views of lip repair performed by a modified Millard technique.

in to the lip on the medial segment. It is important to release the abnormally inserted paranasal and facial muscles at the alar base by subperiosteal dissection in order to approximate the edges without tension.

3. *Retaining cupid's bow and creating a philtral column.* Approximation of the cleft edges should be achieved without loss of natural landmarks: the cupid's bow, philtral dimple, and philtral column. The scar of union of cleft edges should be placed along a natural line – the philtral column. Creating a good philtral column requires proper approximation of the muscle and tension-free closure of the overlying skin.
4. *Muscle reconstruction.* The muscle bellies should be dissected within the skin and mucosal envelope. The muscles should be approximated with interrupted mattress sutures. The muscles at the base of the columella and ala should be dissected and approximated to help reposition the distorted nasal structures.
5. *Restoration of the alveolar continuity.* If the alveolar segments are closely approximated a gingivoperiosteoplasty can be performed at the time of primary lip repair to achieve a continuous alveolar arch.
6. *Primary repair of the distorted nasal anatomy.* This requires wide undermining of the skin drape over the lateral crura of the alar cartilages and repositioning of the nasal septum with a suture (Fig. 45.11). Long-term results of primary rhinoplasty by McComb and others show that the outcomes are better and there is no decrease in overall size or nasal growth inhibition after primary correction.^{59–61}

Bilateral cleft lip

Surgical anatomy

Bilateral cleft lip repair is much more challenging and the results are often less satisfactory than those of unilateral cleft lip. Complete bilateral clefts of lip are rare, accounting for only 10% of cleft lips and therefore the experience in treating these deformities is limited. The typical anatomical abnormalities that make the bilateral cleft lip deformity so difficult to repair are the absence of muscle in the prolabial segment, resulting in lack of philtral dimple, philtral

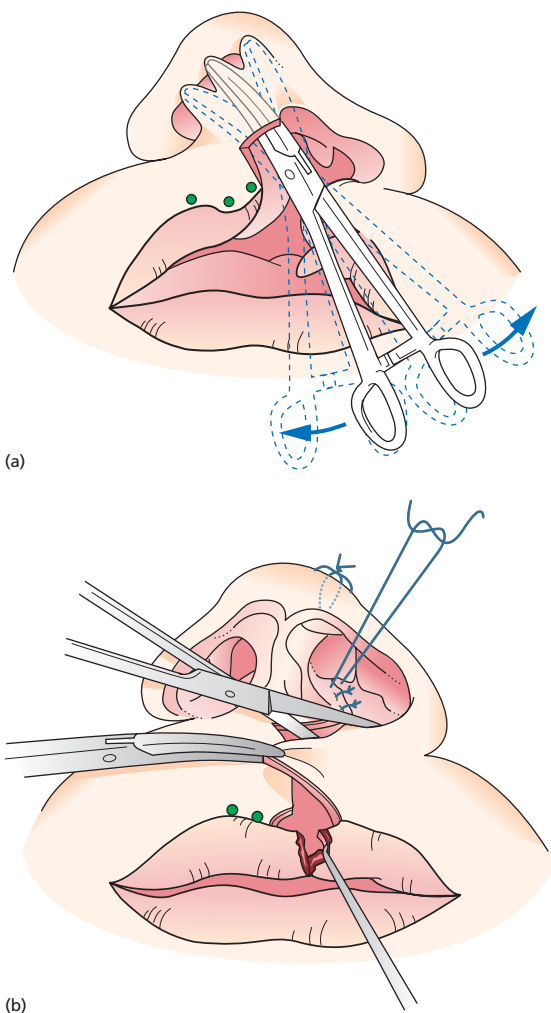


Fig. 45.11 These figures show primary nasal deformity correction in the unilateral cleft lip. (a) Wide undermining of the skin drape over the medial and lateral crura of the alar cartilages. (b) Suturing of the alar dome.

columns, white roll margin, and the median tubercle. The prolabium lacks the angular peaks and the typical cupid's bow. The premaxilla is protuberant and sometimes deviated to one side making tension-free approximation of muscle and cleft margins difficult. The orbicularis oris muscle which is in the lateral lip elements inserts at the alar base on each side. The accompanying nasal deformity consists of a columella that is abnormally short, a wide nasal tip, and a flared alar base due to the malpositioned, splayed alar cartilages. The variations of bilateral CLP deformity are shown in Fig. 45.12.

Evolution of bilateral cleft lip repair

The evolution of repair of the bilateral cleft lip over the years has shown that it is best to repair both sides of the cleft lip at the same time. In many centers today, primary nasal correction is performed along with the repair of the lip. The observation that the alar cartilages are splayed and rotated caudally was important in the evolution of primary rhinoplasty in cleft lip repair. The fear of growth interference when

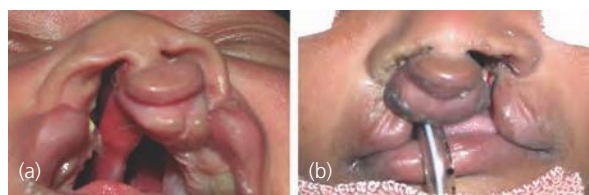


Fig. 45.12 (a, b) Variations of the bilateral cleft lip and palate deformity. These photos show the typical anatomical abnormalities seen in individuals with complete bilateral clefts of lip and palate: lack of muscle in the prolabium, lack of the angular peaks and typical dip of cupid's bow, lack of a definite philtral ridge or dimple in the prolabium, abnormally short and wide columella, lack of adequate labial sulcus depth in the premaxillary area, and flat nasal tip and flared alar base.

nasal cartilage is manipulated during primary repair has been one of the reasons for delaying nasal repair. For many years forked flaps were used to lengthen the columella, but McComb and Mulliken emphasized the importance of early repositioning of the alar cartilages and unraveled the problem of the short columella and broad nasal tip in the bilateral cleft lip. McComb, after reviewing his initial work over a period of 15 years, found that the nostril shape was still abnormal, the tip remained broad, and the columella was very long.⁶² He proposed primary nasal correction by repositioning the alar cartilages through a vertical skin incision.⁶³ Over the years, the techniques that have been used for repair of the bilateral cleft lip and nose deformity include: the straight line closure or veau III operation; Tennison's triangular flap technique (similar to unilateral repair); Millard's technique; Manchester, Skoog, Black, and Mulliken techniques.^{57,64}

Principles of repair of bilateral cleft lip and nose

The basic principles guiding repair of the bilateral cleft lip deformity are: maintaining symmetry; establishing muscle continuity; designing the prolabial flap to achieve appropriate philtral width and shape; forming a cupid's bow and median tubercle from the lateral labial tissue; and finally repositioning the alar cartilages to construct the nasal tip and columella (Fig. 45.13). The principles outlined here are those of the Mulliken simultaneous lip and nose repair.

1. *Establishing symmetry.* Performing both sides simultaneously allows achievement of symmetry.
2. *Designing a prolabial flap of appropriate width.* The design and width of the prolabial flap should be narrow and biconcave to avoid a wide abnormal philtrum as the child grows. This width should be roughly 4mm at the columella base and 6mm at the peak of the cupid's bow. The mucosa of the prolabium can be used either to deepen the sulcus in the premaxillary region or to reconstruct the lip. The author's preference is to use it to deepen the labial sulcus which is usually shallow.

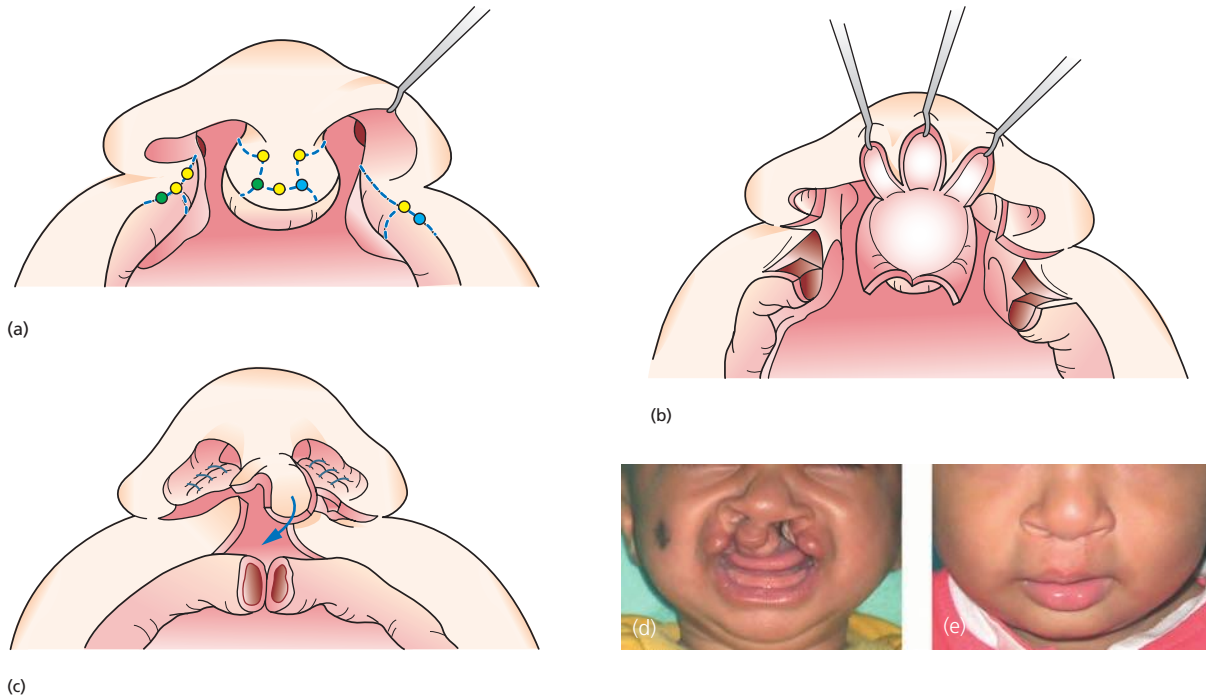


Fig. 45.13 Repair of bilateral cleft lip and palate. (a) Key points to be marked for bilateral cleft lip repair. (b) Designing prolabial flap. (c) Forming cupid's bow and median tubercle from lateral lip elements. (d) Preoperative and (e) postoperative views of a child with bilateral cleft lip repaired by modified Mulliken technique.

3. *Forming the cupid's bow and median tubercle from lateral lip elements.* The prolabium usually has a very narrow strip of vermillion and hence, in the majority of cases, reconstruction of the vermillion in the mid-portion of the lip presents a difficult problem. The incision on the prolabium is made at the mucocutaneous junction and lateral lip vermillion flaps are used to reconstruct the central portion of the vermillion or the tubercle.
4. *Establishing muscle continuity.* In a complete bilateral cleft the prolabium is devoid of muscle fibers. Establishing continuity of muscle beneath the skin of the prolabium is the only way to reconstruct a normal functioning lip. The muscle fibres that are inserted at the alar base on each side are released and reoriented to be approximated in the midline with vertical mattress sutures. This may be difficult, particularly at the upper edge of the lip in wide clefts or a cleft with protruding premaxillae. While a sulcular incision bilaterally beneath the lateral lip elements is essential to mobilize the lip and achieve tension-free closure, the debate has centered on whether the incision should be subperiosteal or supraperiosteal. The author's preference is to perform a subperiosteal dissection following Delaire's principles.^{65,66}
5. *Reconstruction of the nasal tip and columella.* The lower lateral cartilages should be mobilized adequately from the overlying skin by wide dissection and undermining. The splayed cartilage domes can be approached by combining rim incisions with the prolabial flap. The lower lateral cartilages are repositioned anatomically and fixed with interdental mattress sutures to create the tip and columella.
6. *Repositioning the alar base.* The nasal musculature must be mobilized by performing a subperiosteal dissection along the piriform rim and the wide alar base must be cinched with a suture to the base of the anterior nasal spine.
7. *Management of the premaxilla.* A protruding premaxilla in a complete bilateral CLP can impede the approximation of the lip wound edges with a tension-free closure. This can result in a wide scar and a poor cosmetic result. It is therefore important to reposition the premaxilla in the appropriate position with presurgical orthopedics whenever possible.

Repair of cleft palate

The goals of palate repair are to normalize speech by surgical approximation and realignment of the aberrant attachments of the palatal muscles, and to seal the communication between the oral and nasal cavities without fistulae.

Timing of palate repair

The timing of palate repair to achieve optimal speech with minimal facial growth disturbance has been one of the more debated issues in cleft literature. Historically, cleft repair of the hard palate was delayed to minimize impairment of maxillofacial growth. It is now well accepted, and evidence in the

literature shows, that speech outcomes are better when soft and hard palate repair is completed before speech development.⁶⁷ Palate surgery is therefore timed according to the infant's speech developmental stage rather than chronologic age. For most children developing normally, this is around 9–12 months. The majority of the surgeons repair the palate (i.e. hard and soft palate) in one stage before 12 months of age.⁶⁸ Some recommend a two-stage repair with soft palate repair as early as 3–6 months, at the time of primary lip repair, and hard palate by 12–15 months of age.⁶⁹ Children with cleft palate often have other anomalies and it may be necessary to modify the timing of repair in the presence of comorbidities, particularly airway anomalies. Repair of the palate may be delayed up to 14–16 months of age if there are concerns of airway obstruction. Premature babies and infants with micrognathia are particularly at increased risk for postoperative episodes of apnea after palate repair.⁶⁹

Surgical anatomy

Cleft of the palate can range from a minor submucous cleft affecting only the soft palate to a complete bilateral cleft affecting the primary and secondary palate. It is important to look for overt signs of a submucous cleft if there is any suspicion. These signs include a bifid uvula, notching of the posterior nasal spine, or translucency in the mid-palatine region of the soft palate due to lack of muscle.

The muscles of soft palate that help with the function of speech and swallowing include the levator palatini, tensor palatini, palatopharyngeus, palatoglossus, and musculus uvulae. The soft palate in a non-cleft individual acts as a muscular valve that can lift superiorly and posteriorly to appose the pharyngeal wall and achieve velopharyngeal closure during speech. In a child with an unrepaired cleft, the soft palate cannot function as a muscular valve. This is due to the abnormal orientation and attachment of the muscles, primarily the levator palatini. The bundles of the levator on each side are longitudinally directed to insert into the posterior edge of the palatine bone instead of joining in the midline in a transverse orientation and inserting into the palatine aponeurosis (Fig. 45.14).^{70,71} In addition, the sphincter action of the palatoglossus, palatopharyngeus, and superior constrictor muscles at oropharyngeal aperture is compromised leading to velopharyngeal insufficiency. The tensor palatini muscle fibers, which control the opening of the eustachian tube and aerate the middle ear, do not function optimally, often leading to chronic otitis media.

Principles and techniques of palate repair

The main principle of cleft palate repair is to detach and reposition the abnormal insertion of the levator palatini and join the muscles of both halves of the soft palate in the midline at the junction of the middle and posterior third of the soft palate, in order to achieve

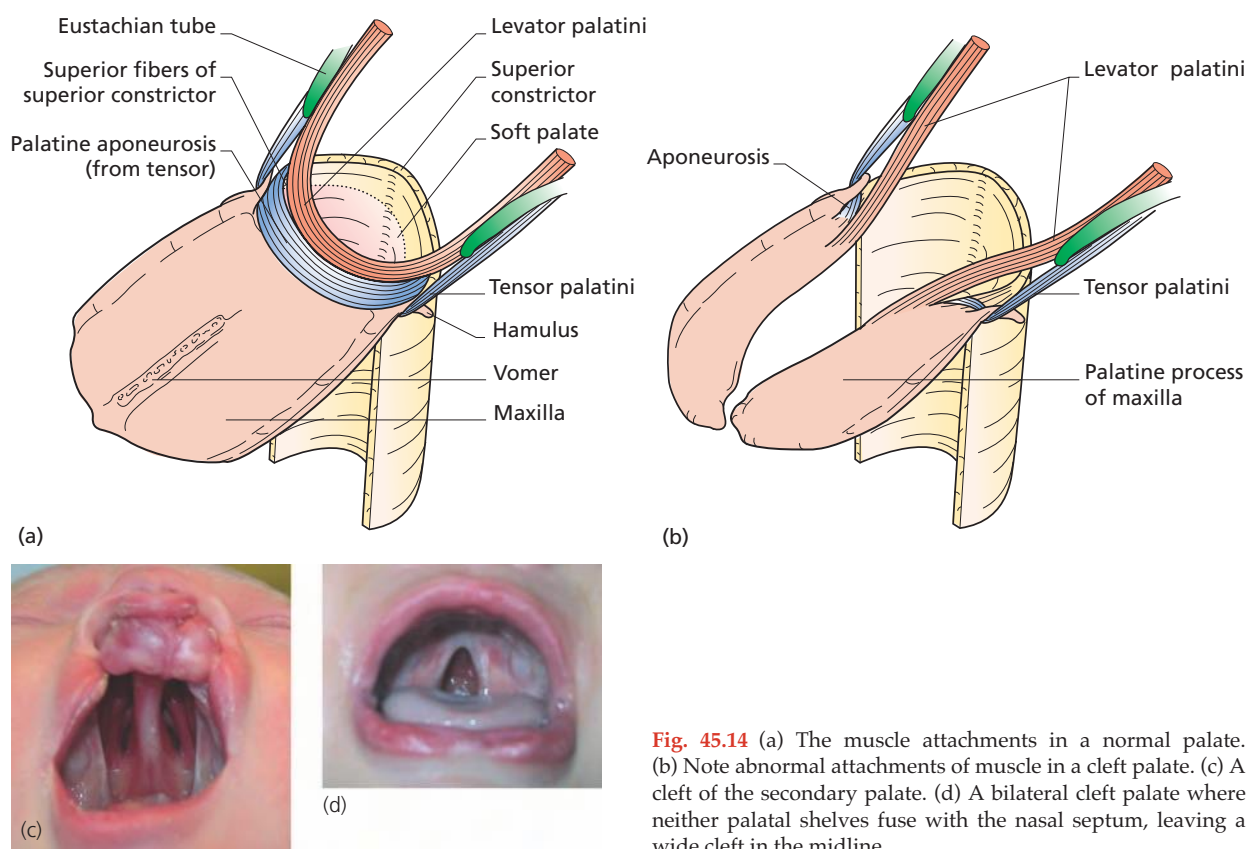


Fig. 45.14 (a) The muscle attachments in a normal palate. (b) Note abnormal attachments of muscle in a cleft palate. (c) A cleft of the secondary palate. (d) A bilateral cleft palate where neither palatal shelves fuse with the nasal septum, leaving a wide cleft in the midline.

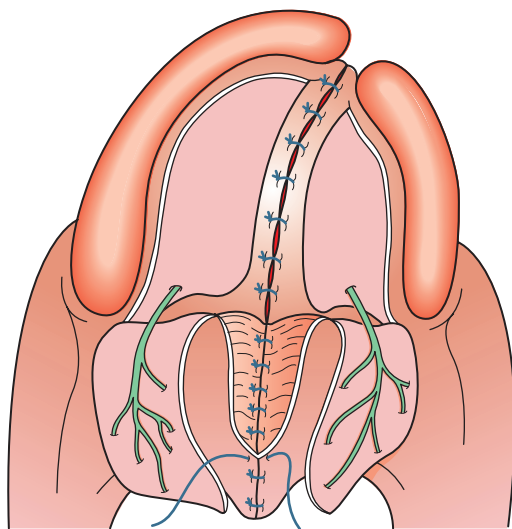
proper elevation of the soft palate.⁷² In the hard palate, the most important principle is to reflect mucoperiosteal flaps based on the greater palatine arteries which emerge from the greater palatine foramen bilaterally at the postero-lateral area of the hard palate.

Cleft palate surgical treatment dates back to the 1760s when a French dentist, Le Monnier, first attempted repair. Several other surgeons, including Philbert Roux, Carl Ferdinand Von Graefe, and Johann Dieffenbach, subsequently described techniques to repair the palate. It was Von Langenbeck, who first described the use of mucoperiosteal flaps for cleft palate surgery.^{73,74} Kriens, in 1969, first introduced the concept of an anatomical approach to veloplasty by restoring the levator sling.⁷² The choice of surgical technique depends on the type of cleft. At the time of the primary palatoplasty the ears should be inspected. If there is evidence of serous otitis, a myringotomy is

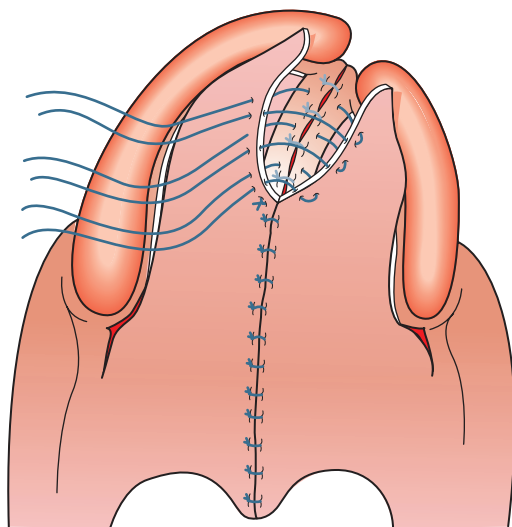
performed and fluid aspirated with placement of grommets or ventilating tubes in the myringotomy incisions.

The two-flap palatoplasty is a commonly used surgical technique for repair of the complete unilateral and bilateral cleft of the palate. The edges of the cleft are incised from the alveolus to the base of the uvula and bilateral full-thickness mucoperiosteal flaps are reflected (Fig. 45.15). The levator palatini muscles are released and dissected to be repositioned horizontally and sutured. Bilateral releasing incisions are made to decrease the tension in the midline.⁷⁵

For the cleft of the secondary palate a Von Langenbeck repair can be used. In this technique bilateral releasing incisions are made and the mucoperiosteum is elevated to complete the stripping and closure of nasal layer, muscle, and oral layers as shown in Fig. 45.16a. The Veau–Wardwill–Kilner (V–Y pushback) technique, named after Thomas Kilner, Victor Veau, and William Wardwill, is used less often for repair of cleft of secondary palate. In this technique the oral mucosa is divided anteriorly, which may lengthen the palate but leaves areas of exposed bone in the anterior hard palate that can potentially cause maxillary growth disturbances. The double reversing Furlow Z-plasty was introduced by Dr Leonard Furlow Jr in 1978.⁷⁶ This technique uses two reversed Z-plasties of the oral and nasal mucosa to repair the cleft. It has two advantages: restoring the normal anatomic position of the levator palatini in the middle and posterior third of soft palate and increasing soft palate length (Fig. 45.16b). Complications of palatoplasty include postoperative bleeding, airway obstruction, wound dehiscence, and fistula formation. Care should be taken to achieve adequate intraoperative hemostasis and careful postoperative monitoring is essential to avoid airway obstruction.⁶⁸



(a)



(b)

Fig. 45.15 (a) Classic two-flap palatoplasty showing two pedicled palatal mucoperiosteal flaps and closure of muscle and nasal mucosa. (b) The two flaps are brought together by good approximation of the oral layer over the nasal layer.

Speech and velopharyngeal dysfunction

Children with palatal clefts are at risk for a wide range of speech problems related to resonance, articulation, phonation, learning, and language delay. These speech abnormalities can be caused by velopharyngeal insufficiency, oronasal fistula, weak lip pressure, abnormal tongue pressure, malpositioned teeth, abnormal jaw relationship, neuromuscular dysfunction, and conductive or sensorineural hearing loss. It is important to identify and associate the cause with effect. Assessment of speech should begin as early as 6 months of age and be monitored throughout adolescence.

Speech in cleft individuals often has a nasal quality. This perceived hypernasality during speech is typically due to incomplete closure of the velopharyngeal port which separates the nasal cavity from the oral cavity during speech production. Typically, velopharyngeal

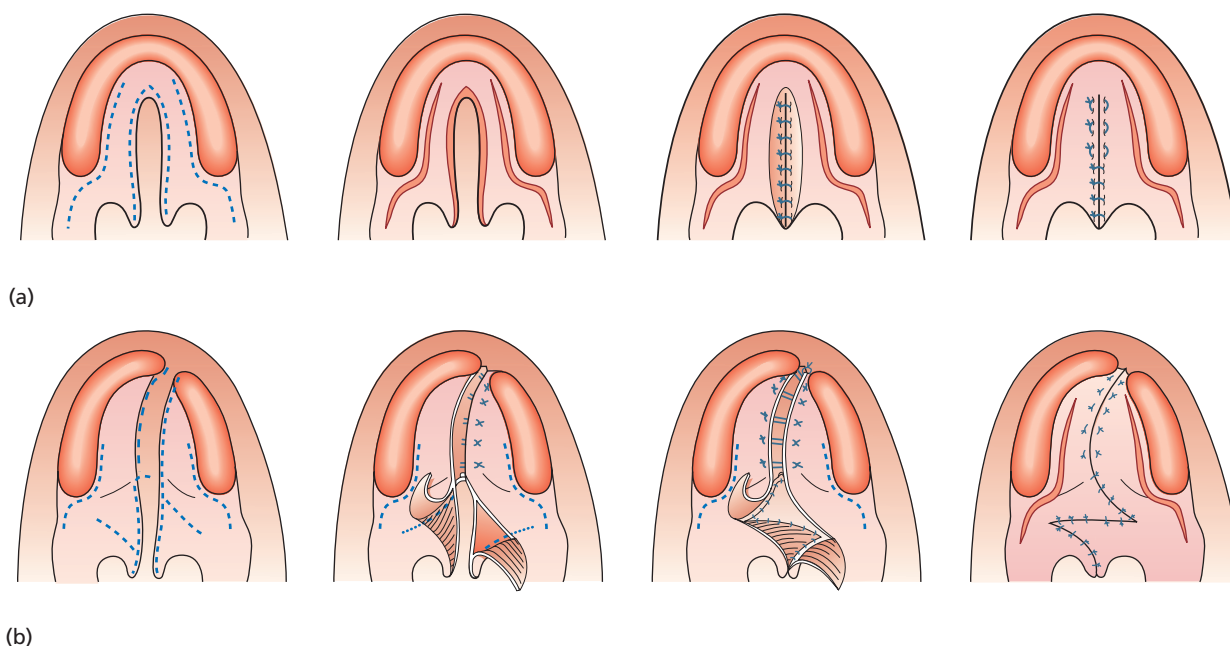


Fig. 45.16 (a) The Von Langenbeck technique of palatoplasty is generally used for clefts of the secondary palate. (b) Schematic diagram of Furlow Z-plasty technique, showing the reverse opposing double Z-plasty of muscle and mucosa.

ryngeal insufficiency (VPI) refers to the inability of the soft palate and the posterior and lateral pharyngeal walls to come together to create a seal during speech production. Nasal air escape during speech may be due to an unrepaired submucous cleft of the palate. This can also occur after repair of the palate, due to improper position of palatal musculature impeding movement and/or insufficient soft palate length.

Assessment of speech for VPI has to be both clinical and instrumental. Clinical assessment of resonance characteristics is best performed as a child's articulatory repertoire develops. Non-instrumental testing utilizing visualization of airflow with a reflecting mirror and nasal pinching often assists with the prediction of velopharyngeal function during speech. If deficits are identified, then further assessment using nasendoscopy to assess posterior and lateral pharyngeal wall motion or videofluoroscopy is indicated. Nasopharyngoscopy is a diagnostic tool for evaluation of velopharyngeal function that helps the surgeon to make a decision regarding the need for therapeutic intervention. Small-diameter pediatric flexible endoscopes with a good light source and topical anesthetic for nasal mucosa will help children become more compliant with this procedure.

Studies show that the need for surgical correction of VPI ranges from 4–30%.^{77,78} The most effective method for correction of VPI is controversial but the choice of the procedure depends on cause and where abnormality is found: lateral or posterior pharyngeal wall.⁷⁹ A posterior flap pharyngoplasty is indicated when there is limited or lack of posterior wall motion. The flap may be superiorly or inferiorly based. A superiorly based flap, with the base at the level of the tubercle of the atlas and insertion into the soft palate, is more popular than the inferiorly based

flap. A lateral pharyngoplasty is advocated for managing decreased lateral wall motion by creating a dynamic sphincter to control the size of the pharyngeal orifice. This was first described by Orticochea and modified by Jackson.⁸⁰ The sphincter is created by posterior tonsillar pillars, including the palatopharyngeus muscle, which are raised and sutured end to end.⁸¹

Correction of oronasal fistulae

One of the complications of primary cleft palate repair is failure of healing or breakdown of wound resulting in oronasal fistulae. Fistulae can occur at any location in the hard or soft palate and may cause nasal air escape as well as regurgitation of oral fluids. The reports on incidence of fistula formation are variable and range from 2–43%. Incidence of oronasal fistulae depends on several variables, including experience of the surgeon and age at the time of repair; it is less often related to the type of repair and severity of cleft deformity. The most significant variable seems to be the experience of the surgeon.^{77,82,83}

Repair of the fistula varies according to its anatomical location, whether it is in the anterior or posterior part of the palate. Various local pedicled flaps have been described to close the defect. Posteriorly or anteriorly based tongue flaps, musculo-mucosal flaps based on the facial artery, a temporalis flap, or local palatal mucoperiosteal flaps can be used to close fistulae. More recently, interpositional grafts made of acellular dermis have been used to achieve tension-free closure of oronasal fistulae.⁸⁴ It is important to have at least a two-layered closure without tension and maintain good supply to the flap in order to

achieve a watertight seal of the defect without recurrence. Almost all of the fistulae can be repaired by local pedicled flaps as described above. However, occasionally a vascularized flap may be helpful in closure of a scarred palate with a large defect.

Orthodontic management of the cleft individual

The orthodontist plays an important role in the care of the cleft individual during infancy, mixed dentition and permanent dentition (Table 45.3). Presurgical orthopedic treatment is facilitated by the orthodontist between 2–10 weeks after birth in infants with wide clefts and poorly aligned alveolar segments or a protruding premaxilla. In the early mixed dentition phase, children with complete clefts of lip and palate often have a posterior and anterior crossbite. The crossbite is asymmetric in unilateral clefts, affecting mainly the lesser segment or cleft side. In bilateral clefts, there is collapse of both lateral segments with a bilateral posterior crossbite and protrusion of the premaxilla. The goal of orthodontic treatment in this phase is to prepare for repair of the alveolar cleft by expanding the maxillary alveolar segments, and correcting the position of rotated maxillary incisors. The use of a quad helix or a screw expansion device allows greater expansion in the anterior maxillary arch (Fig. 45.17).

Monitoring facial growth during childhood and early adolescence helps to identify early signs of maxillary hypoplasia and allows intervention if indicated. Maxillary growth may be restricted in the vertical, transverse, and antero-posterior dimensions in chil-

dren with complete CLP. These children may benefit from maxillary protraction using a reverse-pull headgear or early maxillary osteotomy and distraction osteogenesis to minimize the severity of deformity.⁸⁵ The typical orthodontic treatment for a cleft patient following the eruption of the permanent teeth consists of maintaining maxillary arch width after repair of alveolar cleft, and alignment of teeth with full fixed appliances. This should be timed appropriately based on need for orthognathic surgery, the individual's growth potential, and ability to cooperate and maintain oral hygiene. Extraction of teeth may be necessary, particularly in the mandibular arch, if there is arch length deficiency. When surgery is indicated orthodontic treatment is coordinated with timing of growth completion, which is around age 15–16 years for females and 17–19 years for males. It is important to integrate the plan for the replacement or substitution of the absent maxillary lateral incisor and any other missing teeth into the orthodontic treatment plan.⁸⁶

Alveolar cleft repair

The primary goal of alveolar cleft repair is to establish bony continuity of the maxillary alveolar ridge, provide bone support for the teeth adjacent to the cleft, and seal the communication between the nose and oral cavity when there is a patent oronasal fistula. A successful alveolar bone graft should facilitate eruption and orthodontic movement of teeth in the line of the cleft (most often the maxillary canine), maintain the health of the periodontium of teeth adjacent to the cleft, provide alar base support, and improve nasal symmetry.^{87–89} Gingivoperiosteoplasty performed at the time of primary lip repair may seal the oronasal communication but does not always preclude the need for a bone graft. Some studies show that at least 40–50% of these patients require bone grafting in the

Table 45.3 Orthodontic management in cleft individuals

Age	Treatment
2–10 weeks	Presurgical orthopedics
6–10 years	Phase I orthodontics: Maxillary expansion for alveolar bone grafting Maxillary protraction with face mask when indicated
10–14 years	Maintain maxillary expansion and alignment of teeth Monitor facial growth and eruption of permanent teeth Orthodontic treatment with distraction of maxilla if deficiency is severe
14–18 years	Phase II orthodontics: Orthodontic treatment with full fixed appliances to align teeth Prepare for orthognathic surgery when indicated Extract teeth if mandibular arch is crowded Decide whether to replace/substitute the absent maxillary lateral incisor

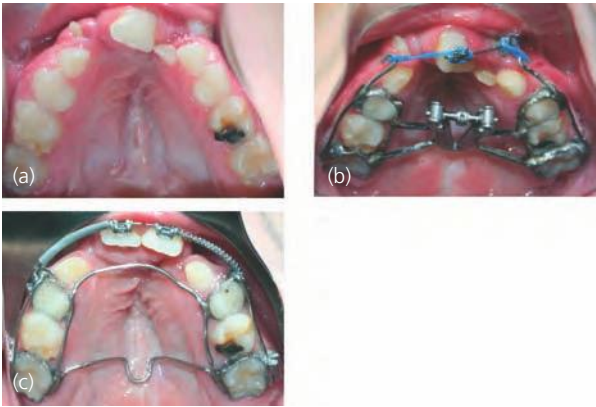


Fig. 45.17 Orthodontic expansion in a bilateral cleft of lip and palate. (a) Collapsed alveolar cleft segments with decreased anterior maxillary arch width. (b) This maxillary expansion device allows greater increase in arch width in the anterior region more than the posterior region. (c) Maxillary width being maintained after expansion.

future.^{90–92} More recently, Meazzini *et al.* have shown that patients who had a gingivoperiosteoplasty at the time of primary repair did not require bone grafting at a later date; however, they report a greater need for surgical correction of maxillary hypoplasia.⁹³

The timing, source of bone, the surgical technique, perioperative management, and outcome of bone grafting have all been studied and debated. The terms primary bone grafting (2–5 years of age) secondary bone grafting (7–11 years of age), and late secondary bone grafting (14–18 years of age) have been used to define the time of alveolar cleft repair.^{94,95} The majority of the centers use secondary alveolar bone grafting in the mixed dentition phase between the ages of 7 and 11 years, after maxillary expansion. Typically, this coincides with one half to two thirds of root development of the maxillary canine or lateral incisor (if present) in the line of the cleft. Completion of the maxillary expansion prior to grafting provides adequate access to the cleft defect and aligns the cleft segments better.

The gold standard for the grafting material has been particulate corticocancellous bone marrow harvested from the iliac crest, which was first described by Boyne and Sands.⁹⁶ Other sites that have been described include rib, symphysis, calvarium, and tibia.⁹⁷ More recently, the use of bone growth factors, such as recombinant human bone morphogenetic protein-2 (rhBMP-2), has been shown to be effective in grafting the defect. BMP-2 promotes differentiation of pluripotential cells into cells that can form new bone in the defect. This eliminates donor site morbidity and minimizes hospital stay and postoperative pain and discomfort.^{98–100}

New imaging techniques, such as cone-beam computed tomography, allow more accurate assessment of the volume of the bony defect before surgery and radiographic outcome of the grafted alveolus after surgery, when compared to the conventional two-dimensional periapical and panoramic radiographs.

The principles of surgical repair are the same for unilateral and bilateral clefts and include: proper closure of nasal floor mucosa to seal the communication between the nose and oral cavity; removal of supernumerary teeth in the cleft defect; filling the defect with cancellous bone; and approximation of the oral mucosa on the labial and palatal aspects to achieve a watertight closure over the grafted bone, as illustrated in Fig. 45.18. In the bilateral cleft, the position of the premaxilla makes the repair more technically challenging. The premaxilla is often inferiorly positioned, and retruded with the incisors located below the level of the occlusal plane. In some instances, the position of the premaxilla can be corrected by presurgical orthodontics, however in others it may have to be corrected by performing a vomerine osteotomy at the time of surgical alveolar cleft repair. The osteotomized premaxilla has to be maintained in position with a splint for 4–6 weeks postoperatively.^{101,102}

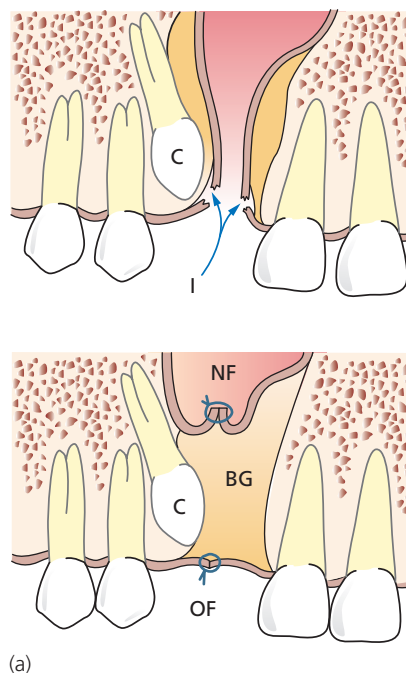


Fig. 45.18 Alveolar cleft repair. (a) Schematic diagram of the alveolar cleft showing closure of the nasal and oral mucosa. (Reproduced from Assael, L. (1995) Cleft lip and palate. *Atlas of the Oral and Maxillofacial Clinics of North America* 3(1), p. 31. Copyright © 1995 Elsevier.) (b) The alveolar cleft deformity is visible and nasal mucosa is closed to seal the communication between the oral and nasal cavities. (c) Cancellous bone is packed into the defect after closure of the palatal oral mucosa.

The amount of maxillary arch expansion prior to surgery should be planned and monitored. Too much presurgical expansion of the lateral segments leaves a large palatal defect making it difficult to advance the scarred palatal mucoperiosteal flaps to cover the bone graft, and too little expansion limits surgical access to the alveolar cleft. After alveolar cleft repair, orthodontic tooth movement, if necessary, can be initiated in 3–4 months. The maxillary width achieved by presurgical expansion should be maintained after surgery with a palatal retainer until phase II orthodontic treatment begins at a later date.

Replacement of absent teeth in the line of the cleft

Congenital absence of the maxillary lateral incisor in the line of the cleft can be managed appropriately by closing the space and substituting the adjacent canine in its position or by opening space and replacing it with a fixed or removable prosthesis. The experience at the center for cleft and craniofacial anomalies at the University of California has shown success of implants in grafted unilateral clefts is better than in bilateral clefts. In the bilateral cases the periodontal health of the central incisors is very often compromised, and the lateral incisors are also absent. The substitution of the lateral incisor with the adjacent canine is cost-effective; however, it can result in loss of arch length and decrease in transverse dimension of the maxillary arch. It can also be unesthetic due to the asymmetry in tooth size and shape in unilateral cases. Replacement with an endosseous implant is an option when there is adequate space and bone quantity. There may be inadequate bone at the time of placement of the implant even if the alveolar cleft was repaired previously.¹⁰³ Alveolar ridge augmentation with a cortical onlay graft performed about 4 months prior to placement of the implant will pro-

vide adequate bone height and width for placement of the implant (Fig. 45.19). A removable prosthesis should also be considered when there is loss of premaxilla, severe deficiency of bone height and width with scarring of the overlying soft tissue, and lack of lip support. Use of teeth-supported fixed prosthetic restorations, such as a bridge, across the unrepaired alveolar cleft segments should be avoided, as movement of the cleft segments results in failure of the prosthesis and loss of abutment teeth.

Surgical correction of maxillary hypoplasia

Cleft lip and palate patients exhibit varying degrees of maxillary hypoplasia due to restriction of midfacial growth that is apparent in the sagittal, vertical, and transverse dimensions. This midface deficiency can be attributed partly to the intrinsic reduction in growth potential due to the congenital malformation and, partly to scar contracture following primary palate surgery.^{104–106} The maxillary deficiency caused by the cleft deformity is superimposed upon the genetically inherited skeletal growth pattern. Hence, an underlying inherited pattern of excessive mandibular growth can present as severe midfacial deficiency and a class III malocclusion in a cleft patient (Fig. 45.20). Regardless of the etiology of maxillary hypoplasia, approximately 25% (reported range 14–50%) of cleft individuals undergo surgical correction of maxillary hypoplasia.^{107–110} This wide range highlights the differing indications for skeletal correction by cleft team specialists. Their decisions are influenced by differing treatment philosophies (orthodontic management with dental compensation vs surgical maxillary advancement to address facial esthetics and occlusion more comprehensively), the availability of appropriately trained surgeons, and hospital/government funding. Good *et al.* reported

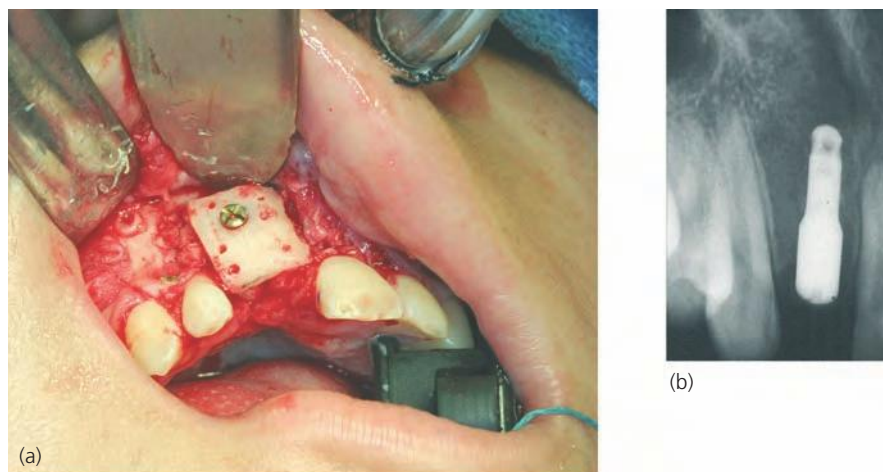


Fig. 45.19 Replacement of missing lateral incisor. (a) Alveolar ridge augmentation with a cortical bone onlay is often necessary to increase width and height, before placement of an implant in the grafted cleft site. (b) Unilateral cleft with a missing lateral incisor replaced with an endosseous implant.



Fig. 45.20 Maxillary growth in a child with a unilateral cleft lip and palate. (a) Note that the signs of maxillary deficiency are apparent in the profile view at 9 years in this child with a unilateral cleft lip and palate. (b) The severe deficiency of the maxilla in antero-posterior and vertical dimensions and its relative position to the mandible are seen at 15 years of age.

that the frequency of a Le Fort I osteotomy for correction of maxillary hypoplasia correlated with the severity of clefting.¹⁰⁹

Technical considerations for cleft orthognathic surgery

Surgical correction of maxillary hypoplasia is usually performed after growth is completed, unless indicated earlier for psychological reasons. When the skeletal discrepancy is severe, a combined maxillary advancement and mandibular reduction can be performed as seen in Fig. 45.21. A staged advancement of the maxilla can be considered, particularly when the mandible is in a normal position. Early surgical maxillary advancement before the completion of growth will help to minimize the reverse overjet and potentially avoid or decrease the magnitude of mandibular reduction at a later date. An example of staged maxillary advancement is shown in Fig. 45.22. In the last decade, distraction osteogenesis of the facial skeleton has become popular and facilitated substantial maxillary advancement without the need for mandibular osteotomies and bone grafting.¹¹¹

Orthognathic surgery in the cleft patient is much more challenging than in the non-cleft patient. Anesthetic management may sometimes be difficult due to the presence of a deviated nasal septum and/

or a pharyngeal flap. It may be necessary to use a fiberoptic-assisted technique or pass an endotracheal tube over a more rigid tube or catheter. The vascularity of the labial and palatal mucoperiosteal tissues is invariably affected by previous surgical procedures for lip and palate repair. Drommer and Luhr demonstrated with the use of angiography that the greater palatine arteries were significantly smaller in 10 of 24 sides in 12 cleft patients prior to maxillary advancement.¹¹² When making mucosal incisions to provide access for the osteotomies, care should be taken to maintain a generous buccal pedicle, preserve the greater palatine arteries if possible, and to avoid unnecessary trauma to the palatal and buccal soft tissue pedicles. Failure to do so may result in the loss of attached gingival tissues, bone, and teeth.

The nasal mucosa and palatal mucosa are fused in the region of the cleft due to the primary palate repair. This tissue should be incised close to the nasal floor just prior to the “down-fracture”. Attenuation of scar tissue close to the nasal floor is necessary to free the maxilla during the down-fracture. Mobilization of the maxilla after down-fracture is more difficult due to the palatal scar tissue and/or a pharyngeal flap. In some cases, release or division of the pharyngeal flap may be indicated to reposition the maxilla into the desired position. The primary palatal repair often results in more bone formation and stronger union at



Fig. 45.21 Cleft orthognathic surgery. An 18-year-old female with secondary skeletal and soft tissue deformities of bilateral CLP corrected with maxillary and mandibular osteotomies, and cheilo-rhinoplasty. (a) Before and (b) after in frontal view. (c) Before and (d) after in profile view. (e, g) Preoperative occlusion reveals anterior and posterior crossbite. (f, h) Postoperative occlusion after combined orthodontic and surgical correction.

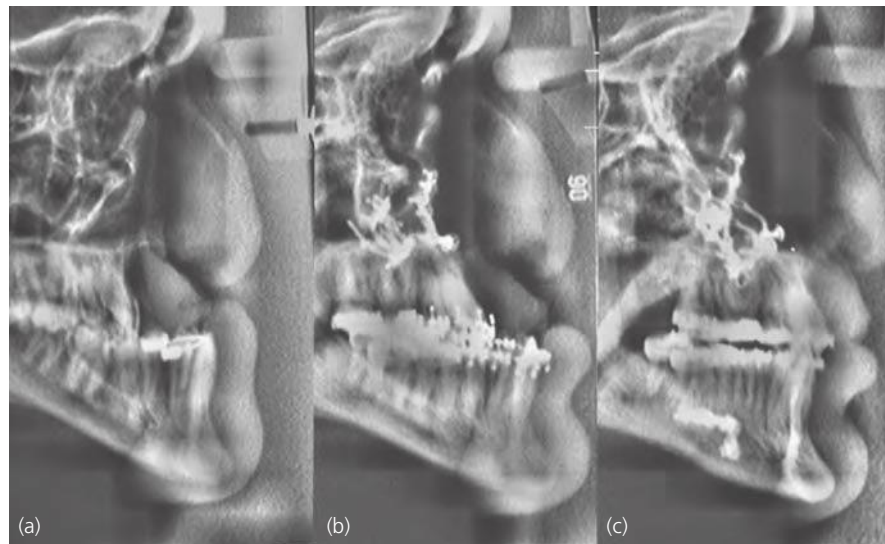


Fig. 45.22 Cleft orthognathic surgery. Radiographs of 18 year-old male with unilateral CLP and severe maxillary hypoplasia corrected by staged orthognathic surgery. (a) Preoperative lateral cephalometric radiograph. (b) Postoperative view after initial maxillary advancement. (c) Postoperative view after maxillary advancement and mandibular setback.

the pterygo-maxillary junction. Use of a curved chisel to osteotomize directly through the maxillary tuberosities rather than the dense bone of the pterygo-maxillary junction is helpful to complete this posterior osteotomy. Complete mobilization to achieve sufficient advancement requires progressive, careful stretching as the tissues are less compliant than normal tissues.

The maxillary deficiency in the vertical, transverse, and sagittal dimensions makes bone grafting a necessity for cleft maxillary osteotomies performed by conventional orthognathic surgery. A persistent oronasal fistula and alveolar cleft defect, when present, require careful soft tissue closure and bone grafting. The maxilla can separate into two segments during the down-fracture and mobilization even when the



Fig. 45.23 (a) A schematic diagram illustrating a Le Fort I osteotomy with simultaneous repair of the unilateral alveolar cleft defect. (Reproduced from Assael, L. (1995) Cleft lip and palate. *Atlas of the Oral and Maxillofacial Clinics of North America* 3(1), p. 55. Copyright © 1995 Elsevier.) (b) Corticocancellous bone harvested from the anterior iliac crest can be used to graft the cleft site and osteotomy gap created by advancement and inferior repositioning. (c) Profile view before maxillary advancement and alveolar cleft repair. (d) Profile view after maxillary advancement and alveolar cleft repair. (e) Frontal view shows mild deficiency of midface with poor alar base support. (f) Frontal view in smile shows better midface projection.

alveolar cleft site has been previously grafted. Corticocancellous bone harvested from the anterior iliac crest can be used to graft the residual alveolar cleft defect, to bridge the gap at the osteotomy site after anterior and inferior repositioning of the maxilla, and to augment the deficient area at the piriform rim on the cleft side (Fig. 45.23).

Rigid fixation with plates and screws has to be performed carefully as the quantity and quality of bone is often compromised. When there is deficiency of the midface and malar region, adjunctive procedures, such as zygomatic osteotomies or a modified Le Fort I osteotomy, can enhance cheek prominence and facial contours (Fig. 45.24).

The soft tissue of the upper lip may be tight with a shallow vestibular depth and deficient vermilion show that may become worse following maxillary advancement. When closing the incision, incorporating a V-Y design to minimize lip shortening should be considered. Alar flaring is a common problem in

the distorted cleft nose following a large maxillary advancement. An alar base suture passed in a figure-of-eight fashion through the anterior nasal spine will help to maintain a satisfactory alar base width.

VPI after maxillary advancement has been well documented.^{113,114} The predictability of VPI after conventional orthognathic surgery is based on premaxillary speech. If there is moderate to severe insufficiency prior to maxillary advancement it is more likely that individuals will need postsurgical pharyngeal flap or pharyngoplasty. It is therefore important that these patients are thoroughly assessed for VPI prior to cleft maxillary surgery.¹¹⁵

Conventional orthognathic surgery vs distraction osteogenesis

Postoperative stability after maxillary osteotomy is less favorable in the cleft than in the non-cleft patient. Relapse in the cleft individual is mainly due to the inability to mobilize the maxilla adequately. The tight scarred soft tissue envelope of the palate and upper lip limit the ability to mobilize and achieve the desired movement. Despite bone grafting and rigid internal fixation, relapse in the sagittal and vertical dimensions after maxillary osteotomy has been reported in several studies.^{116–121} Distraction osteogenesis can overcome some of the difficulties encountered with mobilization and stability in conventional orthognathic surgery: it is the technique of gradually repositioning the osteotomized bone segments while forming new bone in the gap created by the osteotomy. Distraction osteogenesis is technique-sensitive, costly, and requires patient cooperation and surgical expertise to properly align, place, and activate the device in order to achieve a good outcome.

Polley and Figueroa first described the use of a rigid external frame to distract the maxilla in a series of cleft individuals. They demonstrated a mean advancement of 11.7 mm.¹²² Since then, several centers have reported their experiences in maxillary distraction in cleft individuals using face masks to provide the external anchorage^{123,124} and internal distractors.¹²⁵ A meta-analysis of literature from 1996–2003 by Cheung *et al.* on cleft maxillary osteotomy and distraction osteogenesis showed that the majority of patients who underwent conventional maxillary osteotomies were older (16–20 years) compared to those who had distraction (11–15 years). The mean advancement for both groups was similar but larger maximum advancement was achieved in the distraction group. The most commonly used system was the rigid external distractor (RED) device.¹²⁶ They subsequently conducted a randomized, controlled study comparing maxillary distraction and orthognathic surgery in 29 non-growing, cleft patients. Intraoral distractors were used in 15 patients in the distraction group and routine miniplate fixation for the 14 patients in the orthognathic surgery group. Clinical morbidity and stability were assessed using a ques-

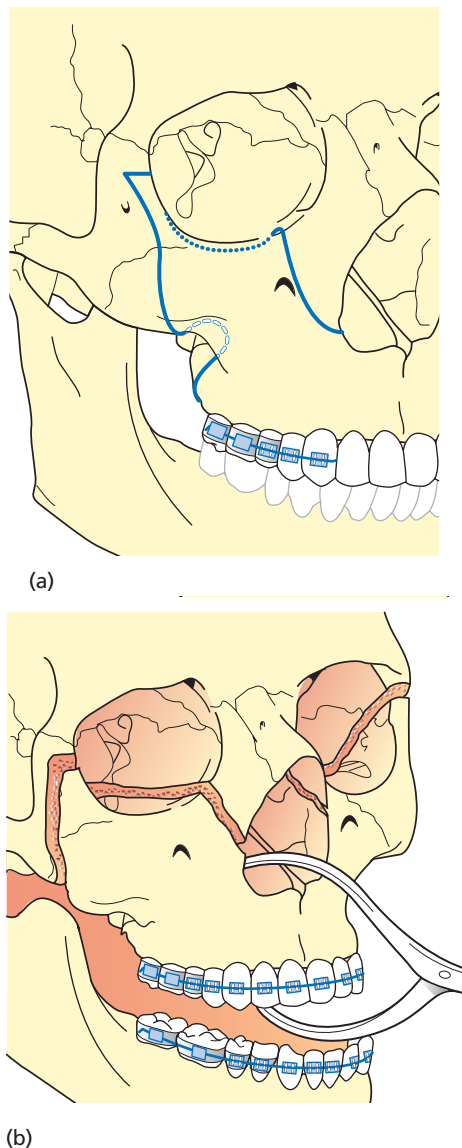


Fig. 45.24 (a, b) Midface osteotomy including the zygoma and infraorbital rim for correction of severe midface deficiency.

tionnaire and lateral cephalometric tracings respectively. It was found that there was no significant difference in clinical morbidity but the maxillary movement in the distraction group was more stable than in the orthognathic repositioning group. Skeletal relapse was evident in the first 3 months following conventional cleft maxillary advancement.¹²⁷ Improved stability and speech, and marked improvement in hard and soft tissue profile were also reported for distraction by other investigators.^{128–131} In a 2-year follow-up of 12 cleft patients with moderate to severe maxillary retrusion who underwent distraction with internal distractors, a mean maxillary advancement of 14mm with good stability was reported by Rachmiel *et al.*¹²⁵ At the Royal Children's Hospital of Melbourne, internal distractors have been used over the past 10 years in select cases. Unlike external distractors, the internal distractors lack three-dimensional control and particularly the ability to manage the vertical vector. Other disadvantages of internal distraction include a limitation of distractor length and the need for a separate procedure for removal. External frames are bulky and uncomfortable, but provide better vector control and changes can be made to the vector during distraction. Also there is no need for a separate procedure to remove the distractor (Fig. 45.25).

For severe maxillary hypoplasia in the cleft patient, distraction is considered an option as an interim procedure during growth. The aim is to minimize or eliminate the need for mandibular reduction where the mandible is within normal dimensions and relationship to the facial structure. Distraction obviates the need for internal fixation and bone grafting but disadvantages include prolonged facial edema and the occasional inflammatory response to the transmucosal presence of the device activation arms. Traditional staged maxillary advancements remain a predictable option for patients, but the ability to produce large advancements using distraction is attractive in those with marked retrusion. Fig. 45.26 illustrates correction of severe maxillary hypoplasia with distraction. As more clinical studies evaluate the results of midfacial distraction techniques in comparison with conventional osteotomies, it is hoped that protocols can be developed to reflect the best timing and indication for each technique.

Surgical correction of secondary lip and nose deformities

The final phase of treatment for the cleft patient involves correction of the residual lip and nasal deformities to achieve balanced facial esthetics. Typically, the lip and nose revision is performed during late adolescence after skeletal correction of maxillary hypoplasia, and after postsurgical orthodontic treatment is completed to provide a stable dental occlusion.

Secondary lip deformities

Secondary lip deformities that require correction are mainly asymmetries or disproportions. For the majority of patients where the primary repair was carefully planned and executed, secondary lip revisions are minor. In unilateral cleft lip patients, the residual deformities include mismatch of the cutaneous–vermillion line, notching of the vermillion or a “whistle deformity”, vertical shortening of the lateral lip element, a hypertrophic scar, and poor muscle function due to a discontinuity of the orbicularis oris.¹³²

Notching or mismatch at the vermillion–cutaneous junction can be corrected by realignment, small triangular flaps, or a Z-plasty procedure. A poorly defined tubercle can be corrected by a V–Y advancement. It is important to differentiate and maintain the zone of wet and dry mucosa when correcting these deformities. Vertical shortening of the lip after a Millard repair is due to underestimation of the vertical height and inadequate rotation of lip on the non-cleft side at the time of primary repair. Shortening of the lip can also result from severe scar contracture. Correction of a prominent scar with inadequate lip length and compromised muscle function requires a full-thickness revision where all three layers (skin, muscle, and mucosa) have to be cleanly dissected and meticulously repaired (Fig. 45.27).

In the case of a poorly repaired bilateral cleft lip, the deformity can present as a tight upper lip with poorly defined philtral columns and cupid's bow, a short and wide prolabium, poor vermillion show in the center, unsightly scars, exposed wet mucosa, and a shallow labial sulcus. Deficiency of the labial sulcus can be corrected by releasing the scar and performing a Z-plasty. In the case of a tight upper lip, an Abbe flap can be used successfully for reconstruction of the upper lip deformity. This is an axial pattern flap consisting of mucosa, skin, and muscle from the lower lip described by Abbe in 1898. The advantages of this flap are that it carries the dimple from the lower lip to form a philtral dimple in the cupid's bow and the scars give the semblance of philtral columns.

Cleft nasal deformity

Asymmetry and distortion of the nasal morphology is often the most obvious feature of an individual with a cleft. Correction of the nasal deformity in cleft individuals is a challenge due to soft tissue distortions, scarring, nasal stenosis, and uneven bony foundations. Primary correction of the cleft nose deformity is performed by most surgeons today. This does not eliminate the need for a secondary rhinoplasty, but better results can be achieved as the deformity may be less severe.¹³³ The pathological anatomy in the unilateral cleft nose is an asymmetric nasal pyramid, hypoplastic maxilla on the cleft side, and hypertrophied inferior turbinate with deviation of the anterior

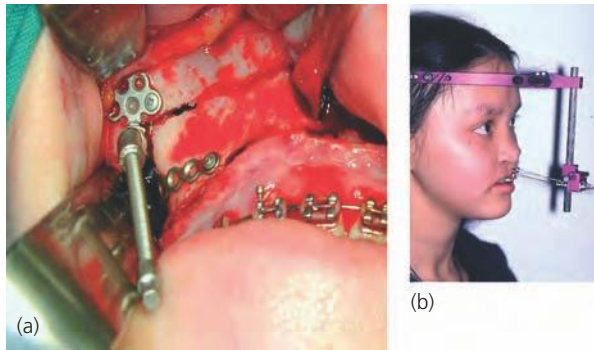


Fig. 45.25 (a) An internal maxillary distractor (KLS Martin) in place secured above and below the Le Fort I osteotomy. (b) A rigid external distractor (RED) provides skeletal anchorage with the head frame in a young female with maxillary hypoplasia.

nasal spine and base of columella to the non-cleft side. There is inadequate nasal tip support, posterior and inferior displacement of the dome of the alar cartilage, and inward buckling of the ala on the cleft side.¹³⁴ The bilateral cleft nose is less asymmetrical and the predominant deformity is that of a short columella. Other features of the nasal deformity include a broad downward-rotated nasal tip that lacks support, lower lateral cartilage domes that are widely separated, and lateral splaying of the alar rims with nostril apertures more horizontally oriented.

Cleft septorhinoplasty

There is a wide variation in timing for secondary nasal revisions, with some authors recommending early rhinoplasty. Unless there is a particularly strong

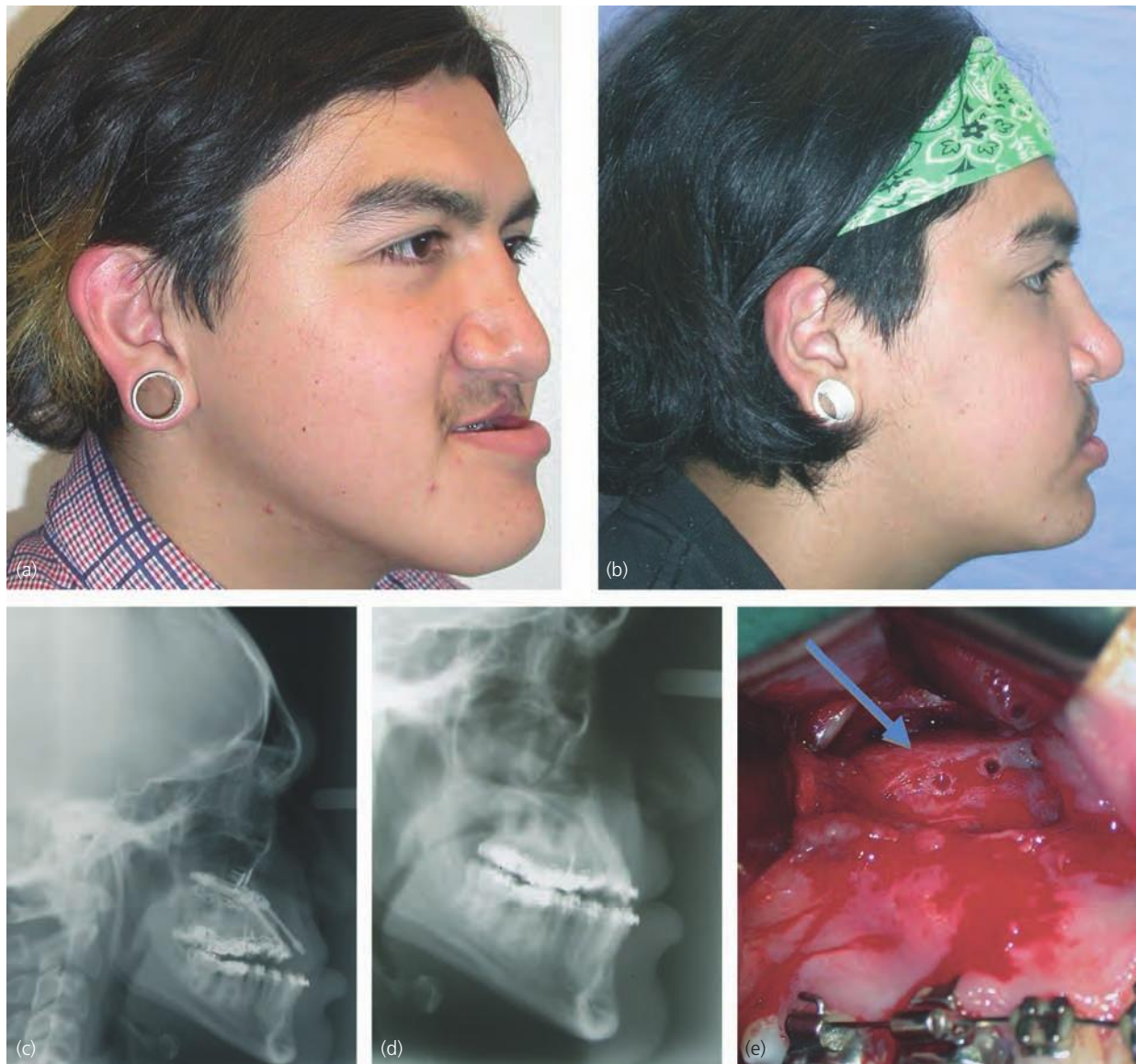


Figure 45.26 Maxillary advancement with internal distraction. (a) Preoperative profile view of 19-year-old male with severe maxillary deficiency and large reverse overjet. (b) Lateral cephalometric radiographic after osteotomy and placement of distractor. (c) Postoperative radiograph 6 months after removal of distractors showing some relapse. (d) Postoperative facial profile view shows improvement in midface projection and nasal tip support. (e) Bone generated at the osteotomy site by maxillary distraction (arrow).



Fig. 45.27 (a) Whistle notch deformity of a unilateral repaired cleft lip. (b) Lip contour following naso-labial revision.



Fig. 45.28 (a, b) Preoperative nasal deformity in frontal and inferior facial views showing collapsed alar cartilage with poor tip support. (c, d) Postoperative frontal and inferior facial views following septorhinoplasty show improved tip support and symmetry.

psychological indication or severe nasal obstruction, definite secondary septorhinoplasty in the cleft patient should follow skeletal maxillary reconstruction. The total correction of the cleft nasal deformity is best approached by an open rhinoplasty under direct vision. Wide exposure provides access for mobilization and repair of the collapsed and deformed alar cartilages.^{135,136} In unilateral cleft patients, an open rhinoplasty approach is performed using a trans-columellar incision combined with rim incisions. Complete degloving of the lower lateral cartilages and dissection to the cartilaginous septum enables a full septoplasty with relocation of the caudal septum and the harvesting of cartilage grafts if indicated. Dorsal reduction is commonly undertaken with bony osteotomies to restore the bony vault. Additionally, stiffening the columella by a cartilage strut is necessary to provide tip projection and support for a more symmetrical nasal tip. Reshaping the deficient lateral crus may require a batten graft, and alar base repositioning may be required to improve nostril symmetry (Fig. 45.28). In bilateral clefts, various approaches, including a V-Y advancement to lengthen the columella, have been undertaken, but undesirable scarring is often the outcome. The entire prolabium can be used to lengthen the columella and an Abbe flap can be used to fill the upper lip defect.¹³⁷ The goal is to stretch the nasal tip and to dissect and approximate the lower lateral cartilages to achieve a more triangular inferior nasal shape.

Summary

An esthetic smile and normal speech are two important characteristics which give an individual his or her identity, confidence and self-esteem. The ability to restore lip and palate function without jeopardizing growth with various surgical interventions is the key to management of cleft deformities. This requires a thorough understanding of the embryological, physical and psychological development of the child.

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Chapter 46

Diagnosis and Treatment Planning for Orthognathic Surgery

Johan P. Reyneke and Carlo Ferretti

The chapter is a modern and extremely relevant overview of how to perform and think in terms of diagnosis and planning for the orthognathic surgical patient. The therapeutic goals should be to establish functional occlusion and harmony between muscles, teeth, and joints. Furthermore one should aim to improve facial balance, correct speech problems, correct abnormalities related to airway compromise, improve stability of orthodontic and surgical results, and also improve dental and periodontal health. Thorough planning is the key to a successful result in orthognathic surgical cases. Orthognathic surgery techniques are described and commented upon.

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Correct diagnosis and efficient treatment planning for dentofacial deformities demand a systematic and objective approach. The basic therapeutic goals of orthognathic surgery are directed towards the correction of specific neuromuscular, dento-skeletal, and soft tissue deformities.

In the past the diagnosis and treatment planning for orthognathic surgery started with analysis of the dentition followed by skeletal and esthetic soft tissue considerations of the dentofacial abnormalities. The modern approach in treatment planning, however, focuses more on soft tissue objectives. Consideration is then given to where the dentition should be positioned by the orthodontist to allow for appropriate surgical repositioning of the jaws which would then result in the best possible esthetic result, normal occlusal function, and stability.¹ The authors believe

that this new direction of facial planning will help to avoid treatment errors such as orthodontic dental compensations worsening the facial imbalance of patients. Inappropriate treatment can often lead to poor facial balance, occlusal relapse, periodontal decline, temporomandibular joint problems, and inadequate airway space. It is, however, interesting to note that, when facial harmony and pleasing esthetics are achieved through proper diagnosis and treatment planning, complications such as those mentioned above are often avoided.

Patients present with a wide range of malocclusions and facial imbalances. Most patients with malocclusions will have normal skeletal relationships and will only require orthodontic treatment without compromise or complications (group 1) (Fig. 46.1a). There is a group of patients that will present with

malocclusions combined with a mild to moderate skeletal discrepancy (group 2). The treatment of some of the patients in this group could be managed by dental compensation and an attempt to control growth (Fig. 46.1b). It would be inappropriate to treat patients in this group with surgery if an acceptable result could be achieved by orthodontic treatment alone. For these cases it is, however, important to explain, demonstrate, and discuss the downside of the expected compromise result (Fig. 46.2). The third group of patients will present with moderate to severe facial imbalance and malocclusion that will require combined orthodontic and surgical treatment (Fig. 46.1c). There will obviously be borderline cases between the three groups of patients. Most errors and probably the more significant errors occur in the diagnosis, treatment planning, and treatment of borderline cases between group 2 and 3 patients (Fig. 46.3).

The differentiation between the groups of patients with mild (group 2) and severe (group 3) dentofacial

deformities is important and will be influenced by various factors:

- The patient's main concern – for some patients a functional occlusion and straight teeth are their main objective while for others facial esthetics and a balanced profile are more important.
- Treatment time – in most cases the combination of orthodontic treatment and surgery will shorten the total treatment time. This fact may influence some patients' decisions regarding treatment.
- The compromise – worsening of the esthetics as a result of orthodontic dental compensation in severe skeletal discrepancy cases (group 3) may be unacceptable to some patients or make no treatment at all a better option than compromised treatment.
- Affordability – due to inappropriate insurance cover many patients cannot afford the combination of surgery and orthodontic treatment.
- Orthodontist's approach – for various reasons, such as a bad experience as a result of inappro-

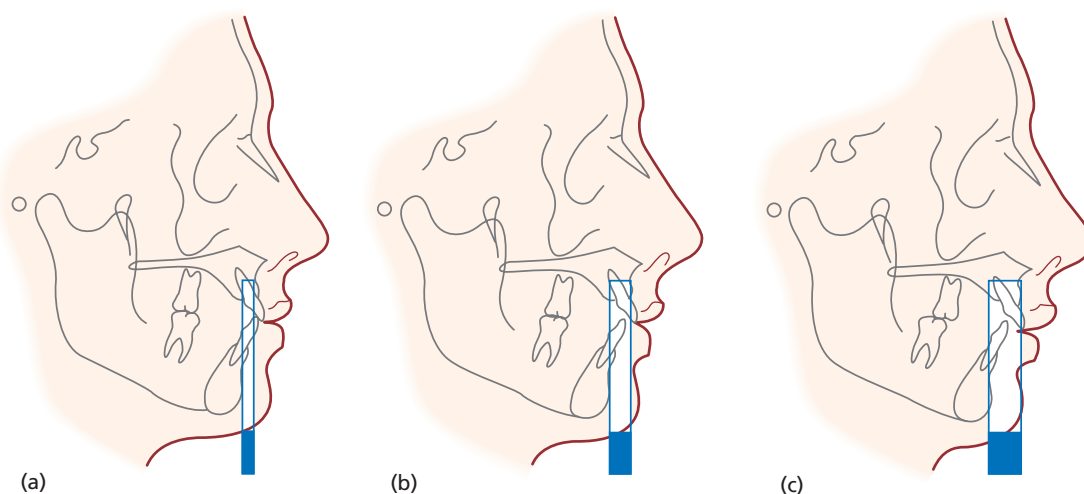


Fig. 46.1 (a) The patient illustrated has normal skeletal relations and any malocclusion can be corrected by means of orthodontic treatment alone. (b) The patient has a mild to moderate class II skeletal discrepancy and the malocclusion may be managed by means of dental compensation and growth modification. (c) The patient demonstrates a severe skeletal discrepancy and should be treated by a combination of surgery and orthodontics.

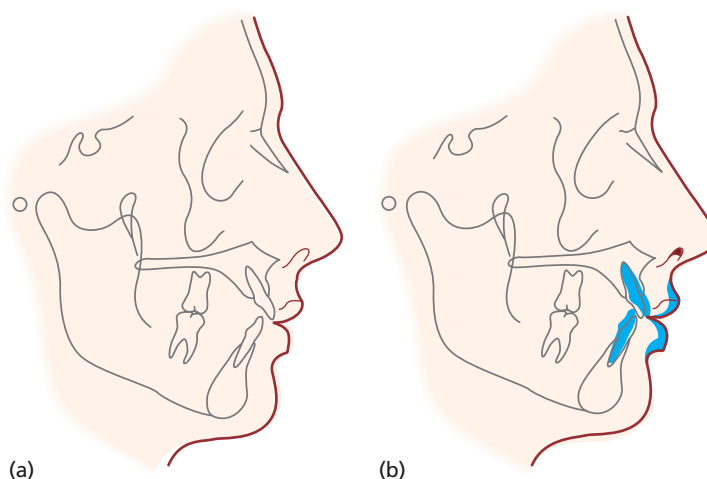


Fig. 46.2 (a) A patient in group 2. (b) The predicted soft tissue result following orthodontic compensation of the dentition. The esthetics and possible instability of the result should be evaluated and discussed with the patient.

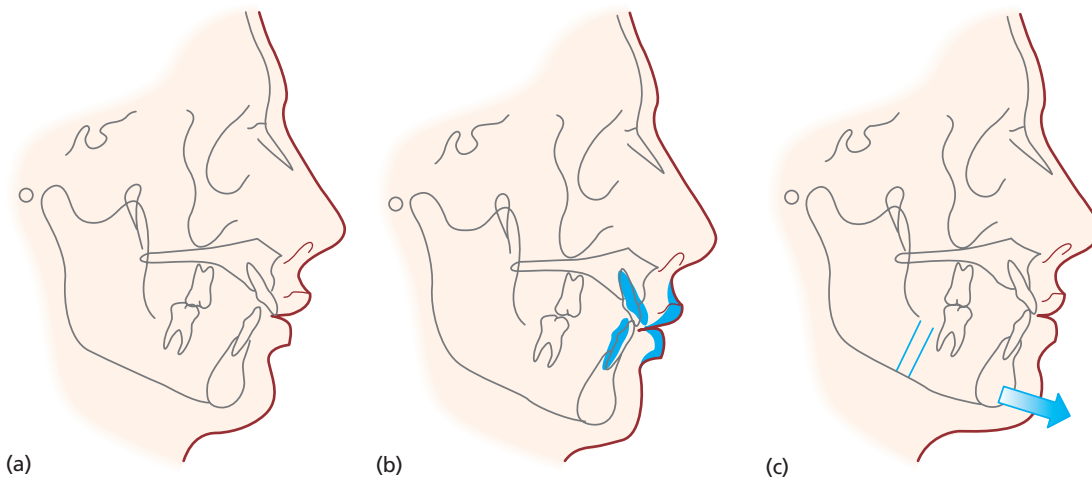


Fig. 46.3 (a) A patient in group 3. (b) The soft tissue prediction following orthodontic compensation reveals a poor esthetic result, while a more acceptable result can be achieved following surgical advancement of the mandible (c).

priate surgery or unavailability of specialized surgical treatment in the area, some orthodontists are reluctant to refer patients for orthognathic surgery.

The professional treatment team should have clear objectives in mind when making the diagnosis and developing a treatment plan so the patient can be well informed regarding the best possible treatment approach. The therapeutic goals that should be considered are one or more of the goals listed below:²

- establish a functional occlusion;
- establish harmony between the neuromusculature, the teeth, and the temporomandibular joints;
- improve facial balance;
- correct abnormalities related to airway compromise;
- correct speech problems;
- improve the stability of the orthodontic and surgical result;
- improve dental and periodontal health;
- shorten treatment time.

Introducing the patient to orthognathic treatment concepts

Most patients that may need orthognathic surgery and referral to the orthodontist will be oblivious of the fact that surgical correction may be a treatment option. The patient should therefore be gently but confidently introduced to the concept of surgery. The clinician should provide the patient with a realistic and understandable overview of orthognathic treatment principles and general treatment possibilities in relation to the patient's own specific dentofacial problem. Understanding the patient's concerns, motivation, and expectations will provide insight into the patient's psychological health, and the ease or difficulties to be anticipated when treating the patient in

the future. The following questions may help identify the patient's concerns:

1. What are your concerns or problems?
2. Have you had previous treatment for the condition and what was the outcome?
3. Why is treatment required and why now?
4. What do you expect from treatment?

Some patients may find it difficult to answer these questions and may need time to discuss future treatment with family and friends. Patients' priorities may also differ, i.e. for some patients a normal occlusion and straight teeth are important while for others good facial balance and an attractive smile will be a priority. Further counseling about realistic treatment expectations may be necessary, and it may be best if treatment is delayed until, through psychologic guidance, the patient can cope with treatment realities. There is however very seldom need for the above. The perception of one's own appearance is often the "motor" behind direction and confidence in life. Surgical orthodontic change of facial appearance inevitably has a profound effect on this motor.

Systematic patient evaluation

Following the general patient evaluation which would include a medical history, general dental evaluation, and psychologic evaluation (where indicated), a clinical evaluation of the face is carried out. Although orthognathic surgery is usually carried out on healthy individuals it does not preclude the significance of obtaining an appropriate and current medical history as it may affect treatment planning and help the surgeon prevent potential life-threatening complications. The clinical assessment of the face is the most valuable of all diagnostic procedures and should be performed in a systematic fashion with the patient seated comfortably in natural head posture and the teeth in

centric occlusion. Orthodontic and surgical treatments are planned to produce ideal function in centric occlusion. All examination data should therefore be recorded in centric occlusion. However exceptions to the rule would be patients with vertical maxillary deficiency and severe closed bites. To evaluate these patients' lips and upper incisor–lip relationship, they should be examined with their teeth apart and lips just touching. Balance, proportions, and harmony between the various facial structures are more important than numerical values. Only abnormal data should be recorded and the clinician should guard against becoming overwhelmed by a myriad of irrelevant data.

At the first consultation the clinician should, based on first impressions and without data from special investigations, pose the following questions:

1. What is the basic diagnosis of the face? Vertical problem, horizontal problem, maxillary antero-posterior excess/deficiency, mandibular antero-posterior excess/deficiency, etc.
2. What surgery would be required to correct the problem? Superior repositioning of the maxilla, advancement of the mandible, genioplasty, etc.
3. Where would I like the orthodontist to position the teeth to allow for the intended surgery, to achieve facial harmony, and to ensure stability of the results?

Facial evaluation

Several textbooks dedicated to orthognathic surgery and many publications in the medical and dental literature have comprehensively dealt with facial, cephalometric, and dental occlusal analysis as well as many special investigations available to assist in the diagnosis and treatment planning of patients with dentofacial deformities.^{1–3} It is the aim of the authors of this chapter to give the clinician a somewhat different approach to the analysis and diagnosis while still using some basic cephalometric and clinical parameters considered to be the norm for facial form and function.

Whilst the skill to identify esthetically pleasing faces is common to humanity, medical professionals have distilled and crystallized what it is that defines facial esthetics. Of necessity this has been reduced to numerization of facial characteristics which are then compared to normal values. However experienced surgeons have the ability to identify those facial features which detract from facial harmony without resorting to cephalometrics. This ability to identify facial harmony or the lack thereof is a skill attained after viewing many faces over many years. The authors' aim is not to undermine the value of adjunctive assessments such as cephalometrics and dental casts to orthognathic treatment planning, as these are critical contributors to treatment planning. However, they wish to facilitate the development of facial

assessment skills in surgeons embarking on a career in orthognathic surgery.

Facial esthetics is the cumulative result of optimum proportional relations of the esthetics subunits which make up a face. This interdependence makes the assessment of an individual facial component meaningless (i.e. we cannot comment on the antero-posterior position of a mandible until we see the rest of the face it belongs to). Moreover the arbitrary subdivision of the face into thirds is of limited value as the effects of treatment in a single jaw may stretch across two of three facial thirds and a single jaw may contribute to disharmony of more than a single third. It is challenging to formalize a qualitative facial assessment due to the fact that visual examination of a face automatically and instantly assesses facial harmony. Therefore some degree of facial subdivision is required to assist in facial description. The authors believe a more pragmatic approach to facial assessment is to divide the face into zones of influence, i.e. zones which can be modified by orthognathic surgery. Thus the face can be divided into an upper **forehead complex** (extending inferiorly from the hairline to a line connecting the eyebrows across the glabella), an intermediate **oculonasal complex** (extending inferiorly from the forehead complex to a line extending from the lower border of the zygomatic arch curving upwards to the infraorbital foramen, on to the nose above the supratip break continuing on to the opposite side), and finally the lower **gnathic complex**. This latter complex is subdivided into an upper maxillary component (which extends from the lower aspect of the oculonasal complex to a curved line extending along the lower margin of the upper lip, or the lower edge of the incisor teeth if these are visible below the upper lip, to the angle of the mouth and proceeding in a curvilinear fashion to the lower attachment of the auricle) and a lower mandibular component, which extends to the lower border of the mandible and contains in its anterior aspect the oval mental subunit which delimits the soft tissue chin (Fig. 46.4).

With this subdivision in mind one can proceed to facial evaluation. It is a critical point to remember that facial evaluation is not the search for deviation from the norm of a single facial unit but the search for proportion (e.g. a face that is vertically excessive means that, in relation to the transverse dimension, the face is excessively long and not that it is longer than another face (Fig. 46.5). By increasing only the vertical dimension facial harmony is lost, but by increasing both transverse and vertical dimensions harmony is restored.

Most soft tissue discrepancies are indicators of underlying dental and/or skeletal deformities. Knowledge of the soft tissue problems and the relationship with the supporting hard tissue elements (dental and skeletal) will give the clinician a good idea of the orthodontic tooth movement and surgical repositioning of the jaw(s) required for correction of both the hard and soft tissue problems. Some key



Fig. 46.4 The facial division into oculonasal, maxillary, and mandibular complexes (the latter including the genial sub-complex).

soft tissue parameters used for an overview of facial esthetics and the interpretation and differentiations of the possible underlying deformities are listed below.

Profile view

Nasolabial angle

The angle is measured between the columella of the nose and the upper lip and should range from 85° – 105° (Fig. 46.6, Table 46.1).

Lip–chin–throat angle

The angle is formed between the lower lip, the lower border of the chin and the throat and should measure $110 \pm 5^{\circ}$ (Fig. 46.7, Table 46.2).

Chin–throat length

This is measured from the angle of the throat to the soft tissue menton (Fig. 46.8). A length of 42 ± 6 mm is considered to be normal. This measurement can only be meaningful with the patient's head in natural posture. This measurement is significant in assessing the severity of mandibular antero-posterior excess or



Fig. 46.5 The importance of the concept of facial proportion is illustrated by digitally modifying a face with manmade ideal proportions (Venus de Milo). The first image shows a harmonious balance between vertical and horizontal dimensions. Maintaining the transverse dimension but increasing the vertical results in loss of proportion. Increasing both re-establishes harmony. This emphasizes that facial esthetics are not a question of absolute values but more importantly of achieving proportion.

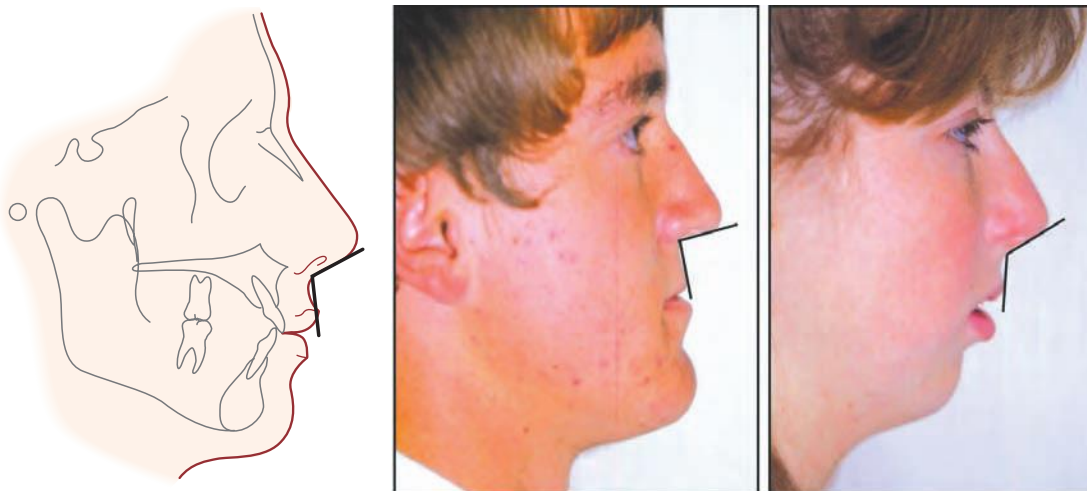


Fig. 46.6 The nasolabial angle is measured between the columella of the nose and the upper lip. The angle should be $90 \pm 10^{\circ}$ and is a guide to the upper lip support by the maxillary incisors. It is, however, also influenced by the decreased vertical dimension due to maxillary vertical deficiency.

Table 46.1 Nasolabial angle.

Acute angle	Obtuse angle
Upper incisor protrusion	Upper incisor upright or retro-inclined
Drooping nasal tip	Prominent or hanging columella
Class III occlusion	Class II occlusion
Deep bite	Open bite
Maxillary vertical deficiency	Maxillary vertical excess

Table 46.2 Lip–chin–throat angle.

Acute angle	Obtuse angle
Mandibular antero-posterior excess	Mandibular antero-posterior deficiency
Thin patient	Presence of submandibular adipose tissue
Class III occlusion	Class II occlusion

deficiency as well as differentiating between mandibular antero-posterior excess and maxillary antero-posterior deficiency (Table 46.3). For a patient with class III malocclusion and normal chin–throat length, maxillary deficiency should be suspected.

Upper lip length

The upper lip length is measured from subnasale to lower lip (stomion superius) and should be 20 ± 2 mm for females and 22 ± 2 mm for males (Fig. 46.9, Table 46.4). Ensure when evaluating the lips that they

Table 46.3 Chin–throat length.

Increased length	Decreased length
Mandibular antero-posterior excess	Mandibular antero-posterior deficiency
Class III occlusion	Class II occlusion
Macrogenia	Microgenia



Fig. 46.7 The lip–chin–throat angle is formed between the lower border of the chin and a line connecting the lower lip and soft tissue pogonion ($110\pm10^\circ$). It is most commonly acute in flat or concave profiles with class III dentoskeletal patterns. An obtuse angle is seen in class II malocclusion, mandibular antero-posterior deficiency, and is often exacerbated by microgenia.



Fig. 46.8 The chin–throat length. This is measured from the neck throat angle to the soft tissue pogonion and is pertinent when considering mandibular setback or advancement procedures, genioplasty, and submental liposuction.

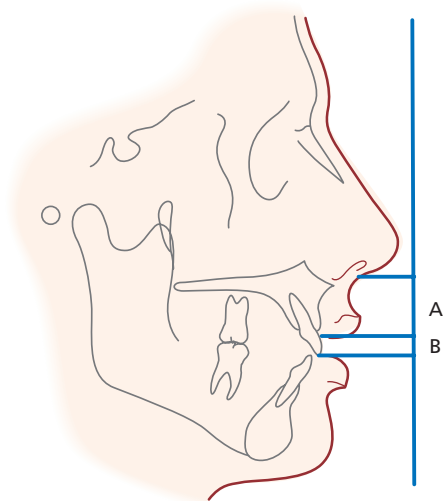


Fig. 46.9 A. The upper lip length is measured from subnasale to lower lip (stomion inferius). B. The interlabial gap is measured between stomion superius and stomion inferius. Critically the assessment should be in repose as patients may assume a lip position which may mask vertical excess. The clinician should guard against using this assessment as this measurement only reflects the central incisor exposure under the upper lip. The shape of the lip and the upper lateral incisor exposure cannot be judged on this view.

Table 46.4 Upper lip length.

Increased lip length	Decreased lip length
Less upper incisor exposure	Increased upper incisor exposure
Differentiate from vertical maxillary deficiency	Differentiate from vertical maxillary excess
Toothless look	Gummy smile
Adequate lip seal	Increased interlabial gap

are in repose. During treatment planning it should be kept in mind that the upper lip length will increase with age.

Interlabial gap

The interlabial gap should be assessed with the lips in repose and the teeth in occlusion. The distance between stomion superius and stomion inferius is measured and should be 0–4 mm (Fig. 46.9, Table 46.5). If the lips touch when the teeth are in occlusion the upper incisor–upper lip relationship should be assessed with the jaws slightly open and the lips in repose.

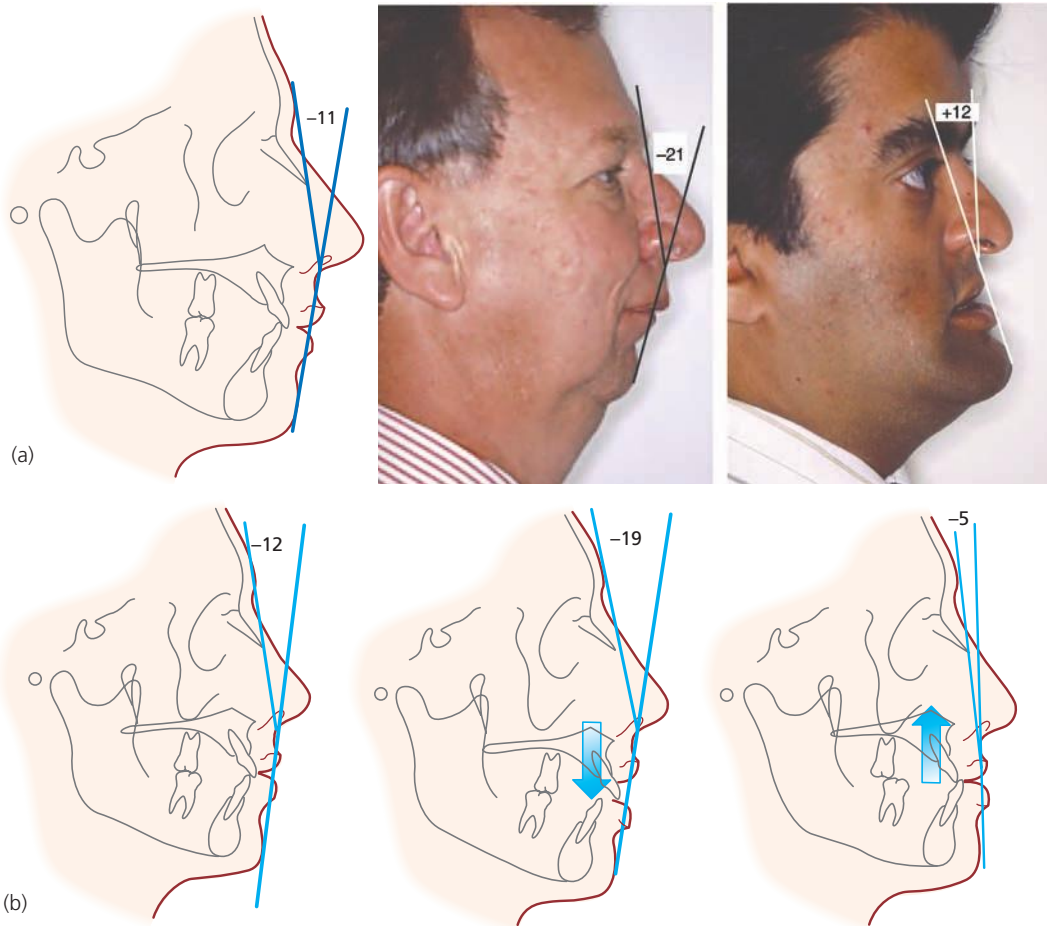


Fig. 46.10 (a) The facial contour angle is the angle between the upper facial plane (glabella–subnasale) and lower facial planes (subnasale–pogonion) and should be $-11 \pm 3^\circ$ for males and $-13 \pm 3^\circ$ for females. If measured above subnasale the figure is expressed as negative and below as positive. This measurement is an indication of the convexity or concavity of the face. An increased angle generally suggests a relative or absolute mandibular deficiency and a decreased angle a relative or absolute mandibular excess. Differentiating relative from absolute deviations from the norm requires an assessment of the vertical and antero-posterior position of the maxilla. (b) Vertical alteration of the maxillary height, superior repositioning or downgrafting of the maxilla, the mandible will autorotate either counterclockwise or clockwise at the condyle.

Table 46.5 Interlabial gap.

Increased interlabial gap	Decreased interlabial gap/over closed
Increased incisor exposure	Less incisor exposure. Assess with lips apart
Gummy smile	Less than the full incisor tooth crown exposed
Mouth breathing	Over closed appearance
Vertical maxillary excess	Vertical maxillary deficiency/deep bite
Short upper lip	Long upper lip
Excessive vermillion exposure	Less vermillion exposure

Table 46.6 Facial contour angle.

Increased facial contour angle	Decreased facial contour angle
Mandibular antero-posterior deficiency	Mandibular antero-posterior excess
Maxillary antero-posterior excess	Maxillary antero-posterior deficiency
Microgenia	Macrogenia
Vertical maxillary excess	Vertical maxillary deficiency
Class II occlusion	Class III occlusion

Facial contour angle

The angle between the upper facial plane (glabella–subnasale) and lower facial planes (subnasale–pogonion) is an indication of the convexity or concavity of the face. The angle, when measured above subnasale, is expressed as negative and should be $-13 \pm 2^\circ$ for females and $-11 \pm 2^\circ$ for males (Fig. 46.10a, Table 46.6). Although it is an indication of the antero-posterior relationship between the glabella, subnasale, and pogonion, the measurement is certainly influenced by the vertical dimensions of the facial structures. The mandible will autorotate around

the condyle as a result of vertical problems, i.e vertical maxillary excess, vertical maxillary deficiency, increase or decrease in posterior mandibular height, and open or deep bite, and thus the antero-posterior position of the pogonion will change (Fig. 46.10b).

Labiomental angle

The chin forms a very important part of the total facial form and harmony; as with all parts of the face, the chin should be evaluated in relation to all the aspects of the face and evaluated separately for harmony. The lower lip, labiomental fold, and the chin button should form a harmonious S-shaped curve and the depth of the labiomental fold should divide the chin (from stomion superius to soft tissue menton) in an upper third and lower two thirds. The labiomental angle is formed by the intersection of the lower lip and chin measured at the soft tissue B-point (Fig. 46.11). The angle should be gently curved (mean = $120 \pm 10^\circ$) (Table 46.7). Chin contour is just as important as chin position. It is therefore important to note that genioplasty should be performed to improve the shape and contour of the chin and not to essentially place the soft tissue pogonion on its ideal antero-posterior position.

Table 46.7 Labiomental angle

Acute angle	Obtuse angle
Increased overjet	Anterior crossbite
Class II occlusion	Class III occlusion
Macrogenia	Microgenia
Deep bite	Open bite
Lower incisor protrusion	Retroclined lower incisors
Vertical chin deficiency	Vertical chin excess

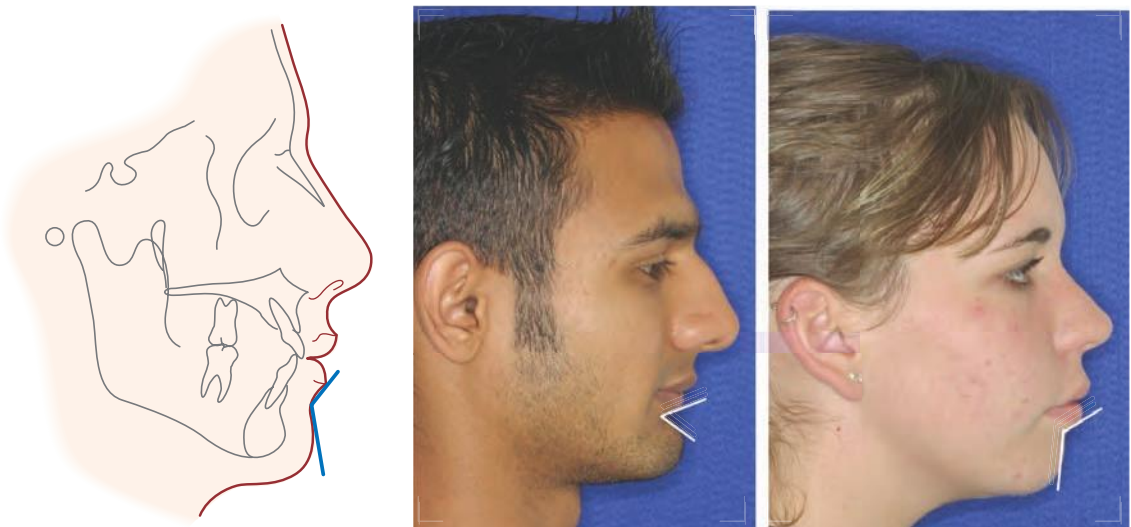


Fig. 46.11 The labiomental angle is formed by the intersection of the lower lip and the chin and is measured at soft tissue B-point. The angle should be gently curved (mean = $120 \pm 10^\circ$). Since both lower lip position and chin position can affect the labiomental angle, a deviation from ideal requires differentiation between lower lip and chin effects (or a combination of both).

Frontal view

Facial form

The relationship between the height and the width of the face has an important influence on facial form and harmony and should also be correlated with the patient's overall body build (i.e. short and stocky vs long and thin). The height-to-width proportions are 1.3:1 for females and 1.35:1 for males (Fig. 46.12, Table 46.8). The bigonial width should be approximately 30% less than the bizygomatic dimension and the width and shape of the chin should form a harmonious part of the overall facial contour.

Transverse dimensions

The normal interpupillary distance should be 65 ± 3 mm while the intercanthal distance should measure 32 ± 2 mm. Vertical lines drawn through the medial canthi should coincide with the ala of the nose while vertical lines drawn through the medial margins of the irides of the eyes should coincide with the corners of the mouth. For black people this distance would be slightly increased. The orbital dimensions cannot be changed unless craniofacial surgery is contemplated. The measurements will, however, give the clinician an indication of harmony between the nose, the mouth, and the eyes. The bigonial width should be

approximately 30% less than the bizygomatic width (Fig. 46.13a, b). The alar base width will be an important consideration as Le Fort I osteotomy, especially superior repositioning and advancement of the maxilla, may increase the width of the base of the nose. Surgical precautions should be taken to control unwanted nasal changes following Le Fort I osteotomy. The width of the nasal dorsum should be approximately half of the intercanthal width while the width of the nasal lobule should be two thirds of the intercanthal distance. The form and shape of the nose are not only important in the diagnosis and the possible

Table 46.8 Facial form.

Narrow face	Broad face
Long face	Short square-shaped face
Class II and/or open bite	Class II deep bite
Vertical maxillary excess	Vertical maxillary deficiency
Decreased intergonial distance	Masseter hyperplasia
Narrow nose	Broad nose
Microgenia (narrow chin)	Macrogenia (broad chin)
High mandibular plane angle	Low mandibular plane angle
High occlusal plane angle	Low occlusal plane angle



Fig. 46.12 Facial form is a guide to the vertical and transverse proportions of the face. (a) A leptoprosopic facial form. Since the transverse dimension of a face cannot be significantly altered it is pragmatic to assume that the long face has a vertical dimension too large for its transverse dimension. (b) A dolichoprosopic facial form. The short face has a vertical dimension too small for its transverse dimension.

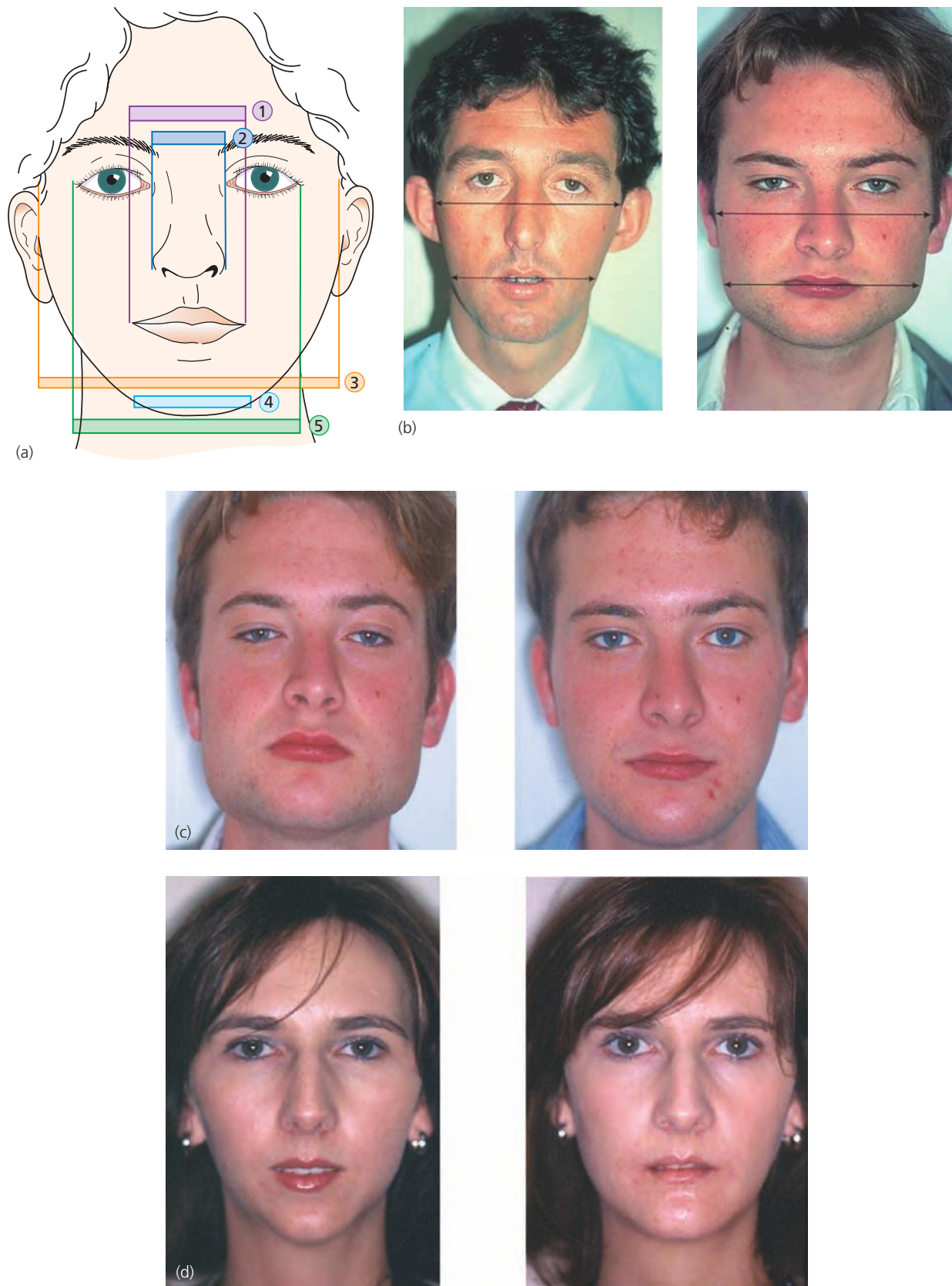


Fig. 46.13 (a) The transverse facial dimensions and facial form. (1) The width of the medial margins of the irides of the eyes should coincide with the corners of the mouth, (2) the medial canthal width should be equal to the alar base width, (3) the bigonial width should be 30% less than the (5) bizygomatic width, (4) the width of the chin should be in harmony with the rest of the face (female chins are in general narrower than the chins of males). (b) Left: a patient with a V-shaped face. The bigonial width is more than 30% less than the bizygomatic width. The patient on the right has a broad square face. The bigonial width and bizygomatic width are almost equal. (c) Left: a patient with a dolichoprosopic facial form as a result of bilateral masseter muscle hypertrophy. Right: the excessive width at the mandibular angles was reduced by partial osteotomy of the mandibular angles and reduction of the masseter muscles. (d) Left: a patient with a V-shaped facial form and a narrow chin. Right: the form of the face is changed to a more oval shape by widening the chin by means of a two-piece genioplasty.

surgical correction of a nasal deformity but consideration should also be given to negative as well as positive effects that orthognathic surgery may have on the nose. Abnormal interpupillary and intercanthal distance are often observed in syndromic patients and can only be altered by means of craniofacial surgery. The width of the midface can be altered by means of either onlay grafts on the zygomas or zygomatic osteotomies. The mandibular angle width can be reduced by means of surgical reduction of the angles and/or reduction of the masseter muscles (Fig. 46.13c), while this dimension can be increased by subperiosteal onlay implants. The transverse dimension of the chin (narrowing or widening) can be altered by a two-piece genioplasty (Fig. 46.13d). Enhancement of the transverse esthetics can be performed by differential change of the anterior and/or posterior width of the chin.⁴

Vertical evaluation

In the vertical dimension the face can be divided into three equal parts: (1) the upper third, from the hairline (trichion) to glabella; (2) the middle third, from glabella to subnasale; and (3) the lower third, from subnasale to menton (Fig. 46.14). Orthognathic surgery can generally influence the lower third and also have some effects to the middle third. However as mentioned previously it may be more useful to consider the subdivision of the face into forehead, oculonasal, and gnathic complexes to better describe the effects of maxillary and/or mandibular derangements.

Table 46.9 shows effects of increased and decreased middle third. In assessment of the lower third, lip thickness, the amount of vermillion exposure, and the severity of the cupid's bow should be noted (Table 46.10). Special attention should be given to the presence of a gummy smile as well as the symmetry of the smile. The amount of gingival exposure when

Table 46.9 Middle third.

Increased middle third	Decreased middle third
Vertical maxillary excess	Vertical maxillary deficiency
Sallow cheeks	Full cheeks
Excessive sclera show below the iris	Normal sclera show
Flat cheek bones	Prominent broad cheek bones
Narrow nose	Short, broad nose

Table 46.10 Lower third.

Increased lower third	Decreased lower third
Vertical maxillary excess	Vertical maxillary deficiency
Increased interlabial gap	Overclosed appearance
Excessive upper incisor exposure under the upper lip	Little or no upper incisor exposure under the upper lip
Flat paranasal areas	Fullness in the paranasal areas
Narrow nose	Short, broad nose
Narrow chin	Broad chin
Class II with or without anterior open bite	Class III closed bite or class II deep bite

smiling and the amount of upper incisor exposure under the upper lip in repose should be correlated. Ideally, the upper lip vermillion should fall at the cervicogingival margin with no more than 2 mm of gingival exposure during smiling. Keep in mind that, although there are several factors to be considered, the amount of incisor exposure or lack of exposure under the relaxed upper lip is the prime indicator of the amount of vertical reposition of the maxilla.

The upper lip vermillion should be about one half of the lower lip vermillion exposure while the depth of the labiomental fold falls about one third down from stomion superius to menton. The length of the upper lip forms one third of the lower third; that is, the lower lip stomion to menton is twice the length of a normal upper lip.

Facial symmetry

The symmetry of the facial midline structures such as the forehead (glabella), nasal tip, upper lip, maxillary dental midline, mandibular dental midline, lower lip, and chin should be assessed (Fig. 46.15a). The clinician should consider whether orthodontic or surgical correction of the dental midlines should be planned. The left-to-right symmetry must be correlated with the facial midline and the face should be reasonably symmetric vertically and transversely. Keep in mind that no face is perfectly symmetric. When evaluating the symmetry of the face it is also very important to assess the presence of any occlusal plane cants. The occlusal plane should be parallel to the interpupillary line, provided that there is no orbital distopia. Surgical correction of an occlusal plane cant often facilitates correction of the asymmetry of the face and the

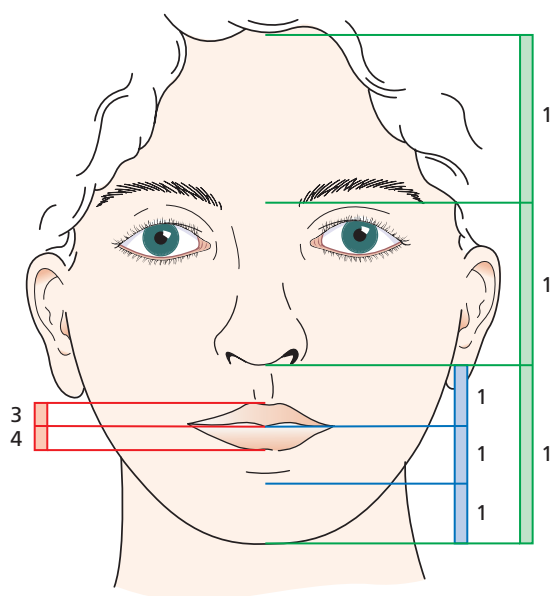


Fig. 46.14 The vertical assessment of the face for ideal esthetics requires achievement of the proportions set out in the figure.

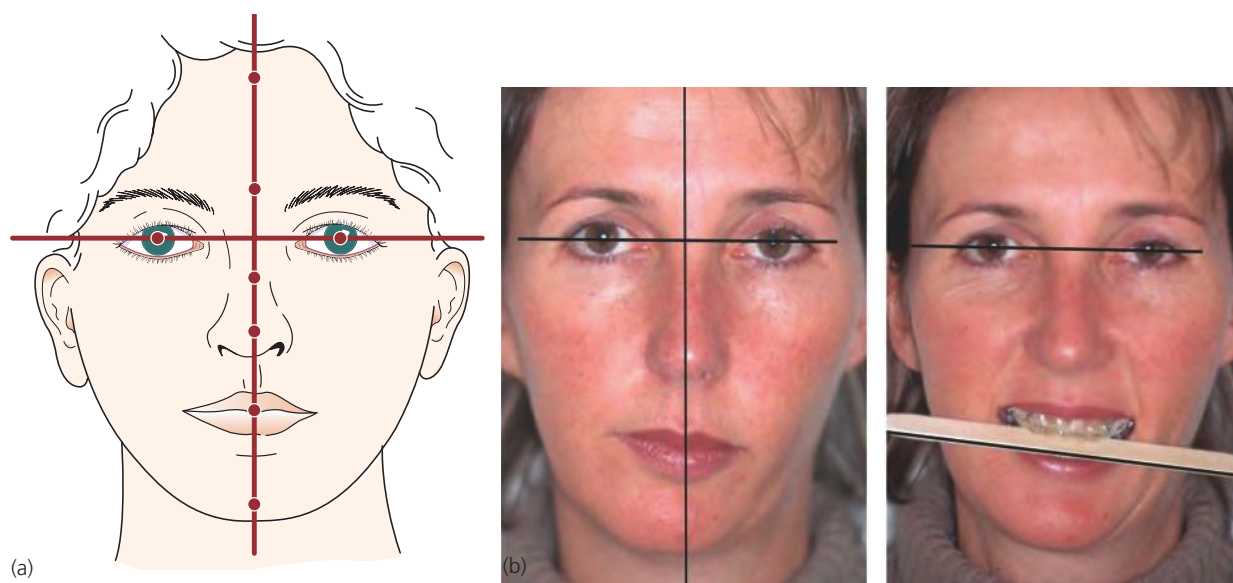


Fig. 46.15 (a) Facial symmetry is assessed by comparing the left and right sides of the face obtained by a line perpendicular to and passing through the midpoint of the interpupillary line. (b) In addition the parallelism of the interpupillary line and the occlusal plane should be assessed.

severity of the cant should be correlated with the dental and the facial asymmetry (Fig. 46.15b). The vertical discrepancy between the left and right side of the occlusal cant should be noted and correlated with the vertical position of the upper incisor tooth, as correction of the cant will influence the incisor lip relationship.

Intraoral examination

General dental evaluation

The dental history is often a good indicator of the patient's motivation and commitment to future treatment. Previous restorative, periodontal, and orthodontic treatment should be reviewed. Caries, periodontal and periapical pathology, and the presence of impacted teeth should be noted. It is preferred that impacted teeth be removed prior to commencement of definitive treatment or at least 9 months before surgery. When a sagittal split ramus osteotomy is performed, the presence of an impacted tooth influences the difficulty of the surgery and increases the complication rate.⁵ Missing teeth and the need for implants should be evaluated and integrated into the treatment plan. Final prosthodontic treatment is deferred until after completion of orthognathic treatment.

Periodontal considerations

Patients with pre-existing periodontal disease or gingival pathology have an increased risk of disease exacerbation during orthodontic treatment. The amount of keratinized epithelium is important for decompensation of lower incisors in class III cases, and also in interdental osteotomy areas, subapical osteotomies, and genioplasty procedures. Periodontal

disease and inadequately attached gingiva, especially in the lower incisor areas, must be addressed before commencement of treatment. Long-term management, further treatment, and prognosis should be discussed with the periodontist and the patient.

Careful orthognathic surgical techniques must be applied to protect the periodontium and minimize vascular compromise to the bone, gingiva, and teeth. Bone should be maintained around the necks of teeth adjacent to interdental osteotomies and excessive stretching of soft tissue in these areas should be minimized. Poor surgical technique and lack of attention to risk factors may result in devastating periodontal complications.

Occlusal oral function evaluation

Abnormal mastication, swallowing, speech, and mouth breathing should be assessed and noted. The effect the dentofacial deformity may have on normal speech should be evaluated and the patient referred for pretreatment speech evaluation when indicated. Velopharyngeal incompetence and the effect orthognathic surgery may have on the function of the soft palate should be assessed. Thumb sucking, tongue thrust, and lip biting habits should be noted and the effects of these habits evaluated. The size of the tongue and the presence of crenations on the sides of the tongue should be noted and the possibility of macroglossia as an etiological factor of an open bite should be considered.⁶

Temporomandibular joint evaluation

The temporomandibular joints (TMJs) provide the basis for orthognathic surgery. The status of the TMJs should therefore be carefully assessed before treatment, as well as periodically during treatment.

Pretreatment joint dysfunction or undiagnosed joint pathosis can exacerbate postoperative joint problems, and result in pain, limited movement, condylar resorption, joint dysfunction, malocclusion, and relapse. Patients with dentofacial deformities may present in three categories: (1) patients with dentofacial deformities and subjective complaints about concomitant TMJ dysfunction; (2) patients with dentofacial deformities with only signs of TMJ problems; (3) patients with a primary complaint of TMJ dysfunction but with a concomitant dentofacial deformity that is of no concern to the patient.

Systemic conditions that may affect the TMJs as part of a polyarthritic condition, such as rheumatoid arthritis, systemic lupus erythematosus arthritis, ankylosing spondylitis, or scleroderma, should be identified and ruled out.

Pretreatment clinical examination should carefully assess pain, joint noises, function, and maximum mouth opening. Joint pain and neuromuscular pain should be distinguished. Deviation of the mandible during opening may indicate closed lock or fibrous ankylosis. Popping or clicking may indicate disc displacement while crepitations may indicate osteoarthritis, perforation of the disc, or retrodiscal tissue pathology.

Existing joint problems should be diagnosed, documented, and discussed with the patient. Conditions requiring treatment should be carefully planned and sequenced. Generally the joint dysfunction should be first treated conservatively followed by orthodontic alignment of the teeth; the orthognathic surgery is then performed and the joint is re-evaluated. For patients with a history of joint dysfunction, special care should be taken during surgery when the condyles are positioned. If surgery is indicated as treatment for a joint problem the surgery should be performed before orthodontic treatment and orthognathic surgery. Some clinicians, however, prefer to perform joint surgery at the same time as orthognathic surgery.⁷

Condylar resorption should be suspected in patients presenting with a history of having a good occlusion which then slowly deteriorated into a class II anterior open bite malocclusion. Idiopathic condylar resorption is a condition that may develop in young female patients during puberty and growth spurt, with no pain or limitation of jaw movement. The resorption is a progressive process, however it usually stabilizes and the condyle may resorb down to the coronoid notch. Various treatment modalities have been suggested: limiting the compression forces with an “unloading” splint, muscle relaxants, and/or anti-inflammatory medication;⁸ removal of the remaining condyle and costochondral graft reconstruction;⁹ wait for the resorption to stabilize followed by orthognathic correction – Le Fort I maxillary osteotomy, sagittal split mandibular ramus osteotomy, genioplasty;¹⁰ wait for resorption to stabilize followed by distraction.¹¹

The nose

The nose is an important part of the overall facial esthetics and the form and function of the nose can certainly be affected by orthognathic surgery. In many cases nasal reconstruction may form part of the orthognathic treatment plan and, in some cases, nasal reconstruction can be performed concurrent with the orthognathic surgery. Control of the nasal form should also be considered, especially in cases requiring superior repositioning and/or advancement of the maxilla.

The functional and esthetic nasal evaluation should include a thorough examination of the external and internal nasal structures (Fig. 46.16). An intranasal examination should be performed to identify a possible existing deviated nasal septum, hypertrophied turbinates, or nasal polyps. Esthetic concerns should be noted and the nose should be evaluated from a frontal and profile view.

A discussion of detailed esthetic parameters of the nose falls beyond the scope of the text. However, important esthetic factors to consider are the width of the nasal base, and the distance from the base of the nose to the anterior extent of the nares and from the anterior aspect of the nares to the tip of the nose. The prominence of the dorsum, the shape of the nasal tip as well as the acuteness of the supra tip break must also be considered in relation to the intended orthognathic surgery and the physical and/or relative esthetic effects surgery may have on these structures. The length of the columella, the nasolabial angle, and

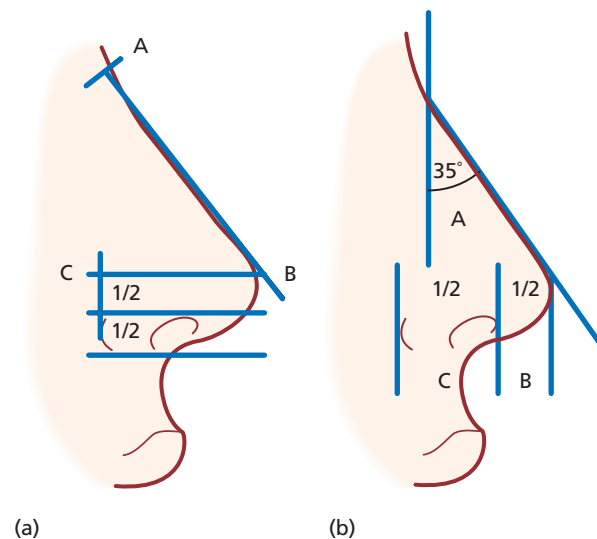


Fig. 46.16 (a) The nasal projection is evaluated by Goode's method. If BC is greater than 60% of AB, the nasal tip usually appears overprojected. The nasal tip-ala-columella relationship should be a one to one relationship and is an indication of a hanging or retracted columella or a retracted ala. (b) The nasal projection can be evaluated by measuring the angle of the nasal dorsum to a vertical line. The angle should be 34° for females and 36° for males. The relationship between the nasal tip-columella lobule-alar base (1:2) will help to distinguish between paranasal flattening (maxillary antero-posterior deficiency) or overprojection of the nasal tip.

the projection and shape of the nares should be considered, as these aspects may be negatively affected by maxillary surgery.

Radiographic evaluation

The radiographs mostly used as routine in the diagnosis of dentofacial deformities are lateral and postero-anterior cephalometric radiographs, panoramic radiographs, and periapical radiographs. Other radiographic imaging modalities, such as TMJ tomograms, magnetic resonance imaging (MRI), and computed tomography (CT) scans, may be required as determined by the needs of each individual case.

Although clinical evaluation must be the primary diagnostic tool in determining surgical treatment of the orthognathic patient, cephalometric analysis is a helpful diagnostic guide. It helps the clinician: to quantify, classify, and communicate; design a treatment plan by the development of an orthodontic and surgical visual treatment objective; plan for tooth extractions; monitor progress during treatment; study short- and long-term treatment changes; evaluate treatment results and study facial growth.

The cephalometric radiograph should be taken with the patient's head in natural posture, the teeth in centric occlusion, and the lips in repose. There are, however, three exceptions to this rule:

1. In patients with vertical maxillary deficiency the radiograph should be taken with the patient's teeth apart with the mandible rotated open until the lips just part. This view will allow the clinician to evaluate more accurately the upper lip length, the shape of the lip, and the upper incisor–upper lip relationship. It will also assist in planning the correct amount of maxillary down graft required for the case.
2. In patients with severe overclosed bites it is difficult to assess the tooth–lip relationship, the amount of vermilion exposure, and the shape of the lips with the teeth in occlusion. A second radiograph should be taken with the mandible rotated open until the lips just part.
3. Where there is a significant difference between centric occlusion and centric relation, a second radiograph should be taken with the teeth in centric relation.

There exists a myriad of cephalometric analysis, which involves measuring, comparing, and relating various linear and angular measurements of the dental, skeletal, and soft tissues of the face.^{12,13} The clinician should however guard against “playing the number game”. Keep in mind that one is not treating radiographs and the primary goal of treatment should be to make the facial appearance harmonious and to establish a functional occlusion rather than to make the cephalometric measurements normal. It should be emphasized that the primary soft tissue examina-

tion of the profile should be done clinically, guided by cephalometric analytic values.

Diagnosis and treatment planning

The information gathered from the systematic patient evaluation forms a database that will allow the clinician to list all the existing problems. The diagnostic list will include all functional, dental, skeletal, esthetic, and medical problems that may influence the treatment plan. The basic diagnosis should, however, simply describe the case for visualization, communication, and understanding, i.e. class II division 1, mandibular-deficient case or class III anterior open bite, mandibular-excess case.

Careful assessment and consideration should be given to the esthetic concerns, skeletal imbalances, occlusal problems, TMJ and/or myofascial pain, etc., to formulate a treatment plan. It is important to differentiate between various dentofacial deformities that can have similar patterns. For example, a patient with a class II malocclusion may have mandibular antero-posterior deficiency, or maxillary antero-posterior excess, or vertical maxillary excess with clockwise rotation of the mandible, or any combination of the above.

Lateral cephalometric analysis

There is a myriad of cephalometric analysis described in the literature, however the authors use only a selected group of analyses that allows for rapid diagnosis. The clinical soft tissue analysis as discussed previously can be confirmed by analyzing and measuring the soft tissue on the lateral cephalometric radiograph. The hard tissue landmarks on the lateral cephalometric radiograph should be identified and marked and facial planes reconstructed.

Hard tissue landmarks are as follows (Fig. 46.17):

- Nasion (N): the most anterior point on the frontal nasal suture in the midsagittal plane.

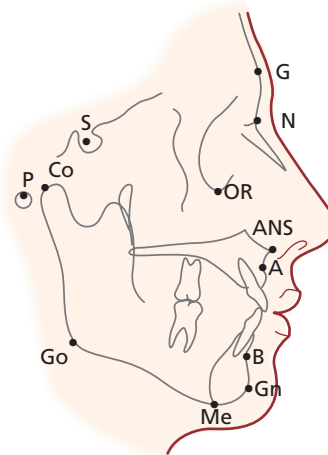


Fig. 46.17 Hard tissue cephalometric landmarks

- Orbitale (OR): the lowest point on the inferior orbital rim.
- Sella (S): the center of the sella turcica, as on the lateral cephalogram, which is located by inspection.
- Anterior nasal spine (ANS): anterior tip of the nasal spine.
- A-point (A): the most posterior midline point in the concavity where the lower anterior edge of the anterior nasal spine meets the alveolar bone overlying the maxillary incisor teeth.
- B-point (B): the most posterior midline point in the concavity of the mandible between the alveolar bone overlying the lower incisor teeth and the pogonion.
- Gonion (Go): the point is defined by using two lines, one tangent to the posterior border of the mandibular ramus and the other tangent to the lower border of the mandibular corpus; found by bisecting the angle formed by the two lines and extending the bisector through the curvature of the mandibular angle.
- Menton (Me): the most inferior point on the symphysis of the mandible in the midline.
- Porion (P): the most superior point of the external auditory meatus (anatomic point); machine porion is the uppermost point on the outline of the rods of the cephalometer.
- Condylion (Co): the most postero-superior point on the head of the condyle.
- Gnathion (Gn): the lowest, most anterior midpoint on the symphysis of the mandible.
- Glabella (G): the most anterior point on the frontal bone.

The following constructed hard tissue facial planes are mostly used (Fig. 46.18):

- Frankfort horizontal plane (FH): extends from porion to orbitale.
- Anterior cranial base (SN): formed by a line drawn from sella to nasion.

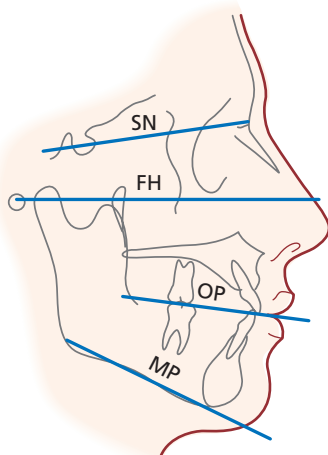


Fig. 46.18 Hard tissue cephalometric planes.

- Occlusal plane (OP): formed by a line drawn through the mesial cusp contact of the first molar teeth and dividing the incisor overbite.
- Mandibular plane (MP): extends from gonion to menton.

Because the anatomic Frankfort plane (FH) does not always correlate with the clinical impression of the patient's facial deformity, the authors prefer to use the anterior cranial base (SN) as a horizontal reference line.

Skeletal antero-posterior relationships

- Maxillary antero-posterior position (Steiner) (Fig. 46.19). The analysis gives an indication of the antero-posterior position of the maxilla in relation to the anterior cranial base. The angle between the anterior cranial base (SN) and a line drawn between the nasion (N) and A-point is measured and should be 82° for a normal maxilla.
- Maxillary depth angle (McNamara) (Fig. 46.19). The angle between the FH and NA also gives indication of the maxillary antero-posterior position and should be 90° . A line perpendicular to FH should therefore fall on A-point.
- Mandibular antero-posterior position (Steiner) (Fig. 46.19). The ANB angle is measured between SN and a line drawn between N and B-point and it relates the antero-posterior position of the mandible to SN. This angle should be 80° for a normal mandibular position.
- ANB angle (Steiner). This angle gives the clinician an indication of the inter-relationship between the upper and lower jaw. In class II mandibular-deficient cases the angle will be increased while in class III cases the angle will be decreased. An angle of 2° indicates a normal relationship.

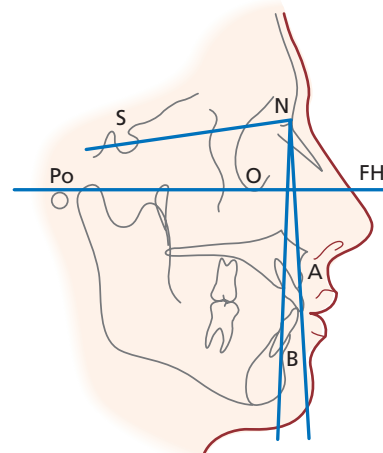


Fig. 46.19 The Steiner analysis provides clues as to the horizontal relationship of the maxilla and mandible (ANB angle). The relationship of the jaws to the cranial base is often a less valuable figure as the dimension is affected by the angle of the cranial base.

- Wits appraisal (Fig. 46.20). The Wits appraisal is a linear measurement between the maxilla and the mandible and is not influenced by the anterior cranium. Perpendicular lines are dropped from A-point and B-point, respectively, on to the occlusal plane (OP). The mean measurement in males is BO 1 mm ahead of AO and in females the lines should coincide.

Vertical skeletal relations

The relationship between the skeletal height of the midface and lower face is more important than the actual measurements. The skeletal height of the midface is measured from N to ANS while the lower skeletal height is measured from ANS to the hard tissue menton (Me) (Fig. 46.21). A vertical line is drawn anterior to the face perpendicular to FH. Lines from N, ANS, and Me are constructed perpendicular to the vertical line. The midface and lower facial heights are measured on the line. Normal values should be 53 mm from N to ANS and 65 mm from ANS to Me. The relationship between the two heights is, however, more

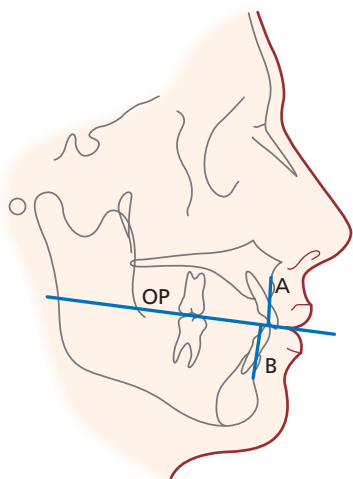


Fig. 46.20 The Wits appraisal provides a linear analysis of the horizontal relationship between maxilla and mandible only (i.e. not to the rest of the face).

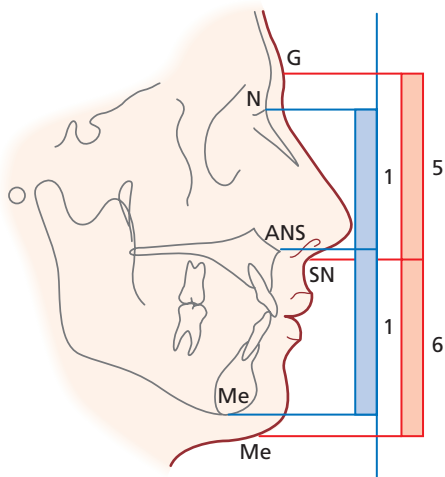


Fig. 46.21 The vertical proportions in a profile view.

significant than the actual measurements, and should be 5:6. The ANS–Me measurement will be relatively increased in individuals with vertical maxillary excess, vertical mandibular excess, and anterior open bite. The ANS–Me distance will be relatively small in individuals with maxillary vertical deficiency, mandibular vertical deficiency, and deep or closed bites.

Analysis of dental relations

- Upper incisor angulation (Fig. 46.22). According to the Steiner analysis the long axis of the upper incisor tooth should make an angle of $22 \pm 2^\circ$ with the NA line. The labial surface of the incisor tip should be 4 ± 2 mm ahead of the NA line. This measurement is an important presurgical orthodontic indicator.
- Lower incisor angulation (Fig. 46.23). The angle between the long axis of the lower incisor tooth and the NB line should be $20 \pm 2^\circ$ and the labial surface of the tooth should be 4 ± 2 mm ahead of the line. The lower incisor can also be related to the mandibular plane and should form an angulation of $90 \pm 5^\circ$ with the MP. This angle gives the clinician an idea of the amount of dental compensation for skeletal discrepancy present.
- Mandibular plane angle (Steiner) (Fig. 46.24). The mandibular plane is constructed by a line connecting the gonion (Go) and the menton (Me). The angle between the mandibular plane and SN should be 32° . Individuals with high mandibular plane angle would tend to have class II malocclusions, vertical maxillary excess, and anterior open bites. Patients with low mandibular plane angle will tend to have deep bites and may be vertically deficient with overclosed bites.

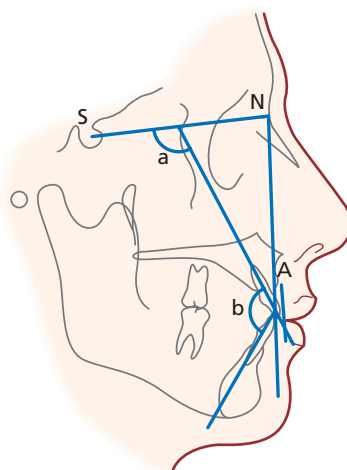


Fig. 46.22 (a) The upper incisor angulation can be referenced against the lower incisor ($131 \pm 5^\circ$), and (b) the base of the skull (106°) to give an idea of incisor pro- or retroclination. In general, however, the most important determinant of upper incisor angulation should be alveolar angulation and to ensure the tooth is in the central trough of bone. The labial surface of the upper incisor should be 4 mm ahead of the NA line.

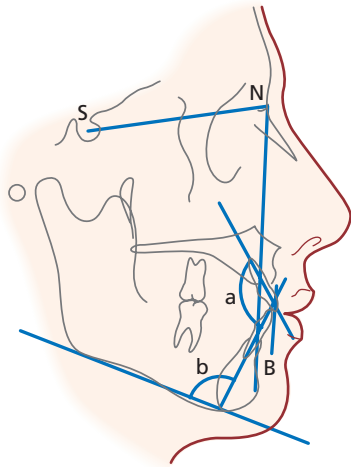


Fig. 46.23 (a) The lower incisor angulation can be referenced against the upper incisor ($131 \pm 5^\circ$), and (b), the mandibular plane ($90 \pm 5^\circ$) to give an idea of incisor pro- or retroclination. Due to the extreme narrowness of the lower anterior alveolus, especially in class III cases, great care must be taken when considering correction of incisor angulations. The alveolar angulation should form the basis for determining where the lower incisor can be placed. The labial surface of the lower incisor should be 4mm ahead of the NB line.

- Occlusal plane angle (Fig. 46.24). The occlusal plane angle is formed between a line drawn through the region of the overlapping cusps of the first bicuspids and first molars bisecting the incisal overbite (occlusal plane) and the anterior cranial base. The normal value for this angle is 14° . The occlusal plane has significant influence on function, esthetics, and treatment planning for double jaw surgery.

Postero-anterior cephalometric analysis

This analysis should be done in combination with the lateral cephalometric analysis for patients with facial asymmetry. The authors find the analysis according to transverse planes and the triangular analysis helpful when assessing individuals with skeletal facial

asymmetry (Fig. 46.25).¹⁴ The hard tissue landmarks on the postero-anterior cephalometric radiograph should be identified and include the following:

- SF: the point where the smaller wing of the sphenoid crosses the medial orbital ridge.
- Anterior nasal spine (ANS): the center point at the base of the nose.
- Jugulare (J): the most superior and medial point on the zygomatic buttress.
- Mastoid (M): the most inferior point on the mastoid bone.
- A: the contact point between the upper incisors.
- B: the contact point between the lower incisors.
- Y: the most lateral point on the buccal surface of the upper first molar. L and R will designate left and right sides.
- Z: the most lateral point on the buccal surface of the lower first molar. The left and right sides will be indicated by L or R.

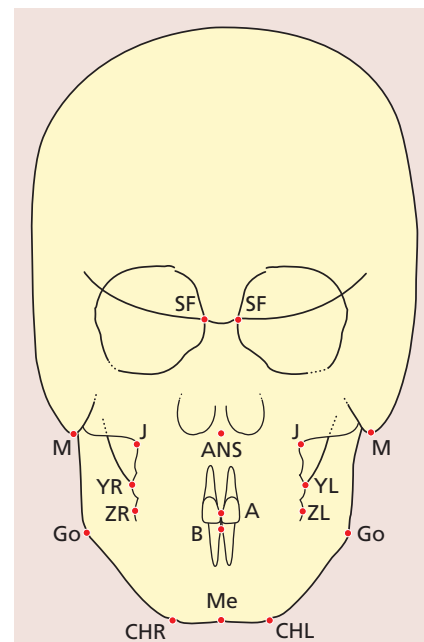


Fig. 46.25 Postero-anterior cephalometric landmarks.

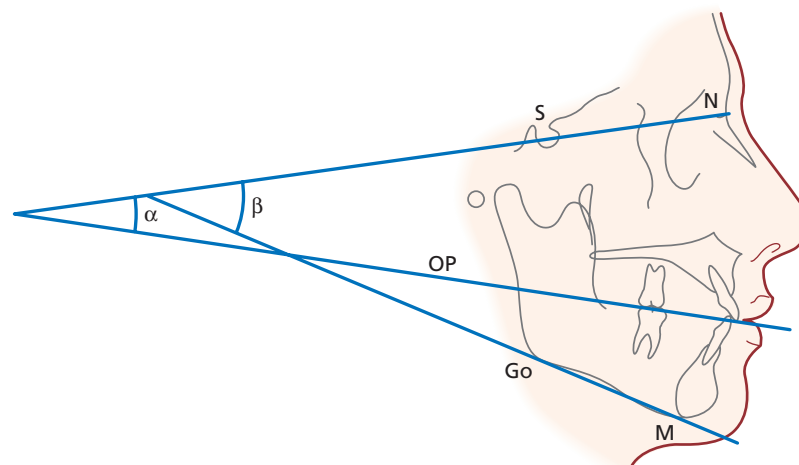


Fig. 46.24 The occlusal plane angle (α) and mandibular plane angle (β).

- Gonion (Go): the most inferior posterior point on the mandibular angle.
- Menton (Me): the most inferior point in the center of the mandibular symphysis.
- CH: the most inferior lateral point on the anterior inferior border of the mandible. The left and right sides will be indicated by L and R.

Transverse planes

These planes indicate transverse skeletal and occlusal plane cants (Fig. 46.26):

1. Cranial base plane (C plane): connect the left and right CF points and extend laterally.
2. Mastoid plane: connect the left and right M points.
3. S plane: the left and right Go points are connected.
4. J plane: connect the left and right J points.
5. Occlusal plane (OP): this plane is formed by a line drawn connecting the left and right occluding points of the first molars.
6. Chin plane (CHP): the plane is formed by connecting left and right Me points.

Vertical cephalometric midline

This line is constructed by dividing the C plane and mastoid plane and connecting the two points then extending the line to the chin (Fig. 46.26). The line is called the geometrically constructed vertical axis (GM).

Triangular analysis

Triangles can be constructed to evaluate the symmetry of the maxilla, mandible and chin (Fig. 46.27).¹⁵

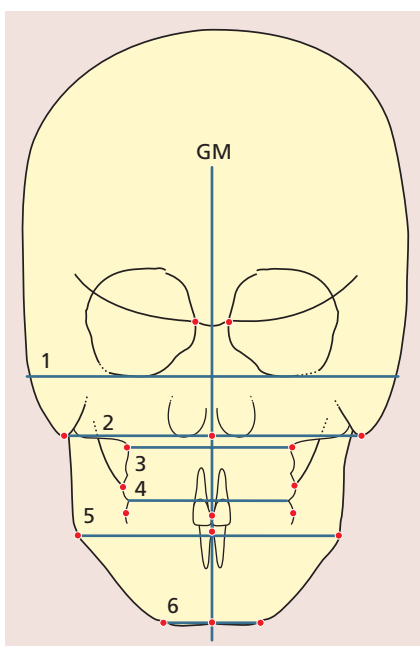


Fig. 46.26 Postero-anterior cephalometric planes.

1. Maxillary triangle: this triangle is constructed by connecting the midpoint of the C plane with J points on either side.
2. Mandibular triangle: the mandibular triangle is formed by connecting the C point with the left and right Go points.
3. Chin triangle: connect C point with CHL and CHR.

By measuring the long legs of the triangles, the cants of the maxilla, mandible, and the chin can be related to the cranial base. Also by comparing the left and right sides of the bases of the triangles, transverse discrepancies can be assessed. Midline asymmetries of structures such as ANS, menton, and dental midlines should be evaluated in relation to GM. The relationship between the dental and skeletal midlines will indicate if dental midlines should be corrected orthodontically or surgically.

Dental model analysis

Careful analysis of dental models will assist in diagnosis, and help to establish a problem list and develop an orthodontic and surgical treatment plan. Handheld models will also improve the clinician's understanding regarding transverse discrepancies between the upper and lower dental arches and differentiation between relative and absolute crossbites. There are several aspects of dental model analysis that should be considered:

1. Missing teeth (congenital or extracted), broken down or crowned teeth. This information will assist in the decision about the extraction pattern and the mode of space closure, whether orthodon-

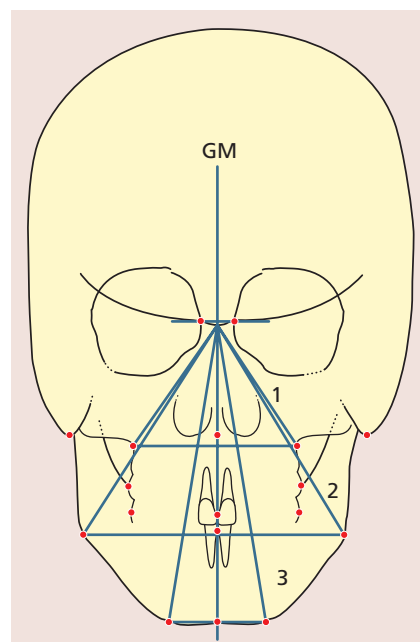


Fig. 46.27 Postero-anterior diagnostic triangles: (1) the maxillary triangle, (2) the mandibular triangle, and (3) the chin triangle.

- tically, surgically, or later by placement of an implant.
2. Occlusal curves in the upper and/or lower dental arches can be more accurately assessed on dental models. Orthodontic leveling of the curve of Spee in the lower arch will require 1mm of antero-posterior space for every 1mm of leveling of the curve. It should be kept in mind that when leveling occlusal curves is done by intruding or extruding teeth more than 2mm the result may not be stable. Careful assessment of the occlusal curves is significant to decide whether extractions are required and whether the curve should be leveled orthodontically or surgically (Fig. 46.28).
 3. Tooth sizes and arch length. Crowding or spacing can be assessed on the models by measuring the arch length and comparing it to the total amount of tooth width.
 4. Tooth mass discrepancies. Tooth mass discrepancies are not uncommon in patients with dentofacial deformities and are often the result of congenitally absent or small upper lateral incisors. In these cases it is problematic to establish an occlusion that will meet all the objectives of an ideal class I dental relationship. Bolton's analysis is a method to correlate the sizes of the six upper incisor teeth with the sizes of the lower six incisor teeth. When the combined width of the lower incisors is divided by the combined width of the upper incisors and

multiplied by 100 it should yield a value of 77.2%. When the combined width of the upper incisors is divided by the lower incisor width it should yield a value of 1.3 and is called the Bolton's intermaxillary index. When the Bolton's index deviates from the normal ratio it may be necessary to either reduce the width of the lower incisors or to leave spaces distal to the upper lateral incisors. The spaces can be closed by means of restorative dentistry. In severe cases, with a discrepancy of more than 5mm, extraction of a lower incisor may be considered. Tooth size discrepancies may also occur between the posterior teeth. For Bolton's tooth ratio analysis the sum of the widths of the 12 mandibular teeth is divided by the sum of the 12 maxillary teeth and multiplied by 100. A value of 91.3% will result in optimal posterior occlusion and incisor overjet and overbite. If the value is greater than 91.3%, the problem is usually as a result of excessive mandibular tooth mass.

5. Tooth-bone relationship. The angulation and position of the upper and lower incisors in relation to the bony base should be assessed. This will determine whether tooth extractions are indicated. In cases where the maxillary arch is narrow and may need expansion, the relationship of the molar teeth and the bone will assist in the decision to either expand the arch orthodontically or surgically.
6. Dental arch form and symmetry. A problem with symmetry usually occurs when a tooth is missing on one side and often results in dental midline asymmetry. Dental arch form is important for arch compatibility and will either require special orthodontic mechanics, asymmetric tooth extraction, or additional surgical procedures.
7. Buccal tooth tipping (curve of Wilson). The curve of Wilson is often accentuated in class III cases with the upper molars buccally inclined and the lower molars lingually inclined. It is not possible to achieve an acceptable occlusion during surgery with the occlusal surfaces of the posterior teeth tipped buccally. Buccal tipping in cases with maxillary transverse deficiency increases the amount of skeletal expansion (Fig. 46.29).

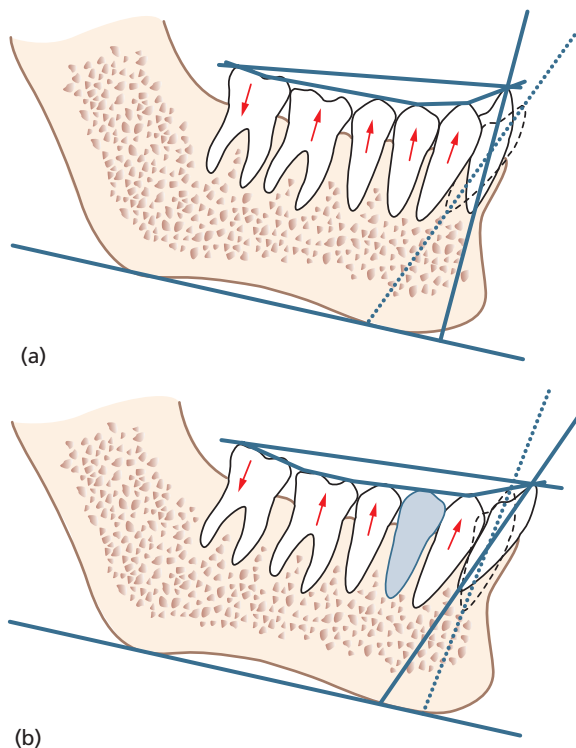


Fig. 46.28 (a) An accentuated curve of Spee is present and the lower incisor teeth are upright. Leveling the occlusal curve will increase the arch length and procline the lower incisors. In this case the bony base will accommodate all the teeth. (b) The lower incisors are proclined in combination with an accentuated curve of Spee. Orthodontic leveling of the arch will procline the incisors excessively. Extractions are indicated to accommodate all the teeth.

Orthodontic principles in orthognathic surgery

Most people with irregular teeth and jaw deformities usually seek treatment from an orthodontist and it is the orthodontist's task to introduce the patient to the treatment principles and the need for surgery as part of the treatment plan. The final pretreatment consultation takes place only after a systematic patient evaluation has been conducted and the orthodontist and surgeon have agreed on a final treatment plan. The clinician should be confident, explanations should be kept simple, and the patient's radiographs and dental casts should be used to demonstrate the problems.

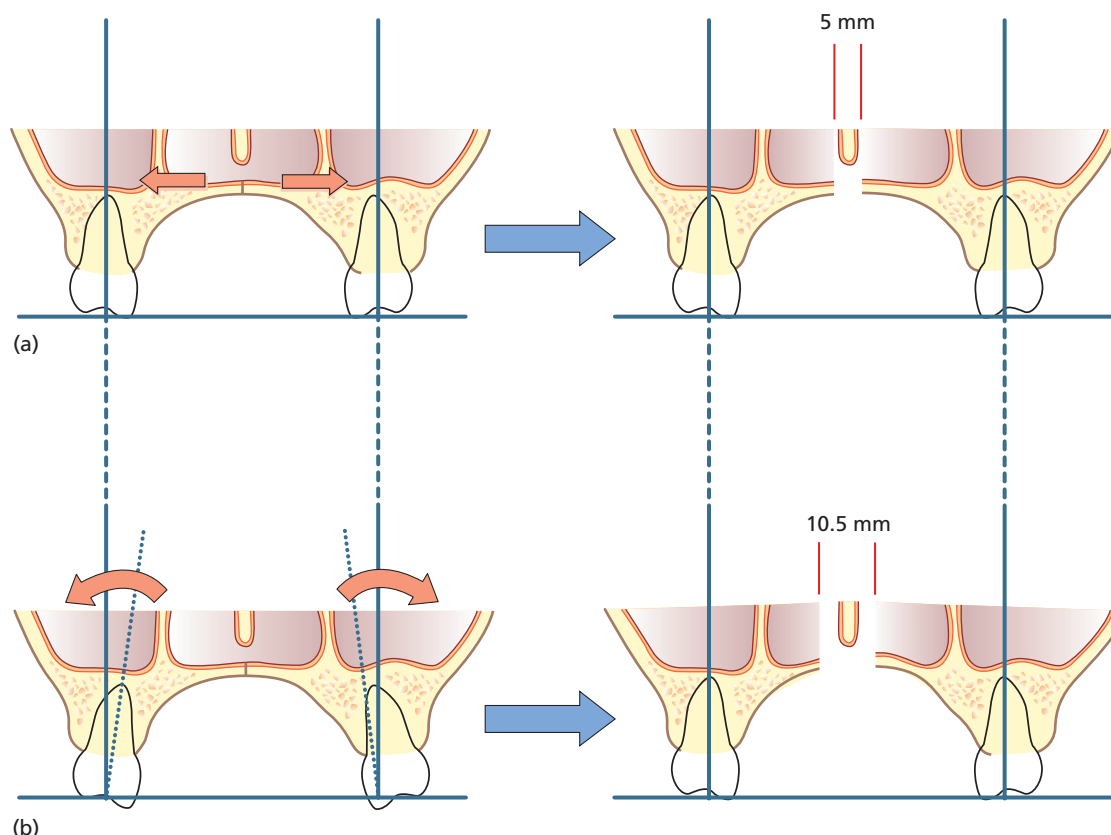


Fig. 46.29 (a) The molar teeth are placed in the center of the bone with normal angulation. Surgical expansion requiring 5 mm increase of the arch width will require 5 mm bony expansion. (b) An accentuated curve of Wilson is present and the molars are tipped buccally. In this case a 5 mm increase of dental arch width will require a 10.5 mm bony expansion.

Treated cases with similar problems can be used to demonstrate the treatment objectives and simple diagrams of the intended surgical procedure should be used. Pictures of actual surgery should be avoided. More detailed information and explanations regarding the surgery should be left to the surgeon.

It is mandatory that the patient (and family) be well informed because, as a general rule, a well informed patient usually follows instructions and is easy to treat. The authors explain to patients that a typical treatment profile consists of six stages:

- *First stage:* placement of orthodontic bands. Before placement of the bands all necessary extractions including third molars should be done.
- *Second stage:* presurgical orthodontic treatment. The teeth will now be aligned according to the treatment plan. Once the orthodontist is happy that the presurgical orthodontic treatment objectives have been achieved the objectives are checked by means of a cephalometric radiograph and cast models. The patient is then referred back to the surgeon. This treatment stage usually lasts an average of 9–18 months.
- *Third stage:* surgical stage. The surgical repositioning of the jaw(s) is now performed. After a short healing time of 2–3 weeks the patient returns to the orthodontist. Although the orthodontist will not actively start with orthodontic tooth movement it is important to: (1) evaluate the postoperative

occlusion and communicate any concerns to the surgeon; (2) consider postoperative orthodontic control of the surgical movements.

- *Fourth stage:* postoperative orthodontic treatment to perfect the bite. This stage of the treatment should take approximately 3–6 months. If this phase takes longer than 6 months the clinician should re-evaluate the treatment plan and achievement to date and assess problems. Patients, in general, become very impatient and their enthusiasm for further orthodontic treatment dwindles following surgery. It is for this reason that the authors recommend that, whenever possible, most orthodontic treatment should be performed before surgery.
- *Fifth stage:* removal of orthodontic bands.
- *Sixth stage:* retention stage (6–12 months). Following orthodontic tooth movement and surgical repositioning of the jaws the teeth need to be stabilized in their new positions for a time. The orthodontist manufactures and fits a retention appliance, which must be worn by the patient as instructed by the orthodontist. The patient should be seen by the clinicians for at least 1 year following band removal to monitor dental and skeletal stability and any unfavorable changes should be recorded.

It is important that the clinicians communicate with each other following each stage, often with a short note confirming the progress of the treatment. Any

orthodontic problems that have occurred that may influence the presurgical preparation, or any orthodontic corrections which the orthodontist may prefer to complete after surgery, should be communicated to the surgeon. Whether the surgical procedure should occur early or late in the orthodontic phase depends on the patient, the patient's growth status, the deformity, and the treatment team. Surgery can be performed either after most of the orthodontic tooth movement has been completed or may be performed early in the orthodontic phase. Completing nearly all the orthodontics before surgery has certain advantages:

- surgical repositioning of the jaws can be done more accurately once the teeth are well aligned;
- the risk of not meeting the treatment goals during postsurgical orthodontics is reduced;
- a better occlusion at the time of surgery enhances stability;
- a certain amount of orthodontic tooth movement has to be done before surgery anyway;
- after surgery and improved esthetics the patient's enthusiasm and cooperation dwindle.

Performing surgery early in the orthodontic phase also has certain advantages:

- orthodontic tooth movement after surgery is probably more rapid as a result of accelerated bone metabolism and increased rate of bone remodeling;
- early esthetic improvement for the patient;
- treatment time may be increased in an attempt to do most orthodontic alignment before surgery.

Postsurgical orthodontics

After the surgery, it is mandatory that the surgeon report to the orthodontist that the surgical objectives have been achieved according to the treatment plan or whether any surgical problems have been encountered which may have an influence on the postoperative orthodontics and/or the final result. The surgeon should not expect the orthodontist to correct surgical problems. Any postoperative problems such as incorrectly positioned condyles, fixation failure, etc. should be promptly recognized and corrected by the surgeon before turning care over to the orthodontist. An advantage of using internal rigid fixation is that intermaxillary fixation is not necessary and it allows early orthodontic intervention following surgery. Earlier physiotherapeutic rehabilitation of range of motion is also possible.

Postsurgical orthodontics can start 3–4 weeks after surgery. The teeth can be moved rapidly in the first 10 weeks following surgery and the orthodontist should see the patient every 1–2 weeks for adjustments during the first 2–3 months so that changes can be monitored closely. The same principles regarding

retention apply for orthognathic patients as for routine orthodontic patients.

Orthodontics without prior surgical consideration

Occasionally the orthodontist may start orthodontic treatment, not considering the need for surgery. Once the indication for surgical correction is realized by the orthodontist, the patient should be informed and the need for surgery explained. In cases where this situation does arise the orthodontist and surgeon should compare the pretreatment records with the current situation and assess the stability of the orthodontic mechanics. If there are any concerns regarding the stability of the dental alignment within the arches as a result of unfavorable tooth movement, the teeth should be allowed to settle for 4–6 months. The arch wires can be sectioned and the teeth be allowed to relapse (vertically and horizontally) while maintaining correction of any rotations. Unfavorable tooth movements with questionable stability may include dental expansion beyond the bony base, proclination of incisors out of the central trough of bone and intrusion or extrusion of teeth to close an anterior open bite.

After a settling period the patient should be re-evaluated and a new diagnosis and treatment plan formulated. Unfortunately some compensatory orthodontic treatment cannot be reversed, e.g. tooth extractions that might not have been done in a combined surgical–orthodontic treatment plan. Correction may now require surgical procedures that may not have been contemplated if surgery was considered in the first place.

Esthetic objectives and surgical solutions

The esthetic goals of each case will dictate the choice of the surgical procedure and are influenced by the patient's main complaint and the facial esthetic examination and treatment goals. Surgical treatment for the correction of dentofacial deformities is based on five basic treatment scenarios:

1. Single jaw surgery – lower jaw.
2. Single jaw surgery – upper jaw.
3. Double jaw surgery – upper and lower jaw.
4. Double jaw surgery – rotation of the maxillomandibular complex.
5. Chin surgery.

The esthetic goals are planned by the development of a cephalometric visual treatment objective. First a soft tissue esthetic objective is developed followed by an orthodontic treatment objective to ascertain where the teeth should be positioned to facilitate the planned surgical procedure and, in turn, the soft tissue goals.

Various possibilities should be considered when integrating the esthetic goals, the orthodontic treatment objectives, and the indicated surgical solutions with the development of the visual treatment objectives. Consider a case with mandibular antero-posterior deficiency requiring an increase in mandibular prominence. Several surgical solutions may be considered:

1. Surgical advancement of the mandible.
2. Surgical advancement of the mandible and the chin.
3. Surgical advancement of the chin.

The choice of the surgical procedure will be influenced by several factors:

1. The dental occlusion.
2. The mandibular skeletal base position.
3. The shape of the chin.

Each scenario will also have certain orthodontic solutions. The preoperative positioning of the teeth will dictate the nature and extent of the surgical procedure and will also influence the final esthetic result. The orthodontic treatment objectives should be coordinated with the esthetic requirement and this will influence the extraction pattern, the amount of dental decompensation, the required occlusal curve leveling, etc. It should be kept in mind that the orthodontic treatment objectives for surgical–orthodontic treatment often differ and, in many cases, are opposite to those of non-surgical cases.

Treatment principles for various surgical treatment scenarios

Single jaw surgery: mandibular repositioning

The dentition, especially the incisors of the unoperated jaw, in this case the maxilla, will determine the

new antero-posterior, transverse, and vertical position of the mandible (Fig. 46.30). The position and shape of the chin may, however, be changed by means of a genioplasty. Chin surgery is not a substitute for mandibular surgery and genioplasty should be performed to improve the shape of the chin (vertically, transversely, and antero-posteriorly) and not primarily to improve the chin projection (to position pogonion correctly). In cases requiring only mandibular surgery, the following aspects should be considered by the orthodontist and the surgeon:

Orthodontic considerations

1. Maxillary incisor position and angulation. Cephalometric guidelines should be followed to position the incisor teeth; however, in mandibular-deficient cases the upper incisors should not be retracted excessively. Excessive retraction of the upper incisors will reduce upper lip support and limit the amount of mandibular advancement in class II cases, while this will increase the amount mandibular setback with negative esthetic effects in class III cases.
2. Mandibular incisor. In mandibular-deficient cases there is often dental crowding present requiring tooth extractions. There are three extraction patterns that may be implemented: (1) where crowding in the anterior mandibular arch is present, the lower first bicuspid should be extracted – this will allow for arch alignment as well as sufficient lower incisor retraction when required; (2) the second bicuspid are usually removed in cases where there is crowding in the mid-arch – extraction of bicuspid in the lower arch only will lead to a class III molar and class I canine relationship. The lower incisors are often compensated (retroclined in class III cases and proclined in class II cases); (3) although seldom necessary, a lower incisor can be extracted. Extraction of a lower incisor may shorten the orthodontic treatment time, however

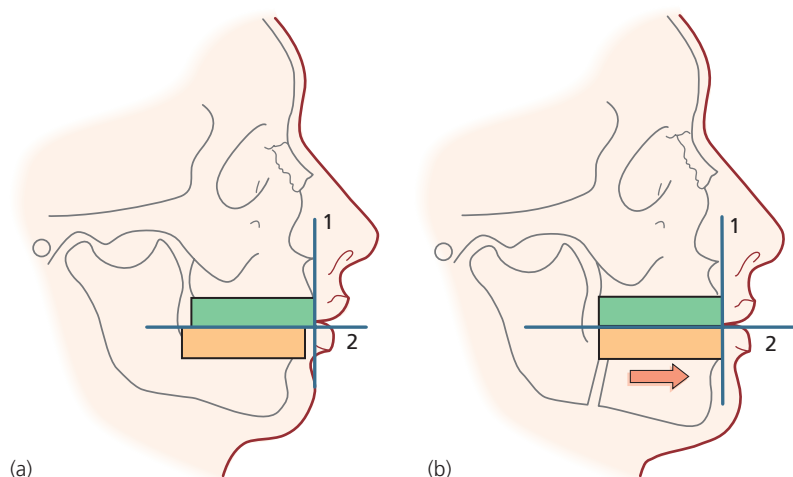


Fig. 46.30 The patient in (a) requires mandibular advancement. The mandible has been advanced (b) and the maxilla (the unoperated jaw) dictated the final antero-posterior (1) and vertical (2) position of the mandible.

will lead to a tooth size discrepancy. The amount of decompensation is primarily influenced by the bone thickness in the symphysis area and the amount of jaw repositioning that is required to achieve the best esthetic result, postoperative stability, and periodontal situation.

3. Dental midline. Ensure that the dental midline in the upper arch (unoperated jaw) is corrected orthodontically. In cases where the mandible is asymmetric the orthodontist should attempt to position the lower dental midline in the center of the chin so that facial asymmetry will be corrected by the mandibular surgery. Final mandibular asymmetry can, however, be corrected by genioplasty.
4. Maxillary arch form and rotation. The upper dental arch forms the template for the surgeon to position the mandible. Although the mandibular arch width can be surgically altered it is not done routinely. The orthodontist should ensure that the maxillary arch width, especially the intercanine width, will accommodate the lower arch after

repositioning. In class III cases there is often a tendency for the maxillary posterior teeth to be buccally inclined while the lower posterior teeth tend to be lingually inclined. This should preferably be corrected orthodontically before surgery.

5. Dental arch compatibility. One of the best tooth retainers is a good occlusion. It is therefore preferable that the teeth fit into a reasonable occlusion at the time of surgery. The orthodontist can monitor the arch compatibility during the presurgical treatment phase by means of check dental casts.
6. Leveling the occlusal curves. The occlusal curves in the maxillary arch should be leveled before surgery, however this is not always the case for the lower dental arch. In class II low mandibular plane angle cases with short lower facial heights, the curve of Spee should not be leveled before surgery. Surgical advancement of the mandible would result in an increase in lower facial height (Fig. 46.31). In cases with normal or increased lower facial height, the occlusal curve in the mandible should be leveled before mandibular advancement.

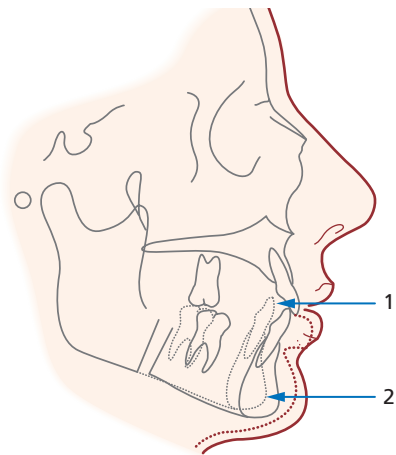


Fig. 46.31 The accentuated curve of Spee is not leveled before surgery. Advancement of the mandible will result in a three-point contact occlusion and bilateral open bites. The lower incisors will advance more than the chin (Pog) (1). The advancement and rotation of the mandible will also result in an increase in mandibular height (2).

Surgical considerations

1. The maxillary dental arch will dictate the amount of surgical advancement or setback of the mandible. The transverse mandibular position will be determined by the upper posterior teeth, while the antero-posterior position, midline, and height of the lower incisors will be guided by the upper incisors.
2. The chin position and shape can be altered by genioplasty

Single jaw surgery: maxillary surgery

As in the first treatment scenario, the final position of the jaw to be operated on (maxilla) will be determined by the unoperated jaw (mandible) (Fig. 46.32). In this

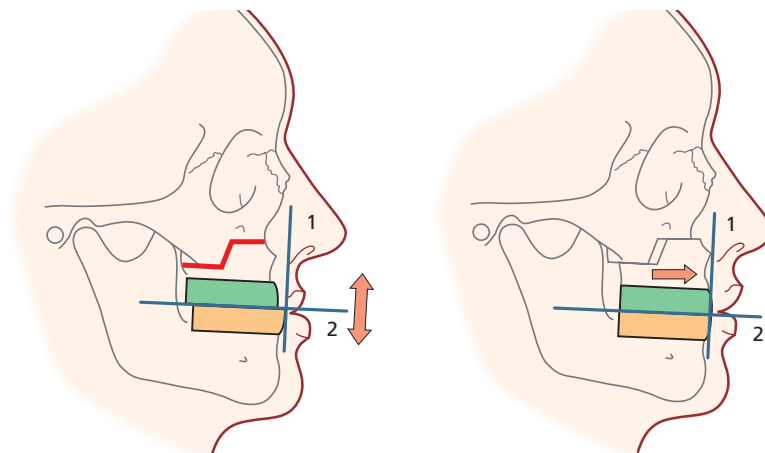


Fig. 46.32 The mandible (the unoperated jaw) will determine the final antero-posterior position of the maxilla. However any vertical correction of the maxillary height will result in upward and forward rotation following superior repositioning or downward and backward rotation of the mandible following down graft (2).

scenario, however, the surgeon may alter the height of the maxilla by inferior or superior repositioning. When the maxillary height is increased by downgrafting or decreased by superior repositioning, the mandible will autorotate around a point at the condyle. This clockwise or counterclockwise rotation will alter the antero-posterior position of the lower incisors which will demand consideration. This treatment scenario will require additional surgical and orthodontic considerations.

Orthodontic considerations

1. Maxillary incisor position and angulation. Dental crowding is often present in class III cases as a result of maxillary antero-posterior deficiency, and extractions may be required in the upper arch to align the teeth. Three extraction patterns may be implemented: (a) crowding in the maxillary arch and no crowding in the lower arch – extract the first bicuspid; (b) crowding in the posterior maxillary arch – extract the second bicuspid; (c) crowding in both arches – extract the first upper bicuspid and the second lower bicuspid. These extraction patterns will obviously be influenced by other factors, such as tooth prognosis, the area of crowding, and tooth angulation or compensation. Orthodontic alignment and correction of incisor angulation should be done before surgery.
2. Lower incisor position. The lower incisor position is determined by factors described above, however in this scenario the final antero-posterior position of the lower incisors will change following vertical change and mandibular autorotation and should be planned according to the surgical visual treatment objective. In high occlusal plane angle cases the antero-posterior change is more than in low occlusal plane cases.
3. Occlusal curves. The curve of Spee should be leveled, as in this scenario the lower arch acts as the template. Occlusal curves in the upper arch should also be leveled before surgery, however there is often a natural step in the maxillary arch especially in open bite cases. In these cases as well as cases requiring surgical expansion, the arch should be aligned in segments and the roots of the teeth adjacent to the intended interdental osteotomies must be deviated.
4. Dental arch compatibility. The arch form of the segments should be coordinated with the lower arch form.
5. Dental midlines. The dental midline of the lower arch should be coordinated with the facial midline. The orthodontist should not waste treatment time by correcting the upper dental midline as this can be done surgically.
6. Preparation for segmental surgery. Surgical alteration of the maxillary arch or correction of occlusal curves (open bite cases) may be indicated. The orthodontist should deviate the roots of the teeth

in the intended interdental osteotomy areas before surgery. Root deviation should be more prominent when interdental closure of spaces is planned than when only differential vertical repositioning of the segments is required.

Surgical considerations

1. Antero-posterior positioning of maxilla. The final horizontal position of the maxilla will be determined by the antero-posterior position of the lower incisor. However, with any vertical change in the position of the maxilla, the mandible will autorotate thereby changing the horizontal position of the lower incisor. With superior repositioning of the maxilla the lower incisor will rotate anteriorly, while with inferior repositioning of the maxilla the lower incisor will rotate posteriorly. With high mandibular plane angle cases the antero-posterior changes following autorotation are more than with low angle cases. These geographic changes should be considered in relation to the esthetic consequences (Fig. 46.33).
2. Vertical positioning of the maxilla. Surgical repositioning of the maxillary incisor in its most favorable antero-posterior and vertical position is one of the most important keys to orthognathic treatment.
3. Transverse corrections. Maxillary surgery also allows the surgeon to alter the transverse dimension of the upper arch. By performing segmental surgery the surgeon can either expand or narrow the maxillary dental arch to fit the mandibular arch. The interdental osteotomies are placed either between the upper lateral incisor and canine tooth or between the canine and bicuspid tooth. The authors prefer to place the interdental osteotomies between the lateral incisor and the canine tooth. The alveolar bone is thin in this area, surgical sight is easily accessible, the surgeon can control the

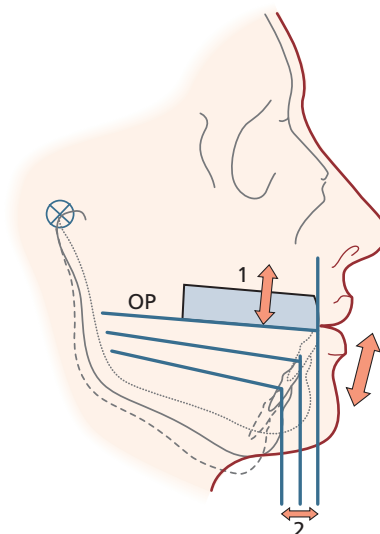


Fig. 46.33 The height of the maxilla (1) may be changed, which will have an effect on the final antero-posterior position of the lower incisor as a result of the rotation of the mandible (2).

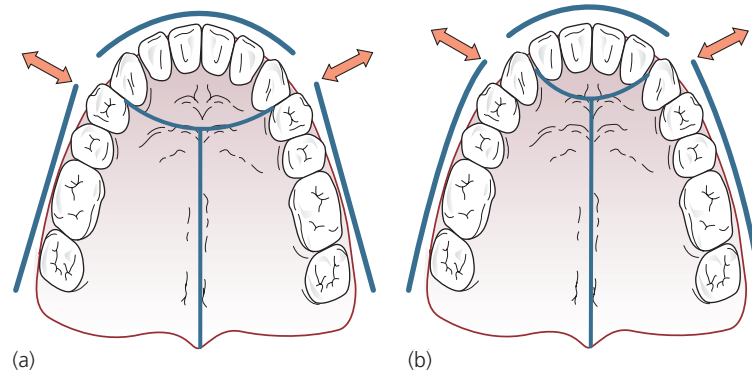


Fig. 46.34 (a) The interstitial osteotomies are placed between the canine and first bicuspid teeth. (b) The interstitial osteotomies are placed between the lateral incisor and canine teeth. In both cases the interstitial osteotomies are combined with palatal osteotomies.

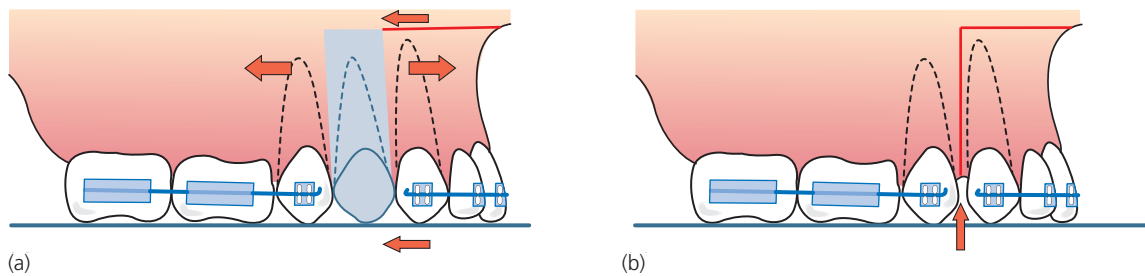


Fig. 46.35 (a) Surgical closure of interdental spaces will require adequate deviation of the roots of the teeth adjacent to the interdental osteotomies. (b) To ensure integrity of the periodontium, interdental spaces should never be completely closed, and a small amount of bone should always be left intact at the alveolar crest.

intercanine width, and, by deviating the roots, the canine tooth is orthodontically close to its final position and need not be moved following surgery (Fig. 46.34).

4. Occlusal curves. By performing segmental surgery the surgeon can level occlusal curves that may exist in the upper arch. Differential vertical repositioning of the anterior and posterior segments will allow the mandible to autorotate and, for example, close an anterior open bite.
5. Closure of interdental spaces. Segmental surgery also allows the surgeon to close interdental spaces. Orthodontic deviation of the roots of the teeth adjacent to interdental osteotomy areas must be adequate to prevent damage to the tooth roots during surgery (Fig. 46.35a). An area of alveolar bone should always be maintained around the crest area to prevent periodontal problems (Fig. 46.35b).

Double jaw surgery: repositioning the maxilla and the mandible

When the treatment plan demands surgery to both jaws, the surgery to either jaw can be performed first. The authors prefer to perform upper jaw surgery first. Once the upper jaw procedure has been completed the final position of the mandible will be determined by the maxilla and the principles of single jaw surgery then apply.

Orthodontic considerations

1. Tooth extractions. The extraction of teeth when indicated will be influenced by the following: crowding (anterior or posterior), the required incisor discrepancy (overjet or crossbite) to facilitate the ideal jaw movements, dental stability, and leveling of occlusal curves.
2. Correction of transverse occlusal cants. If an occlusal cant exists in a patient with facial asymmetry, surgical correction of the cant will often play an important role in the correction. The cant should therefore not be corrected orthodontically.
3. Incisor position. Ensuring proper incisor angulation as well as establishing the planned discrepancy between the upper and lower incisor teeth will facilitate the required jaw movement.
4. Dental arch form and interarch compatibility will enhance good intercuspation during surgery which will improve postoperative stability.
5. Dental midlines. The orthodontist should not waste treatment time by correcting dental midlines. Dental midlines can be corrected during surgery.
6. Transverse problems. Transverse discrepancies involving dental tipping should be corrected orthodontically. The teeth must not be moved beyond their bony base, however, as this may result in relapse (Fig. 46.29). Although it is possible to alter the width of the mandibular arch surgically

it is seldom indicated. It is generally necessary to establish a good lower arch form before surgery. Transverse skeletal discrepancies can be corrected by either surgically assisted palatal expansion or segmental surgery. Buccal tipping of the posterior teeth will accentuate the curve of Wilson. Before surgical expansion the orthodontist should ensure that the posterior teeth are upright (level the curve of Wilson) and in the central trough of bone because buccally tipped molars will increase the amount of surgical palatal expansion (Fig. 46.29).

Surgical considerations

The “responsibility” and considerations of the surgeon increase when planning and performing two jaw surgeries. However, once the first jaw is positioned, it is like a single jaw surgery case, with the jaw positioned first determining the position of the second jaw. It is therefore mandatory that surgery be performed accurately. With the advent of rigid fixation, either jaw can be operated on first, however the authors prefer to perform surgery to the upper jaw first. The following are important factors to consider by the surgeon:

1. Vertical position of the anterior maxilla. The position is determined by the upper incisor upper lip relationship.
2. Antero-posterior position of the mandible. The antero-posterior position of the mandible will change following alteration of the height of the maxilla. A decrease in maxillary height (maxillary superior repositioning) will cause the mandible to rotate anteriorly (clockwise around the condyle). An increase in the maxillary height (maxillary downgraft) will cause the mandible to rotate posteriorly (Fig. 46.33).
3. Vertical position of the posterior maxilla. In conventional treatment planning, when maxillomandibular complex rotation is not contemplated, the vertical position of the posterior maxillary teeth is dictated by the mandibular molars. Any vertical change in the maxilla (superior or inferior) will result in autorotation of the mandible (around a point at the condyle) altering the height of the mandibular molars. Correction of anterior open bite often requires differential superior repositioning of the anterior and posterior maxilla.
4. The height of the right and left sides of the maxilla. There is often a transverse cant of the occlusal plane present in facial asymmetry cases. Surgical correction of the cant usually assists with overall correction of the facial asymmetry and the final occlusal plane should be parallel to the interpupillary line, given that the interpupillary line is horizontally correct. Correction of the height of the left and right sides of the maxilla will have an influence on the vertical position of the maxillary incisor, the transverse position of the upper dental

midline, as well as the symmetry of the chin, which should be kept in mind during planning and execution of surgery.

5. Antero-posterior position of the maxilla. Esthetic considerations, such as paranasal form, upper lip support, nasolabial angle, and facial contour, will determine the final antero-posterior position of the maxilla.
6. Maxillary arch rotations. The left and right sides of the maxilla should be advanced equally to ensure facial symmetry.
7. Dental midlines. When both jaws are surgically repositioned the surgeon is responsible for positioning the dental midlines in the middle of the face and the orthodontist should not waste treatment time by correcting dental midlines. It is extremely disappointing to realize that the dental midlines were eccentrically positioned during surgery – the day following surgery.
8. Transverse width of the dental arches. The width of the maxillary dental arch is determined by the width of the mandibular arch unless surgical change of the lower arch is contemplated. A narrow upper arch due to dental tipping should be corrected orthodontically. Skeletal transverse deficiency in growing individuals can be corrected by orthodontic rapid palatal expansion, while in adults the transverse dimension of the maxilla can be increased by surgically assisted expansion or surgical segmental expansion. Care should be taken not to damage the tooth roots of the teeth adjacent to the interdental osteotomies and to preserve the integrity of the periodontium by leaving a small amount of bone at the alveolar crest (Fig. 46.35).

Double jaw surgery: rotation of the maxillomandibular complex

A satisfactory functional, stable, and esthetic result may be achieved by correcting dentofacial deformities according to conventional orthognathic treatment design. Adhering to conventional treatment designs may, however, hamper achievement of optimal esthetic results, especially in severe high or low occlusal plane angle cases. Rotation of the maxillomandibular complex and the consequent alteration of the occlusal plane is a surgical design that will allow the surgeon to achieve certain esthetic results that cannot be achieved by conventional treatment methods.¹⁶ In this treatment scenario the final occlusal plane angle will not be determined by the occlusal plane of the mandible.¹⁷

Orthodontic considerations

The orthodontic treatment considerations in this scenario are the same as discussed for two jaw surgery. Special attention should, however, be given to the angulation of the incisor teeth, because the incisor

angulation will also change as a consequence of the rotation of the maxillomandibular complex. The orthodontist may have to compensate for the change in angulation.

Surgical considerations

The surgeon has two more decisions to make in this scenario.

1. The direction of rotation of the maxillomandibular complex. In general patients with concave profiles will require clockwise rotation, while patients with convex profiles will require counterclockwise rotation of the maxillomandibular complex (Fig. 46.36). See Tables 46.11–46.14.
2. The point around which the maxillomandibular complex should be rotated. To simplify the planning and to allow visualization of the effects of the rotation, a triangle is constructed involving the anterior nasal spine (ANS), the posterior nasal spine (PNS), and pogonion (Pog). By changing the direction of rotation and varying the point of rotation on the triangle the esthetic possibilities can be investigated (Fig. 46.37). The actual rotation point

at the time of surgery will differ from the point on the treatment objective. It will be on the Le Fort I osteotomy line and should be determined by the surgical visual treatment objective.¹⁸ In general the following rules will apply:

- *The antero-posterior position of the chin (Pog).* The surgeon should first ensure that the shape and form of the chin are esthetically pleasing. This may require genioplasty and the position of pogonion should be evaluated once the chin shape is satisfactory. The higher and more anterior the rotation point the more pronounced the antero-posterior movement of the chin (Fig. 46.38).
- *Paranasal areas and ANS.* The lower the rotation point on the line between the ANS and Pog, the more pronounced the antero-posterior movement of the ANS (Fig. 46.39).
- *Vertical changes.* Any rotation point on the line between the PNS and the ANS will also have vertical implications (Fig. 46.40).

Counterclockwise rotation is very seldom performed using Pog as the rotation point as this design will result in excessive setback of the maxilla. Maxillary setback, in most cases, would result

Table 46.11 Counterclockwise rotation of the maxillomandibular complex – hard tissue changes.

	Rotation point and effects			
	ANS	Incisor tip	Zygomatic buttress	PNS
OP angle	Decrease	Decrease +	Decrease +	Decrease ++
Maxillary incisor tip	Advance	No change	Advance and superior	Advance + and superior +
Pog position	Advance +	Advance	Advance and superior	Advance and superior +
Maxillary incisor angle	Increase	Increase	Increase	Increase +
Maxilla at ANS	No change	Setback	Superior	Superior +
MP angle	Decrease	Decrease	Decrease	Decrease +
Posterior maxillary height	Increase +	Increase	Increase	No change

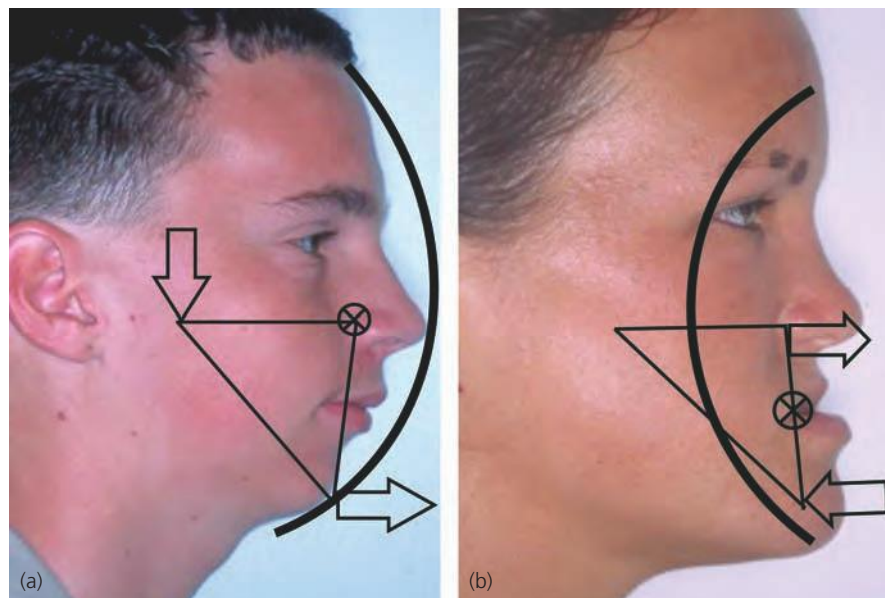


Fig. 46.36 (a) The surgical correction of patients with convex profiles can be enhanced by rotation of the maxillomandibular complex in a counterclockwise direction. (b) The correction of concave profiles will be enhanced by rotating the maxillomandibular complex in a clockwise direction.

Table 46.12 Counter clockwise rotation of the maxillomandibular complex – soft tissue changes.

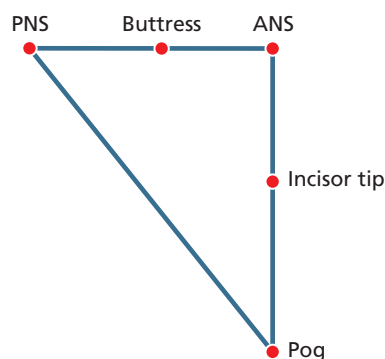
	Rotation point and effects			
	ANS	Incisor tip	Zygomatic buttress	PNS
Subnasale	No change	Setback	Superior	Superior
Upper lip support	Increase +	No change	Increase	Increase
Facial convexity	Decrease +	Decrease	Decrease	Decrease +
Mandibular prominence	Increase +	Increase	Increase	Increase +
Paranasal fullness	No change	No change	Increase	Increase
Nasolabial angle	Decrease +	Decrease	Decrease	Decrease +
Anterior facial height	Decrease +	No change	Decrease	Decrease ++
Chin throat length	Increase +	Increase	Increase	Increase ++

Table 46.13 Clockwise rotation of the maxillomandibular complex – hard tissue changes.

	Rotation point and effects				
	ANS	Incisor tip	Zygomatic buttress	PNS	Pog
OP angle	Increase +	Increase	Increase	Increase +	Increase +
Maxillary incisor tip	Retraction	No change	Inferior and retraction	Retraction + and inferior	Advance
Pog position	Setback + and vertical increase	Setback	Setback	Setback + and vertical increase	No change
Maxillary incisor angle	Decrease	Decrease	Decrease	Decrease	Decrease
Maxilla at ANS	No change	Advance	Increase	Inferior	Advance +
MP angle	Increase	Increase	Increase	Increase	Increase
Posterior maxillary height	Decrease +	Decrease	Decrease	No change	Decrease

Table 46.14 Clockwise rotation of the maxillomandibular complex – soft tissue changes.

	Rotation point and effects				
	ANS	Incisor tip	Zygomatic buttress	PNS	Pog
Subnasale	No change	Advance	Advance	Vertical increase	Advance +
Upper lip support	Decrease	No change	Decrease	Decrease +	Increase
Facial convexity	Increase +	Increase	Increased	Increase +	Increase +
Mandibular prominence	Decrease +	Decrease	No change	Decrease +	No change
Paranasal fullness	No change	Increase	Increase	Inferior	Increase +
Nasolabial angle	Increase +	Increase	Increase	Increase +	Increase +
Anterior facial height	No change	No change	Increase	Increase	No change
Chin throat length	Decrease	Decrease	Decrease	Decrease	No change

**Fig. 46.37** The concept of rotating the maxillomandibular complex can be simplified by constructing a triangle including PNS, ANS, and Pog.

in poor esthetics. Clockwise rotation, however, would enhance maxillary advancement and, in many cases, is esthetically advantageous. Tables 46.11–46.14 summarize the effects, at seven sites, of rotation of the MMC at the four most commonly used rotation points.

Chin surgery

The prime goal of genioplasty is to improve the shape of the chin. Surgical advancement of the chin should not be performed for individuals requiring mandibular advancement. This may achieve the correct chin projection (pogonion in correct antero-posterior

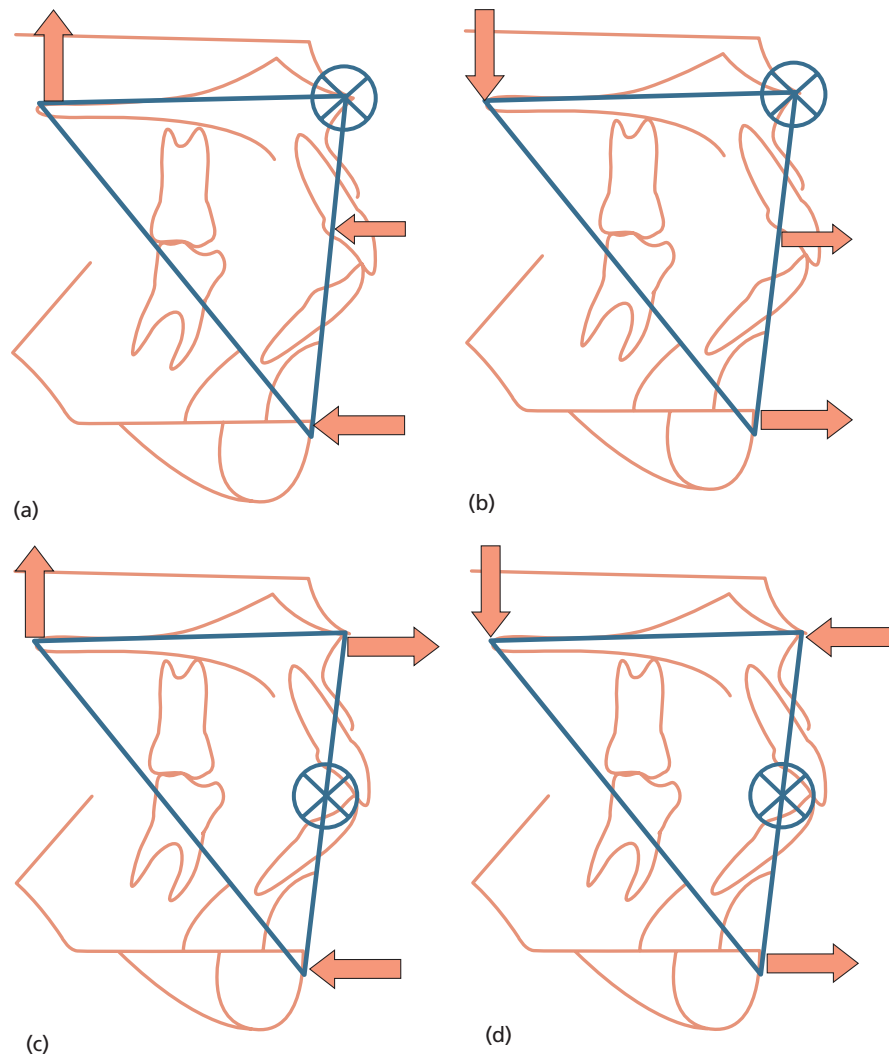


Fig. 46.38 (a) Counterclockwise rotation of the maxillomandibular complex around ANS. The posterior maxilla is repositioned inferiorly, and the incisor teeth and chin are advanced. The expected hard and soft tissue changes are summarized in Tables 46.11 and 46.12. (b) Clockwise rotation of the maxillomandibular complex around ANS. The posterior maxilla is repositioned superiorly, and the incisor teeth and chin move posteriorly. The hard and soft tissue changes are summarized in Tables 46.13 and 46.14. (c) Clockwise rotation of the maxillomandibular complex around the incisor tip. The posterior maxilla is superiorly repositioned, ANS is advanced and the chin moved posteriorly. The expected hard and soft tissue changes are summarized in Tables 46.13 and 46.14. (d) Counterclockwise rotation of the maxillomandibular complex around the incisor tip. The posterior maxilla is inferiorly repositioned, ANS is moved posteriorly and the chin advanced. The expected hard and soft tissue changes are summarized in Tables 46.11 and 46.12.

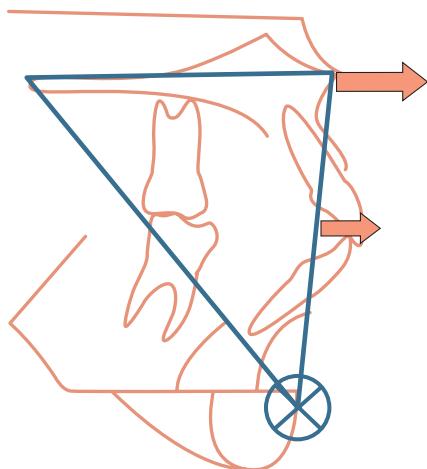


Fig. 46.39 Clockwise rotation of the maxillomandibular complex around Pog. ANS and the incisors are advanced. The expected hard and soft tissue changes are summarized in Tables 46.13 and 46.14.

position), however the balance and harmony of the chin will be compromised (Figs 46.41 and 46.42). In profile view the authors use seven criteria for the esthetic evaluation of the chin which also serve as a guide to surgical treatment planning (Fig. 46.43):

1. The height of the chin. The height of the chin should be equivalent to two thirds of the lower facial height (subnasale to stomion inferius (S^1): stomion inferius (S^1) to Me). The linear measurement should be 40 ± 2 mm for females and 44 ± 2 mm for males (1 in Fig. 46.43).
2. Vermillion exposure. The lower lip vermillion exposure should be approximately 25% more than the upper lip vermillion (2 in Fig. 46.43).
3. The labiomental fold. The contour of this fold should form a smooth curve connecting the lower lip with the chin. The depth of the fold should

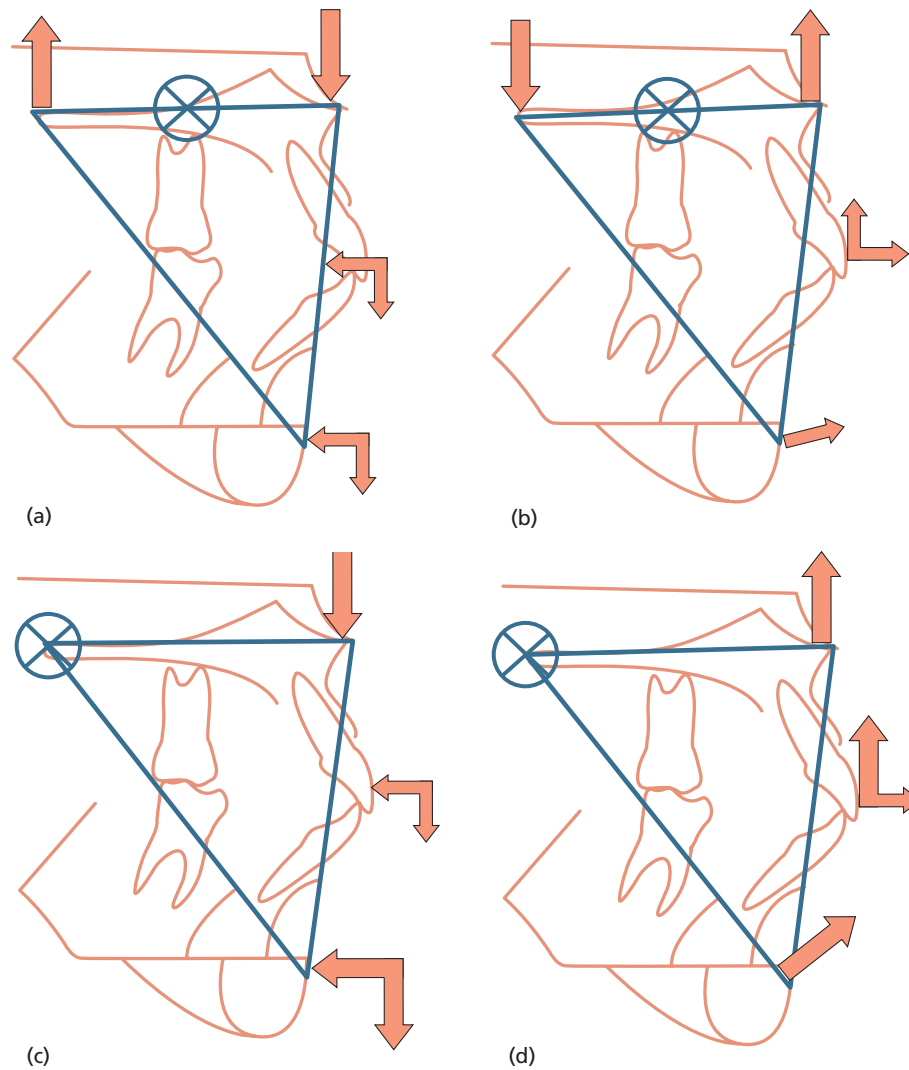


Fig. 46.40 (a) Clockwise rotation of the maxillomandibular complex around the zygomatic buttress. ANS moves inferiorly and the incisors and Pog move posteriorly. The expected hard and soft tissue changes are summarized in Tables 46.13 and 46.14. (b) Counterclockwise rotation of the MMC around the zygomatic buttress. ANS, incisor tip and Pog move anteriorly and superiorly. The expected hard and soft tissue changes are summarized in Tables 46.11 and 46.12. (c) Clockwise rotation of the MMC around the PNS. The incisor tip and Pog move posteriorly and inferiorly. The expected hard and soft tissue changes are summarized in Tables 46.13 and 46.14. (d) Counterclockwise rotation of the MMC around the PNS. The incisor tip and Pog move anteriorly and superiorly. The expected hard and soft tissue changes are summarized in Tables 46.11 and 46.12.

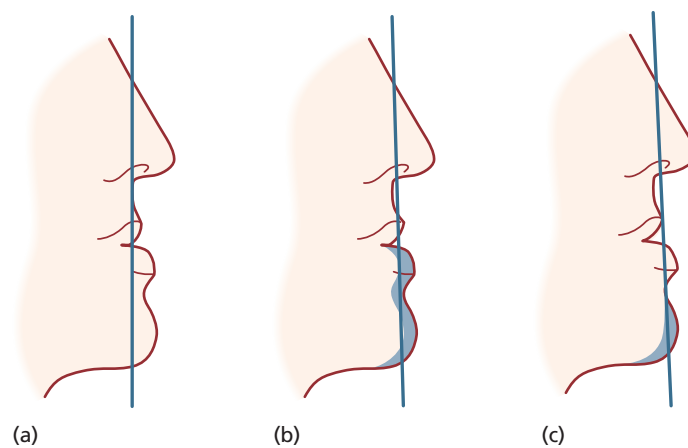


Fig. 46.41 (a) The mandible is antero-posteriorly excessive, however the chin contour is esthetically pleasing. (b) Mandibular excess corrected by mandibular setback results in an esthetically pleasing profile. (c) Reduction genioplasty for the patient results in poor chin esthetics with obliteration of the labiomental fold.

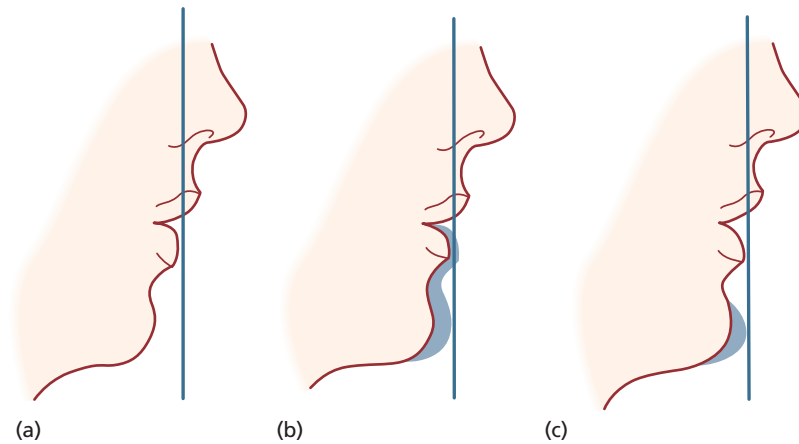


Fig. 46.42 (a) The profile of a patient with mandibular antero-posterior deficiency but with esthetically pleasing chin contour. (b) Mandibular advancement results in a pleasing profile. (c) Advancement of the chin by means of a genioplasty deepens the labiomental fold and gives the chin a knobby appearance.

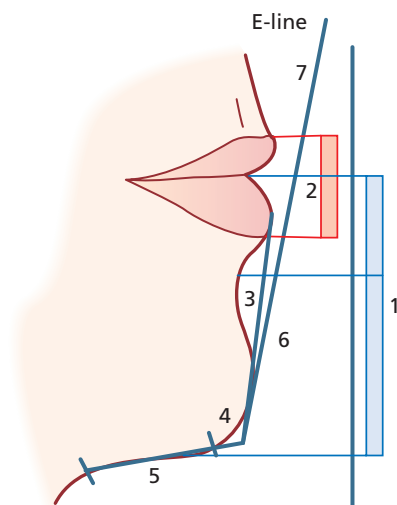


Fig. 46.43 The seven criteria used for the esthetic evaluation of the chin.

divide the lower lip chin into an upper third and lower two thirds. Individuals with mandibular deficiency often exhibit deep labiomental folds while mandibular excessive individuals will often exhibit flat folds (3 in Fig. 46.43).

4. Lower lip-chin-throat angle. This angle should measure $110 \pm 8^\circ$ and tends to be more acute in mandibular prognathic individuals and more obtuse in patients with mandibular deficiency (4 in Fig. 46.43).
5. Chin-throat length. The normal length is considered to be 42 ± 6 mm. Individuals with short chin-throat lengths would obviously benefit from chin advancement while those with prominent chin buttons and deep labiomental folds would benefit from a chin setback procedure (5 in Fig. 46.43).
6. Curvature of the chin. The profile of the chin should form a smooth S-shaped curve from lower lip superior to Me (6 in Fig. 46.43).
7. Lower lip position. The lower lip should be 2 ± 2 mm behind the E-line. The E-line is drawn from the nasal tip (Pn) to pogonion (Pog) (7 in Fig. 46.43).

Patients see themselves from the frontal view and evaluate their chin esthetics from their image in a mirror. Frontal evaluation of the face and chin is therefore as important as the profile assessment.

1. Facial and chin form. The general facial form, round, oval, square, or long, should correlate with the form of the chin. It is expected that females have smaller more rounded chins, while it is considered more attractive for males to have broad, square-shaped chins. The mandible should have a well defined smooth inferior border, from angle to symphysis, with a definite separation of the lower third of the face from the neck. An ill defined lower border of the mandible distracts from good chin-neck esthetics.
2. Transverse dimension. The width of the chin should be in balance with the bigonial and bizygomatic facial widths. Patients with leptoprosopic facial features often have a transversely deficient, "pointed" chin that seems separate from the mandible. Advancement of the chin by means of a sliding genioplasty may improve the profile, however this will accentuate the "pointiness" from the frontal view. For these patients an advancement of the chin should be combined with simultaneous transverse augmentation by means of a two-piece genioplasty (Fig. 46.13d). The esthetically pleasing chin should not have any parasymphyseal depression or "marionette grooves" which are often accentuated with age. An advancement genioplasty without lateral augmentation will deepen these grooves.
3. Chin asymmetry. There are three factors which should be considered when assessing the symmetry of the chin. Firstly, the relationship between the midline of the lower incisors and the midline of the chin. If the lower incisor midline coincides with the facial midline, then the chin asymmetry should be corrected by means of a lateral sliding genioplasty. Secondly, the relationship between the facial midline and the chin midline. In cases

where the lower incisor midline coincides with the midline of the chin, the asymmetry should be corrected by means of mandibular surgery. Finally, the presence of a cant of the lower border of the chin. The cant should be related to the interpupillary line and the mandibular occlusal plane and correction of the cant should form part of the lateral correction. Keep in mind that correction of facial asymmetry by maxillary and mandibular surgery, including correction of an occlusal cant, will often also have an effect on the midline of the chin as well as the lower border cant.

4. Chin proportion should be assessed in the frontal plane for transverse and vertical asymmetry. The critical aspect of asymmetry diagnosis of the genial complex is to decide whether the genial complex alone is deviating from symmetry or whether the entire mandible is the cause of the asymmetry (of course a combination of these may also occur). In general if the mandible is asymmetric the lower incisor midline and the genial midline are coincident and correction requires mandibular surgery. If these do not correspond, transverse asymmetry of the chin exists which requires a lateral sliding genioplasty for correction.

Treatment priorities and sequencing the treatment

Before the patient commits to treatment, the treatment plan, the envisaged treatment time of each phase, and the sequence of the steps should be discussed with the patient. To ensure that the dentition and soft tissues are sound, basic restorative dentistry, periodontal treatment, surgical removal of impacted teeth, and bicuspid extractions (when indicated) should be completed before commencement of treatment. Although it may be preferred in some cases to perform surgery early in the orthodontic treatment phase, e.g. class II deep bite cases, it is usually preferable to perform surgery only once good interarch compatibility has been established. This will allow the surgeon to position the jaws more accurately, the teeth will interdigitate better after surgery improving postoperative stability, and in general most patients' enthusiasm and compliance falter after surgery. The postoperative orthodontic phase should not be longer than 3–6 months.

It may be advantageous to place dental implants at the same time as the orthognathic surgery as it will save the patient a second surgical procedure. The authors, however, feel that implants at this stage should be limited to single-tooth implants. The orthognathic surgery stage is an ideal time to perform bone grafting, however, if required for the placement of implants later. This surgical stage also allows for easy intraoral harvesting of bone for grafting. The ideal time for the placement of dental implants is after an orthodontic retention period of approximate-

ly 3 months following band removal. Prosthodontic restoration will enhance orthodontic stability and should be performed as soon as possible.

Visual treatment objective

Many methods of developing a cephalometric treatment objective have been described, varying from hand-drawn orthodontic and surgical changes on an acetate tracing paper, computerized digital predictions, and video imaging techniques.¹⁹ There are three types of visual treatment objectives:

1. A pretreatment orthodontic visual treatment objective. This visual treatment objective is developed from the lateral cephalometric radiograph using all the information gathered from the systematic patient evaluation and database. The orthodontic prediction tracing indicates the required tooth movements and will include: planned extractions, closure of spaces, improving tooth angulations, leveling of occlusal curves where indicated, etc. The prime goal is to place the teeth in a position that will allow the surgeon to achieve the optimal soft and hard tissue results.
2. The pretreatment surgical prediction tracing is developed from the orthodontic prediction tracing. The jaw(s) are now moved and traced in their planned position and the soft tissue changes predicted. The expected soft tissue changes are now evaluated. In cases where an unacceptable esthetic result is achieved on the surgical prediction, the treatment plan should be revised.
3. An immediate presurgical visual objective is created a few days before surgery using the presurgical lateral cephalometric radiograph. Accurate definitive surgical movements are planned and soft tissue changes predicted. The planned dental and skeletal movements are then simulated on articulated dental casts by means of model surgery.

Model surgery

The primary goal of presurgical model surgery is to recreate the patient's jaws and teeth relationship as accurately as possible on an articulator to allow accurate simulation of the intended surgery. The surgical movements as indicated by the surgical cephalometric visual treatment objective are simulated on the articulated cast models and the surgical changes are then recorded. Numerous techniques for model surgery have been described.²⁰ Most techniques involving repositioning of both jaws advocate repositioning the maxilla first and then fabricating an intermediate acrylic splint with the unoperated mandible as a reference. The mandibular arch is now positioned with the teeth in the best occlusion and a final acrylic splint can be fabricated, although the authors very seldom

make use of a final splint. The surgical splint is the key to accurate surgery and thus it is mandatory that model surgery is performed accurately and special care taken during fabrication of the splint.

Surgical technique

Our knowledge and understanding of all aspects of orthognathic surgery have grown over the past 3 decades. At the same time numerous surgical techniques have been developed and refined which enable the modern orthognathic surgeon to treat most complex dentofacial deformities with confidence. The three surgical techniques currently used for the correction of dentofacial deformities, with various modifications, are the bilateral sagittal split ramus osteotomy of the mandible, the Le Fort I maxillary osteotomy, and the genioplasty.

Following the formulation of a treatment plan accurate surgery is required to ensure a successful treatment outcome. Good surgical technique involves the following:

1. Proper treatment planning. The surgeon should be guided by a comprehensive treatment plan including accurate cephalometric, surgical visual treatment objectives and model surgery.
2. Surgical routine. The surgeon should develop a specific routine for each procedure. By following the same sequence (step by step) it will enable the assistants to anticipate each subsequent step, thus increasing efficiency, and decreasing operating time and ultimately patient morbidity. The surgeon should have a clear understanding of each surgical step, the complications that may arise from each step, and the management of each complication.
3. Instrumentation. A myriad of surgical instruments has been designed, all to help the surgeon to eventually achieve the same goal. It is, however, recommended that the surgeon only use a small select group of "favorite" instruments: it is less confusing to both the surgeon and the surgical team.

Bilateral sagittal split ramus osteotomy

Surgical repositioning of the mandible was first described by Blair in 1907;²¹ however, the first report of splitting the mandibular ramus in a sagittal direction was by Trauner and Obwegeser in 1957,²² and also in 1961 by Dal Pont.²³ This technique underwent several modifications over the years.^{24,25} Surgical repositioning of the mandible has now developed from a potentially life-threatening procedure to outpatient surgery (in some parts of the world). The advent of rigid fixation has made postoperative recovery safer and much more comfortable for patients. It is not the intention to describe the surgical

procedure in detail as in a surgical manual, however a short overview in a step-by-step fashion with notes regarding recommendations, possible complications, and their management is provided. For more in-depth discussion on the detail of the surgical technique the reader is referred to text specifically describing surgical technique.²⁶⁻²⁸

1. Soft tissue infiltration with vasoconstrictor. To assist in intraoperative control of hemorrhage, all areas of dissection are infiltrated with vasoconstrictor (epinephrine at a concentration of 1:80 000), 10 minutes before surgery.
2. Soft tissue incision. The soft tissue incision is made through mucosa, muscle, and periosteum, starting superiorly halfway up the mandibular ramus, just lingual to the external oblique ridge carrying the incision downwards ending mesial to the second molar. Leave at least 5 mm of non-keratinized epithelium buccally at the lower end for ease of suturing later.
3. Subperiosteal dissection. Dissection of the periosteum should be limited only to areas where the osteotomies will be performed and muscle attachments that need to be separated from the bone to facilitate bone repositioning and ensure postoperative stability. Take special care during lingual dissection not to damage the inferior alveolar nerve. It is, however, mandatory that the lingula and entrance of the nerve at the foramen be positively identified. The dissection should be decisive, clean, and neat, and should remain under the periosteum at all times.
4. Perform the osteotomies. The sagittal osteotomies can be divided into three parts:
 - The medial ramus osteotomy. Use a 701 fissure bur and aim at the notch of the lingula and terminate the osteotomy in the fossa just posterior of the lingula (Fig. 46.44a). When the osteotomy is terminated short of the lingula, the bone will tend to split anterior to the foramen, leaving the lingula and superior part of the alveolar canal (including the nerve) attached to the proximal segment during the splitting procedure. If this happens the canal should be carefully dissected from the proximal segment, but this will increase the sensory morbidity of the inferior alveolar nerve.
 - The vertical section. Start superiorly at the medial osteotomy, stay just inside the buccal cortex, and end the osteotomy mesial to the second molar.
 - With a channel retractor in place, start the buccal osteotomy at the lower border of the mandibular body, and join it superiorly at the vertical osteotomy. The buccal cortex should be perforated, but take care not to damage the inferior alveolar nerve. Ensure that the lower part of the lingual cortex is included in the inferior osteotomy (Fig. 46.44b).

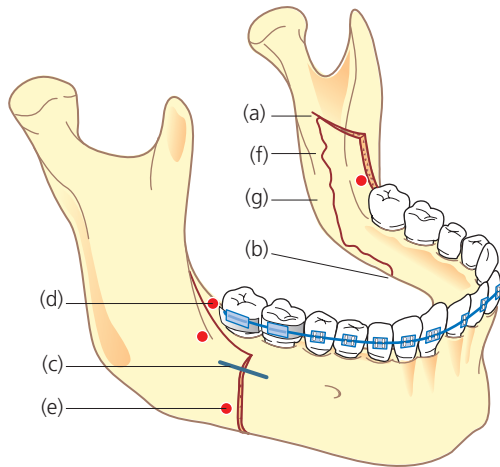


Fig. 46.44 The surgical design of the bilateral sagittal split ramus osteotomy. (a) The horizontal osteotomy on the medial side of the ramus should extend to just posterior of the lingula into the fossa posterior to the entrance of the inferior alveolar nerve. (b) Ensure that the vertical osteotomy on the lateral side of the body extends to include the lingual cortex. (c) A reference mark across the vertical osteotomy to assist in realignment of the segments. (d) Holes placed for the holding wire, distal on the proximal segment and anterior on the distal segment. (e) A purchase hole placed low down on the proximal segment to facilitate condylar positioning. (f) The stylomandibular ligament is stripped from the angle. (g) The medial pterygoid muscle attachment is stripped from the angle.

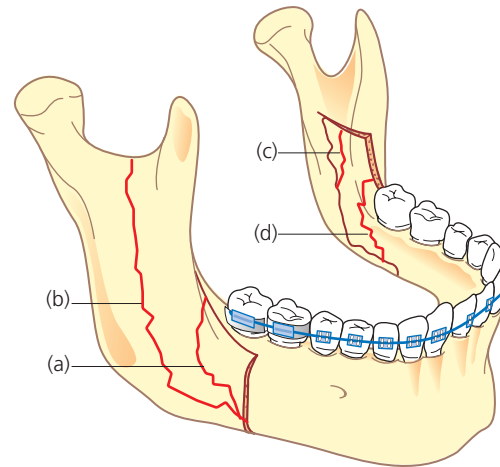


Fig. 46.45 Failure to incorporate the lingual cortex at the lower border when performing the vertical osteotomy may result in: (a) fracture of the buccal plate or (b) fracture of a large part of the proximal segment including the coronoid process. (c) Fracture of the lingual part of the distal segment may occur as a result of indiscriminate surgery or the presence of an impacted third molar tooth. (d) A fracture short of the inferior alveolar foramen may occur as a result of the horizontal osteotomy being performed short of the lingula.

5. Place reference marks. To ensure correct alignment of the bone segments during fixation, reference marks are placed on the buccal cortex on either side of the vertical osteotomy (Fig. 46.44c).
6. Place the holes for a holding wire. The authors prefer the use of holding wires during placement of rigid fixation. The holes are placed anteriorly through the buccal cortex of the proximal segment and posteriorly through the lingual cortex of the distal segment. The vector of the holding wire will tend to pull the proximal segment posteriorly (enhancing seating of the condyle) and the distal segment anteriorly (supporting the occlusion) during the placement of rigid fixation (Fig. 46.43d). A bone clamp or condylar positioner may be used as an alternative. It is, however, mandatory not to force the segment together with the clamp
7. Place the hole for the condylar positioner. The hole is placed low down on the buccal cortex of the proximal segment using a small round drill. The hole will serve as a purchase point for the condylar positioner during condylar positioning and tightening of the holding wire (Fig. 46.43e).
8. Splitting the mandible. The actual splitting of the mandible can be divided into two stages. The first stage involves the initiation of the split. A 10 mm thin but rigid osteotome is used to tap along the vertical osteotomy line. Keep the osteotome parallel to the buccal cortex and support the mandible at the inferior border with the channel retractor preventing any damaging forces to the condyle.

During the first stage the surgeon should see the proximal segment separate from the distal segment at the lower border and see that the neurovascular bundle is intact and separates from the proximal segment. In the second stage the split is completed. The surgeon places a large osteotome superiorly into the vertical osteotomy and a small Reyneke sagittal splitter low down into the buccal osteotomy. Rotate both instruments and visualize that the lower border continues to split towards the proximal segment, the neurovascular bundle detaches from the proximal segment as the split continues, and the inferior alveolar foramen and proximal part of the canal detach from the proximal bone segment. If the neurovascular canal does not detach from the proximal segment, the splitting procedure should be stopped and the bundle very carefully dissected from the bone. An unfavorable split can be avoided by meticulously adhering to the surgical steps. It is advisable, in cases where the split does not proceed favorably, to stop the procedure and identify the problem. It is much easier to salvage the split if the potential problem is diagnosed early. The following are unfavorable splits that may mostly occur:

- Fracture of the buccal cortex of the mandibular body. Problem: failure to complete the buccal osteotomy at the inferior border and to include the lingual cortex in the osteotomy. Solution: redefine the osteotomy at the lower border and recapture the inferior part of the segment to be included in the buccal cortex of the proximal segment (Fig. 46.45a).
- Fracture of the buccal cortex involving body and ramus of the mandible. Problem: failure to

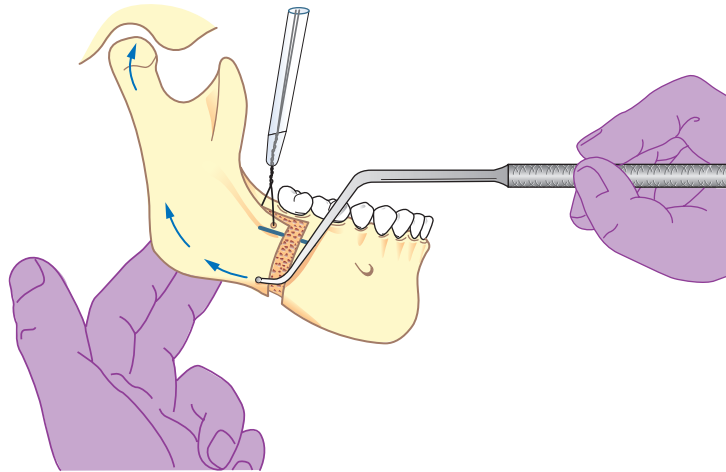


Fig. 46.46 The condylar positioner is engaged into the purchase hole and, by digital pressure on the mandibular angle, the condyle is carefully pushed backward and superiorly to seat the condyle into the glenoid fossa. At the same time the reference marks are used to ensure proper alignment of the lower borders of the segments. The holding wire is then tightened. The wire assists the surgeon to place rigid fixation without the burden of holding the segments in position during placement of rigid fixation.

- include the lingual cortex in the buccal osteotomy and only diagnosing the problem near completion of the split. The fractured segment may include the coronoid process. Solution: redefine the buccal osteotomy at the lower border and recapture the buccal cortex at the lower border (Fig. 46.45b).
- Split on the medial aspect of the ramus running anterior to the lingula. Problem: the horizontal osteotomy on the medial aspect of the mandibular ramus was performed short of the lingula. Solution: redefine the medial osteotomy and extend it past the lingula. Carefully dissect the lingula and nerve from the proximal segment (Fig. 46.45c).
 - Fracture of the retromolar segment of the distal segment. Problem: usually occurs when an impacted third molar tooth is present in the area. Solution: it is advisable to remove impacted third molar teeth 9 months before orthognathic surgery is performed, however if a fracture does occur remove the tooth and use a bone plate as method of fixation (Fig. 46.45d).
9. Stripping the pterygomasseteric sling. Place a curved periosteal elevator (J-stripper) around the lower border of the distal segment and strip the muscle fibers of the masseter and pterygoid muscles from the segment. This will allow free movement of the distal segment.
 11. Smoothen the contact areas between the bone segments. Sharp alveolar bone spicules may damage the inferior alveolar nerve following bone approximation. Smoothening of the bone will also facilitate good bone contact during fixation.
 12. Placement of intermaxillary fixation. Maxillo-mandibular fixation is applied with the teeth in the planned occlusion. A prefabricated splint may be used when necessary.
 13. Removal of bone from the proximal segment. In mandibular setback cases there will be an overlap between the two bone segments. Remove the overlapping bone from the proximal segment and take care not to damage the inferior alveolar nerve.
 14. Condylar positioning. Positioning the condyle into its correct relationship to the glenoid fossa is challenging and probably the most important step of the procedure. Place the condylar positioner into the hole drilled at the lower border of the proximal segment (step 7) and while supporting the mandible by digital pressure at the angle, carefully push the condyle backwards and superiorly into the glenoid fossa (Fig. 46.46). An awareness of the anatomic relationship between the condyle and the fossa will assist the surgeon in this important step. Problem: excessive force on the proximal segment during this maneuver could displace the articular disc, lead to hemarthrosis or joint effusion, or push the condyle too far superiorly or posteriorly. The return of the condyle to its natural fossa relationship following placement of rigid fixation and release of intermaxillary fixation is called condylar sag. The change in the condylar position will lead to an apparent incorrect occlusion.²⁹ Solution: the surgeon should be able to diagnose condylar sag intraoperatively and, if it does occur, the rigid fixation should be removed and the condylar positioning step repeated.
 15. Tightening the holding wire. Use the condylar positioner and gently hold the condyle in the desired position and ensure that the reference marks (placed step 5) line up. The assistant can now tighten the holding wire by twisting the wire carefully so as not to force the segments together.
 16. Placement of rigid fixation. Bicortical screws are now placed through an intraoral approach or by an extraoral approach (using the relevant trochar). The bone segments can also be fixated by means of bone plates and unicortical screws.

17. Evaluating the occlusion. Remove the intermaxillary fixation and pull the mandible forward and laterally to allow the condyle to "settle" into its position. The occlusion is now checked by opening and closing the jaw.²⁹ In cases where a surgical splint is used the teeth should fit into the split without force.
18. Intra- and extraoral sutures. The incisions are now closed by using 3/0 chromic sutures intraorally and a 5/0 nylon suture extraorally.
19. Place intermaxillary elastics. A triangular, vertical, ¼ inch, 4 oz (6 mm, 113 g) elastic is placed in the canine region bilaterally.
20. Apply a pressure bandage. A pressure bandage for the control of postoperative swelling should remain on the face for 2 days following surgery. Physical therapy should follow removal of the pressure bandage to assist in mouth opening and reduction of swelling. The patient is referred back to the orthodontist after about 3 weeks with the elastics in place.

Genioplasty

The chin is one of the most noticeable facial structures and correction of chin deformities demands an artistic approach. The sliding genioplasty can be performed as an isolated procedure or in conjunction with other orthognathic procedures. Genioplasty is, however, not a substitute for the surgical advancement of the mandible. The osteotomy is performed at the anterior part of the inferior border of the mandible and its versatility enables the surgeon to modify the genial area in all three dimensions.³⁰ As with all surgical procedures the genioplasty procedure should be performed following appropriate treatment planning and in a step-by-step fashion.³¹

1. Infiltration with vasoconstrictor. The area of dissection is infiltrated with local anesthetic containing vasoconstrictor (epinephrine at a concentration of 1:80 000) 10 minutes before surgery.
2. Incision. The first incision is made through the mucosa only from just distal to the canine to a similar point on the contralateral side and at least 5 mm away from the attached gingival margin. Branches of the mental nerve can often be identified laterally and should be avoided. The second incision is made through the periosteum on to bone.
3. Mucoperiosteal dissection. Start the subperiosteal dissection in the center and dissect laterally to identify the mental nerves on each side.
4. Mark the reference points. Use a 701 drill and place horizontal and, if required, vertical reference marks in the center and laterally on the buccal cortex.
5. Osteotomy design. The chin shape and the position of pogonion can be altered three-dimensionally by changing the design of the osteotomy. Most

genioplasty procedures are performed to improve the antero-posterior dimensions of the chin, however by changing the angulation of the osteotomy a plane is created along which the segment will slide (Fig. 46.47). The variation of the angle of the osteotomy will lead to changes in the vertical dimension of the anterior mandible with obvious esthetic consequences. The height of the osteotomy is influenced by the roots of the incisor and canine teeth as well as the position of the mental foramen. Keep in mind that the nerve exit at the foramen is approximately 5 mm above the nerve canal containing the nerve. The transverse dimension of the chin can be altered by segmentalizing the genial segment. The width of the anterior part of the chin is altered by an ostectomy in the symphysis area while the posterior area is altered by performing an osteotomy in the center of the chin and then rotating the segments inward or outward (Fig. 46.48). The vertical dimension can be changed by either downgrafting the segment or by performing an ostectomy and then superior repositioning the segment.

6. Osteotomy of the chin. The osteotomy is performed with an oscillating saw by starting at the center and cutting laterally.
7. Placement of a holding wire. Drill a hole through the buccal cortex in the center of the segment and feed a 018 inch (0.065 mm) wire through the hole. The wire is helpful during mobilization and accurate positioning of the segment.
8. Repositioning the chin. By using the holding wire and extraoral digital pressure, the chin is now placed into the planned position.
9. Rigid fixation of the chin. The segment can be fixated either by means of two screws engaging the segment and both cortices of the symphysis of the mandible or by utilizing bone plates (Fig. 46.49a, b).
10. Suturing. The wound is sutured in two layers. Ensure accurate reapproximation of the mentalis muscles by using 3/0 chromic sutures followed by symmetrical suturing of the mucosa using 4/0 sutures.
11. Pressure bandages. To prevent swelling and hematoma formation, a pressure bandage is

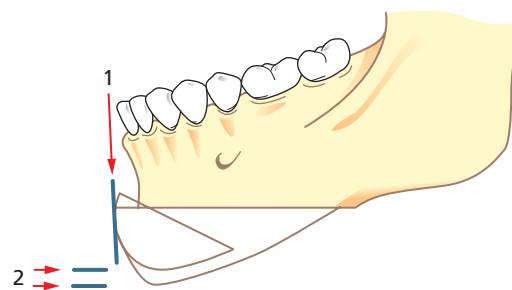


Fig. 46.47 By varying the angle of the genioplasty osteotomy, (1) the height of the chin can be altered when (2) the chin is repositioned horizontally.

placed over the surgical area. The bandages should remain in place for 3–4 days.

Le Fort I maxillary osteotomy

The first Le Fort I osteotomy was performed in Germany by von Langenbeck in 1859 for the removal of nasopharyngeal polyps.³² Cheevers described performing a Le Fort I osteotomy in 1867 using chisels to gain access to the nasal cavity.³³ The first time that a Le Fort I osteotomy was used for the surgical correction of a facial deformity was by Wassmund in 1927.³⁴ Over many years the precision of the operative technique and establishment of a scientific and biological basis for the procedure has been established.^{35,36,37} The Le Fort I osteotomy has become an essential procedure in the surgical armamentarium of the oral and maxillofacial surgeon for the correction of dentofacial deformities.

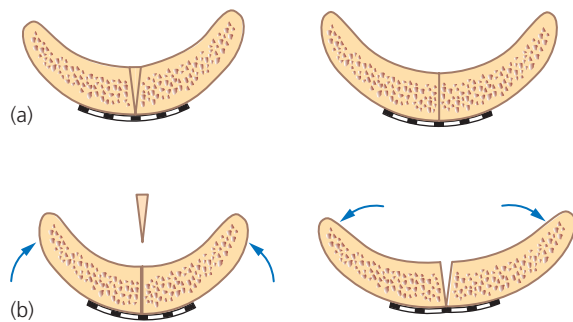


Fig. 46.48 The posterior width of the chin can be altered by performing a midline osteotomy and narrowing the chin by removing a segment of bone (a), or widened by outward rotation of the segments (b). A bone plate is placed on the chin before the segment is mobilized.

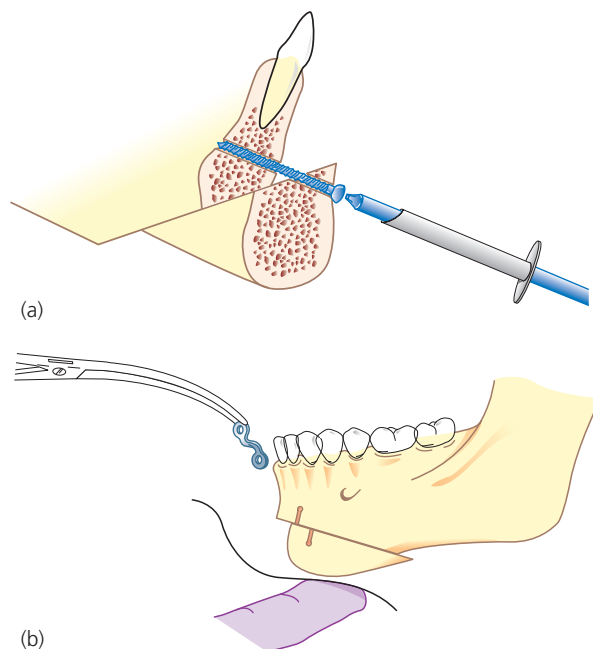


Fig. 46.49 (a) Following advancement of the chin the segment can be immobilized by tricortical fixation. (b) Plate fixation is usually preferred following posterior repositioning of the chin.

1. Soft tissue infiltration with vasoconstrictor. To assist in intraoperative control of hemorrhage, the areas of the proposed incision are infiltrated with vasoconstrictor (epinephrine at a concentration of 1:80 000) 10 minutes before surgery.
2. Soft tissue incision. The initial soft tissue incision is made through mucosa and positioned at the superior depth of the maxillary buccal sulcus, starting from the level of the second premolar, proceeding anteriorly and inferiorly to just below the anterior nasal spine. The incision is repeated and deepened to include muscle and periosteum. Care must be taken not to extend the incision beyond the second premolar or to place it in the attached mucosa as this will compromise an important source of blood supply for the down-fractured maxilla.
3. Subperiosteal dissection. Dissection of the periosteum begins by elevating the periosteum on the superior aspect of the incision. (Periosteal coverage of the maxilla should be maintained unless interdental osteotomies are planned, in which case limited periosteal elevation over the areas of interdental osteotomy is required.) Periosteal reflection proceeds to expose the posterior lateral wall of the maxillary sinus taking care not to perforate the periosteum as this will result in herniation of the buccal fat pad.
4. Reflection of nasal mucosa. Similarly, the nasal mucosa is reflected off the nasal floor and medial and lateral walls as superiorly as possible to allow osteotomies to be performed without lacerating the nasal mucosa.
5. Perform the osteotomies of the lateral walls of the maxillary sinus (Fig. 46.50). The Le Fort I osteotomy may be designed as either a stepped osteotomy parallel to the occlusal plane (if only

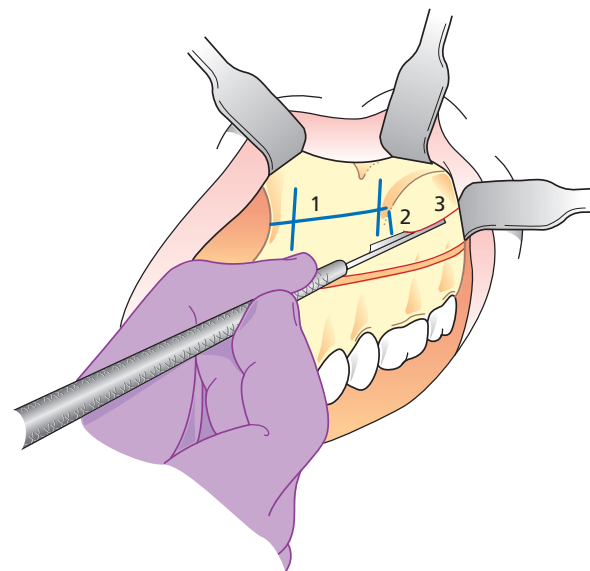


Fig. 46.50 When performing a stepped osteotomy: (1) the anterior osteotomy is performed first extending from the piriform rim to the zygomatic buttress, followed by (2) a vertical osteotomy at the buttress, and then (3) completing the osteotomy posteriorly at the maxillary tuberosity.

antero-posterior movements are required) or a straight osteotomy diverging from the occlusal plane anteriorly (if vertical movements are required). Before commencing the osteotomy the surgeon must orientate the height and angle of the proposed osteotomy, taking into account the esthetic requirements, the presurgical planning, the anatomical landmarks which guide the osteotomy and those to be avoided.^{38,39} The osteotomy is performed from the nasal rim and extended posteriorly to the zygomatic buttress. The step is then performed perpendicular to the first osteotomy running along the zygomatic buttress and continuing along the posterior lateral maxillary sinus wall to the limit of visual access. The step allows the surgeon to perform the osteotomy parallel to the occlusal plane. If a straight osteotomy is required it is a posterior continuation of the anterior osteotomy. If any vertical reduction is planned for, a second osteotomy parallel to the

first is completed at a distance determined by the preoperative planning (Fig. 46.51).

6. Place the holes for a holding wire. Holes are placed above and below the osteotomy slightly anterior and posterior to the zygomatic buttress to allow the placement of a zygomatic buttress wire.
7. Lateral nasal wall osteotomy. The lateral nasal wall is sectioned with the appropriate osteotome until the resistance of the pterygoid plates is felt.
8. Pterygoid plate osteotomy. Separation of the posterior maxilla from the pterygoid plates is completed with the appropriate osteotome positioned to engage the cleft that can be felt inferior to the pterygo-maxillary fissure. With the surgeon placing a palpating finger intraorally on the pterygoid hamulus and holding the osteotome with the other hand, the assistant percusses the osteotome until the surgeon feels the separation of the pterygoid plates intraorally. The lateral wall osteotomy

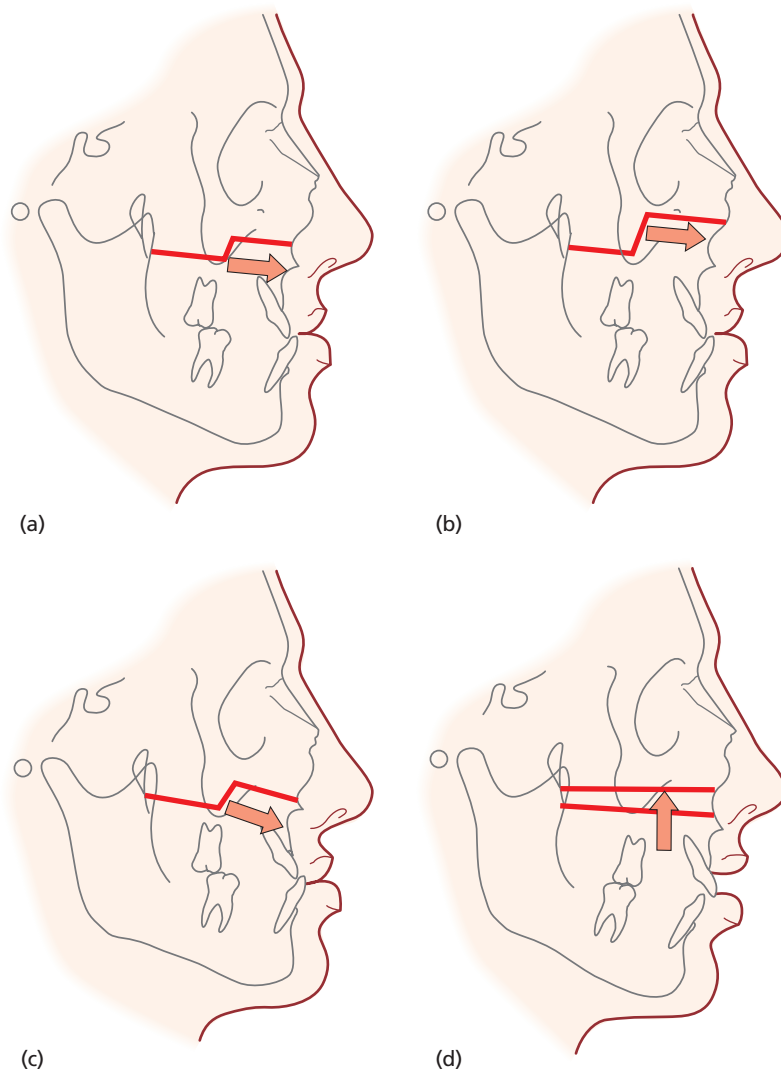


Fig. 46.51 By changing the design of the Le Fort osteotomy various geometric effects can be achieved. (a) When only maxillary advancement is required a stepped osteotomy is preferred to allow the surgeon to perform the osteotomy parallel to the occlusal plane. Advancement of the maxilla along this plane will not alter the vertical dimension. (b) Patient with more severe midface deficiency may require a high Le Fort I osteotomy. (c) Patients with maxillary antero-posterior and vertical deficiency would benefit from a Le Fort I down sliding technique. The osteotomy is performed at an angle to allow the maxilla to slide inferiorly as it is advanced. (d) If the maxilla only requires superior repositioning a straight osteotomy is performed.

is then completed with an appropriate osteotome, again carefully advancing posteriorly until the resistance of the pterygoid plates is felt.

9. Interdental osteotomies. If these are required they are performed at this stage. The periosteum is reflected in the area of the osteotomy, and the root angulations are visualized as well as possible. The osteotomy is commenced with a fine drill commencing from the Le Fort I osteotomy cut 5–6 mm lateral the piriform rim and continued inferiorly between the roots to the apex of the alveolus. This initial cut must extend no deeper than the buccal cortex. A fine osteotome is then used to complete the osteotomy through to the palate. The surgeon positions the osteotome and palpates intraorally for palatal cortex perforation while the assistant carefully percusses the osteotome until the surgeon is satisfied that the palatal osteotomy has been completed.
10. The above procedures (1–9) are repeated on the contralateral side
11. Nasal septal osteotomy. The remaining nasal mucosa is reflected off the septum and the septum is sectioned off the nasal surface of the septum with the appropriate osteotome.
12. Down fracture. The maxilla is down fractured by digital pressure applied to the anterior teeth forcing the maxilla inferiorly. If the down fracture cannot be completed, all the previously performed osseous cuts should be revised.
13. Maxillary mobilization. To passively position the maxilla, it is critical that any residual bone attachments at the pterygo-maxillary junction be severed and the adherent soft tissues are stretched. This is accomplished by means of the appropriate osteotome positioned behind the maxillary tuberosity and the careful application of an anteriorly directed force to the maxilla, taking care not to tear the lateral soft tissue pedicles of the maxilla.
14. Refinement of osteotomy edges. Any jagged edges of bone on the lateral and medial sinus walls are removed.
15. Bone reduction of the posterior maxilla (Fig. 46.52). If superior repositioning of the maxilla is required then the appropriate amount of bone needs to be removed. If possible this should be performed predominantly on the posterior of the down fractured maxilla as it is less likely to damage important blood vessels posterior to the pterygoid plates when significant hemorrhage would be encountered. With large impactions however, bone removal is also required from the pterygoid plates and into the pterygomaxillary fissure. This should be done judiciously and with great care.
16. Reduction of nasal sill and perpendicular plate of the palatine. If anterior or superior repositioning is envisaged then reduction of the nasal sill is required (more for the latter) to obviate against negative esthetic nasal changes. Moreover to prevent septal buckling the requisite amount of septal reduction from the nasal floor is required.
17. Segmentation. If planned, the necessary segments are completed from the superior aspect of the palate and alveolus.
18. Place intermaxillary fixation. The maxilla is secured to the mandible either directly or via a splint with stainless steel wires.
19. Placement of buttress wires. If these are to be used for fixation at the buttress then they are placed now. Multipiece maxillary surgery and downgrafting require rigid fixation at the buttress and in such cases the placement of a buttress wire is omitted.
20. Maxillary positioning. The maxillomandibular complex is now positioned, ensuring that the mandibular condyles are seated and the maxilla achieves the desired position.
21. Application of rigid fixation. With the maxilla in position and held either by buttress wires or manually, titanium plates are adapted to the piriform rims and buttresses and secured with screws.
22. Grafting of defects. At this stage any defects present due to either downgrafting or large advancements should be grafted.
23. Checking of occlusion. The intermaxillary fixation is removed and the mandible allowed to find its neutral condylar position. Light digital pressure on the menton is used to approximate the mandible to the maxilla. Should the occlusion not correspond to the preplanned occlusion, the maxilla should be released, repositioned following reapplication of the maxillomandibular fixation, and resecured.
24. Cinch suture. Negative nasal changes (widening of the nasal base and lifting of the nasal tip) particularly following maxillary advancement and superior repositioning, can be counteracted by the placement of a cinch suture.
25. Soft tissue closure. The incision is closed in layers, first approximating the facial muscles and thereafter mucosal closure is completed.

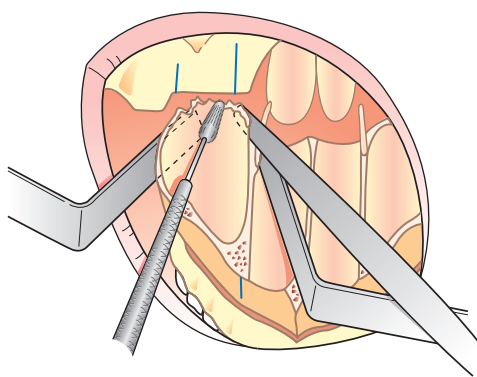


Fig. 46.52 Bone should be removed from the posterior part (tuberosity) of the down fractured maxilla when required. This will avoid damage to important anatomical structures behind the pterygoid plates.

26. Place intermaxillary elastics. A triangular, vertical, ¼ inch, 4 oz (6 mm, 113 g) elastic is placed in the canine region bilaterally.
27. Apply a pressure bandage. A pressure bandage for the control of postoperative swelling should remain on the face for 2 days following surgery. Physical therapy should follow removal of the pressure bandage.

Simultaneous orthognathic surgery and rhinoplasty

The nasal airway is often obstructed with mucus, edema, nasal packing, and blood clots following nasal reconstruction. With the use of rigid fixation the oral airway can, however, be maintained enabling the surgeon to perform certain rhinoplasty procedures concurrent to orthognathic surgery. The orthognathic procedures are first completed whilst the patient is intubated via the nasotracheal route. This is then changed to an oral endotracheal tube to facilitate the rhinoplasty procedure. Rhinoplasty should not be performed as the first surgical procedure. The Le Fort I maxillary osteotomy allows for excellent access to internal nasal structures and procedures such as turbinectomy, septoplasty, and removal of nasal polyps.

Certain maxillary procedures, such as superior repositioning and advancement of the maxilla, have a tendency to affect the shape and size of the nose and paranasal anatomy. Control of these effects requires trimming of the septum, contouring of the piriform rims, and placement of a cinch suture. The authors prefer not to correct external nasal deformities at the same time as Le Fort I maxillary osteotomies but rather to defer it as a secondary procedure. Harvesting of nasal septal cartilage for the use of grafting at a secondary rhinoplasty procedure is easily performed during the orthognathic surgery procedure with the maxilla in the down fractured position. The harvested cartilage can be "stored" by suturing it under the buccal mucosa following the Le Fort I procedure and can be recovered at the time of nasal surgery. When only lower jaw surgery is performed, rhinoplasty can be safely performed concurrently as no absolute nasal effects are to be expected from mandibular surgery.

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Chapter 47

Orthognathic Surgery in Obstructive Sleep Apnea

Scott B. Boyd

The objective of this chapter is to provide guidelines for the evaluation of patients with obstructive sleep apnea (OSA) and their treatment with orthognathic surgery. Evaluation of the patient to confirm the diagnosis is an important issue and different diagnostic tools are listed. The principles of treatment and alternative methods are discussed in the chapter. Maxillomandibular advancement in adults is the clinically most effective application of orthognathic surgery for treatment of OSA. A treatment protocol for children with OSA is also included in the chapter.

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Introduction

Orthognathic surgery is most commonly used to correct developmental or acquired maxillofacial skeletal deformities (MSD). Over the past two decades, orthognathic surgery has increasingly been used for the surgical treatment of individuals with obstructive sleep apnea (OSA). Although similar orthognathic surgical techniques are used, there are multiple important differences that exist between MSD and OSA patients. It is essential for the treating surgeon to understand these differences to facilitate

effective and safe surgical care for the OSA patient. Important differences between the two groups include: goals of therapy, patient profile, underlying medical conditions, and magnitude of surgical movement. Most MSD patients are adolescents or young adults in good general health. In contrast, the typical OSA patient is a middle-aged, obese male, with significant comorbid medical conditions. The OSA patient has anatomic abnormalities of the upper airway and larger surgical movements (10mm or greater) of the mandible and maxilla are routinely required to effectively treat obstruction of the upper airway during sleep.

The major objective of this chapter is to provide practical guidelines for the evaluation of patients with OSA, and treatment by orthognathic surgery. These guidelines are based on a critical review of the current literature and the author's personal experience. The presentation will focus on maxillomandibular advancement (MMA) for adults, as this is the most common and clinically effective application of orthognathic surgery to treat OSA. Adolescents can be treated using essentially the same protocol. In children the etiology of OSA and surgical treatment differ from those for adults and an overview of management of the pediatric patient will be provided.

Obstructive sleep apnea

Epidemiology and pathophysiology

OSA is a common primary sleep disorder that occurs in up to 9% of women and 24% of men aged 30–60.¹ Middle-aged, obese males are individuals most at risk to develop OSA. OSA is a condition characterized by repetitive partial or complete upper airway collapse during sleep. The ensuing reduction in airflow leads to hypoxia and subsequent arousals from sleep, producing sleep deprivation. The major symptoms of OSA are excessive daytime sleepiness, loud snoring, witnessed stop-breathing episodes, and non-restorative sleep. The effect that OSA has on general

health and well-being has been well documented.^{2,3} OSA is associated with hypertension,⁴⁻⁶ cardiovascular disease,⁷⁻¹⁰ metabolic syndrome,^{11,12} stroke, and possible premature death.¹³ There is a reduction in quality of life,^{14,15} including diminished social function and an increased rate of motor vehicle accidents.¹⁶ Deficits in neuropsychological functioning occur, including diminished vigilance, executive functioning, and motor coordination.¹⁷

Diagnosis and definitions of OSA

Nocturnal polysomnography (PSG) is the established standard technique to objectively diagnose OSA.^{18,19} PSG evaluates sleep disordered breathing (SDB), sleep architecture, and oxygen desaturations. The primary measure of SDB is the apnea-hypopnea index (AHI) which is the number of apneas and hypopneas per hour of sleep. An apnea is defined as an interruption of airflow lasting at least 10 seconds in adults or the equivalent of two breaths in children. A hypopnea is a specified reduction in airflow and an associated oxygen desaturation or arousal, lasting at least 10 seconds in adults or the equivalent of two breaths in children. The AHI is the primary measure used to determine the severity of OSA and effectiveness of treatment. OSA is classified as mild (AHI = 5–15), moderate (AHI = 15–30), and severe (AHI > 30).^{18,19}

Principles of treatment

Treatment strategies for OSA include both non-surgical and surgical forms of therapy. Most treatment protocols are designed to open the upper airway at the site of obstruction, while other modalities of therapy treat the associated obesity (e.g. weight loss, bariatric surgery). Surgical treatment is directed towards site-specific anatomic modification of the upper airway with the goal to prevent or diminish obstruction by pharyngeal collapse. Most surgical procedures attempt to treat the obstruction by one of three methods: (1) removal or reduction in size of excessive pharyngeal soft tissue structures; (2) expansion of the surrounding facial skeletal framework; or (3) bypass of the upper airway.

Continuous positive airway pressure

Nasal continuous positive airway pressure (CPAP) is considered the first and most effective form of therapy to treat OSA in adults.²⁰ CPAP is administered via a mask during the night to splint open the airway, leading to reductions in arousals, decreased sleep fragmentation, and resolution of sleep deprivation. CPAP has been shown to be highly effective treatment in individuals with moderate and severe OSA. Significant improvements in objective and subjective daytime sleepiness, quality of life, and cognitive function have been demonstrated following the use

of CPAP.^{20,21} Some studies have also shown that CPAP leads to a decrease in blood pressure and other cardiovascular events.²¹ Although CPAP has been shown to be highly effective, virtually eliminating OSA, long-term acceptance and adherence to therapy are relatively low. Reports indicate that as many as 46–83% of OSA patients are non-adherent to therapy.²² CPAP also requires lifetime nightly use.

Oral appliance

Oral appliances (OAs) are a non-surgical alternative to CPAP and may be indicated in some patients who are not able to tolerate CPAP. An OA is a device that the patient wears during sleep, that positions the mandible forward (about 10mm) for the purpose of increasing the posterior airway space. OAs are less effective than CPAP and are recommended primarily for individuals with mild to moderate apnea.^{23,24} Therapy is more effective in less obese patients and individuals who have certain craniofacial features (e.g. mandibular retrognathia).²⁵

Weight loss/bariatric surgery

Obesity is positively associated with OSA and even modest weight loss has been shown to decrease OSA. A 10% weight gain predicts about a 32% increase in AHI, while a 10% weight loss results in a 26% decrease in AHI.²⁶ Unfortunately, treatment of obesity by a structured program of weight loss shows high rates of failure and relapse with more than 90% of patients unable to maintain a 5–10% decrease in weight for more than 5 years.^{27,28} Bariatric surgery has recently emerged as an effective long-term treatment for weight reduction, as well as an effective treatment for reducing OSA, in morbidly obese patients.^{27,29} For OSA patients, bariatric surgery should be considered for those individuals with a body mass index (BMI) greater than 35.²⁹

Tracheostomy

In the past, tracheostomy was selected as the most viable of the surgical options. Similar to CPAP, it has a high level of success in eliminating OSA.³⁰ However, it has significant drawbacks in that it may increase psychosocial problems. Tracheostomy is generally reserved for those patients with severe apnea and concomitant severe cardiovascular symptoms requiring urgent correction of significant hypoxemia.

Uvulopalatopharyngoplasty

Uvulopalatopharyngoplasty (UPPP) has become the most widely used surgical procedure to treat OSA. UPPP involves the removal of tissue from the soft palate, uvula, and lateral pharyngeal walls in order to create a more open upper airway. UPPP has shown improvements in snoring and other subjective symp-

toms,^{31,32} but a relatively low rate of improvement in SDB. Unfortunately UPPP is effective in only about 41% of patients with OSA.³³ In addition to low response rates, initial improvements that do occur may diminish over time.^{34,35} Patients with mild to moderate apnea tend to have much greater responsiveness than those with severe apnea and UPPP is more effective in patients with a lower BMI.^{33,36} In some patients, OSA can actually become worse after UPPP.³⁷

Maxillomandibular advancement

One of the most successful surgical procedures in the treatment of OSA is maxillomandibular advancement (MMA). MMA involves surgical facial advancement, by performance of concomitant maxillary and mandibular osteotomies, for the purpose of creating an enlarged posterior airway space (Fig. 47.1). In general, MMA surgery yields high success, and is considered to be the most effective treatment aside from CPAP and tracheostomy.³⁸ Reported short-term success rates for MMA range from 65–100%.^{39–42} It appears that the initial improvements in SDB following MMA may be maintained on a long-term basis.^{43,44}

Some surgeons have approached MMA as a stand-alone procedure,^{42,45,46} while others have advocated a staged approach to treatment.⁴⁰ In the staged protocol, a patient diagnosed with OSA will first undergo “phase I” surgery, which includes UPPP and possibly concomitant adjunctive procedures. These adjunctive procedures may include genial advancement, hyoid repositioning, tonsillectomy and adenoidectomy, tongue base surgery, and nasal surgery. If phase I surgery is not clinically effective, the patient would proceed to “phase II” surgery consisting of MMA.

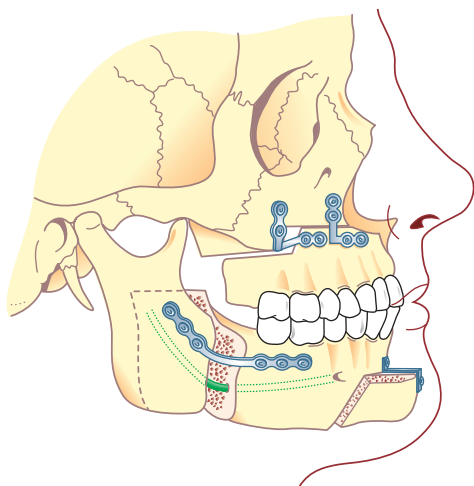


Fig. 47.1 Maxillomandibular advancement procedure consisting of LeFort I maxillary osteotomy with step modification, bilateral sagittal split ramus osteotomies, and genial advancement; stabilized with bone plate fixation. Shaded area depicts sites of anterior nasal spine reduction and piriform rim recontouring. (Adapted from Boyd SB. Management of obstructive sleep apnea by maxillomandibular advancement. *Oral Maxillofac Surg Clin North Am* 2009; 448, with permission.)

Currently it is unknown whether staged surgery provides any benefit over isolated MMA.

Patient evaluation

The purpose of the initial surgical consultation is to confirm a diagnosis of OSA and to determine if the patient is a candidate for orthognathic surgery. Commonly, the patient has already been evaluated by a sleep specialist and has undergone objective evaluation, by polysomnography, to establish a diagnosis of OSA. Furthermore, it is likely that the patient has already attempted to use CPAP. If this initial evaluation and treatment have already occurred, the surgeon should confirm the findings, otherwise an overnight sleep study will need to be obtained to objectively establish a diagnosis of OSA, prior to initiating any treatment.

Major components of the evaluation include a comprehensive history, sleep study, clinical examination, and imaging studies. Most of the evaluation is similar to a routine orthognathic surgery consultation, with the addition of components specific to OSA.

Symptoms and history

A thorough sleep-specific history and comprehensive medical history are essential components of the evaluation. Important elements of the sleep history include: presence and character of snoring, level of daytime sleepiness, self-reported or observed nocturnal episodes of breathing cessation, and the perceived quality and quantity of sleep. Valid and reliable measurement tools to evaluate sleepiness and quality of life in OSA patients are available.⁴⁷ The Epworth Sleepiness Scale (ESS) is an eight-item questionnaire that is commonly used to subjectively assess the patient's level of daytime sleepiness.^{48,49} The patient may also relate a variety of symptoms related to a decreased quality of life, such as poor job performance, decreased ability to concentrate, memory loss, and fatigue. The Calgary Sleep Apnea Quality of Life Index (SAQLI)^{50,51} and the Functional Outcomes of Sleep Questionnaire (FOSQ)⁵² are two valid and reliable sleep-specific quality-of-life questionnaires that may be used.

The patient should also be questioned about previous treatment of OSA and past response to therapy. This should include both the patient's subjective and objective (e.g. post-treatment PSG) response to therapy. Patients should also be questioned about any treatment-related adverse outcomes or complications of previous therapy.

A comprehensive medical history must be obtained because OSA is associated with a wide spectrum of medical conditions (e.g. cardiovascular disease, metabolic syndrome, psychiatric disease) that may affect surgical treatment and the patient's overall health. If there is presence or a suspicion of a significant medical condition, it will be important to

obtain indicated consultations to establish the status of the disease, determine if the patient is a candidate for surgery, determine what can be done to optimize the patient's condition prior to surgery, and to obtain recommendations for intraoperative and postoperative management of the patient with regard to each significant medical condition. This assessment is essential to determine the risk/benefit ratio for surgical intervention. One of the most common medical conditions is hypertension and it is important to optimize blood pressure preoperatively, since patients will typically have modified hypotensive anesthesia administered during surgery.

Polysomnography

When the initial consultation appointment is scheduled, the patient should be instructed to send (or bring with them to the consultation visit) the reports of all previous sleep studies. These reports are essential to establish the presence and severity of OSA. It is likely that the patient has had a CPAP titration performed and the titration results will give an indication of the effectiveness of CPAP if the patient is compliant with CPAP therapy. Furthermore, if the patient has undergone treatment of OSA, post-treatment sleep studies may have been completed to assess the result of therapy. The most recent sleep study should accurately reflect the patient's current status. If significant time has elapsed since the last study and/or the patient's symptoms (e.g. daytime sleepiness) have significantly changed (especially worsened), a repeat sleep study is indicated to accurately assess the current severity of OSA.

Physical examination

A comprehensive head and neck examination should be performed for each patient. This physical examination should include measurement of the patient's BMI, resting blood pressure, and neck circumference. The OSA clinical evaluation is very similar to a routine orthognathic surgery evaluation, with special attention directed to potential sites of upper airway obstruction.

It is important to carefully perform all aspects of the routine orthognathic surgery baseline clinical examination to facilitate surgical treatment planning and to determine if presurgical orthodontic care would be of benefit. The clinical examination should include assessment of temporomandibular joint (TMJ) function and mandibular mobility, occlusion, status of the dentition, neurosensory function, and facial esthetics. For most middle-aged adults, adaptations in the dentition have occurred (e.g. wear and restorations) to maximize occlusal relations of the teeth, and the patient will not significantly benefit from orthodontic therapy prior to surgery.

Important augmented components of the upper airway examination include: inspection of the nasal

cavity to determine sites of possible obstruction, description of the size, character, and function of the tonsils, soft palate, lateral and posterior pharyngeal walls, and base of tongue. In addition to direct visual examination, endoscopic examination (fiberoptic nasopharyngoscopy) of the upper airway may be of benefit to aid in the visualization of the upper airway and identification of the site(s) of pharyngeal collapse and obstruction.

Imaging

A standardized lateral cephalometric radiograph should be taken, with the patient positioned in adjusted natural head position with the mandible in centric relation and the facial soft tissues in repose (Fig. 47.2). A cephalometric analysis is then performed to assist in the identification of potential sites of upper airway obstruction and to characterize craniofacial morphology (Fig. 47.3). If the patient proceeds to surgery, the cephalometric radiograph will be used for both surgical treatment planning and assessment of changes in the facial skeleton and upper airway that occur as a result of surgery. The major limitation of the cephalometric radiograph is that obtaining the radiograph in an upright and awake position may not accurately reflect the anatomic characteristics of the upper airway when the patient is sleeping in a supine position.

Facial and intraoral digital photographs should be taken to document the clinical examination and may be linked to the lateral cephalometric radiograph to develop computerized surgical prediction images (Fig. 47.2). This is important because the magnitude of the surgical movement (10 mm or more of facial advancement) may have a significant impact on facial appearance and the prediction images allow the patient to see the type (not necessarily the actual result) of facial esthetic changes that may occur as a result of surgery.

Computed tomography (CT) imaging has not routinely been used for the surgical treatment of adults with OSA. However, since three-dimensional (3D) CT imaging has recently become available in an outpatient office setting (e.g. cone-beam technology), use of this technology may be beneficial because it has the ability to visualize the entire upper airway and can demonstrate the association between 3D changes in the facial skeleton, as a result of surgery, and the upper airway.

Indications for maxillomandibular advancement

Once the evaluation has been completed, it can be determined if the patient is a surgical candidate. The indications for MMA are as follows: (1) significant OSA (AHI > 15) as objectively diagnosed by PSG; (2) failure of CPAP because of non-acceptance or poor adherence to therapy; (3) craniofacial abnormali-



Fig. 47.2 Frontal facial (a), lateral facial (b), lateral cephalometric radiograph (c), and intraoral (d–f) pretreatment images of a 35-year-old man with severe obstructive sleep apnea (AHI = 49), decreased posterior airway space, retrusive facial profile and normal Class I dental occlusion.

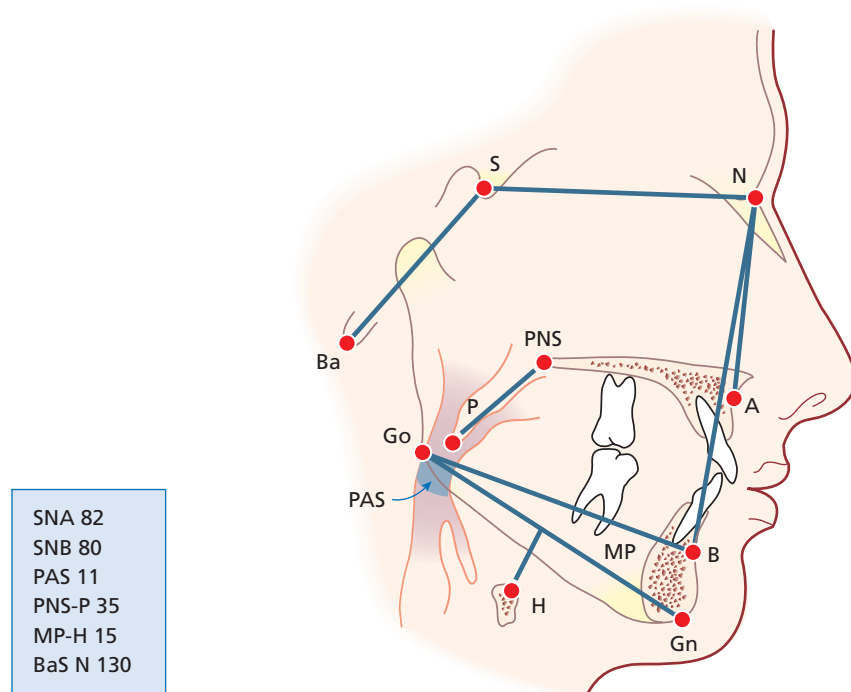


Fig. 47.3 Cephalometric analysis for obstructive sleep apnea with depiction of anatomic landmarks and associated measurements (normal values).

ties (e.g. children with micrognathia); and (4) ability to undergo surgical treatment (consideration of concomitant medical conditions). It is also important to determine if the patient is a candidate for other forms of treatment. For example, if the patient has mild OSA, oral appliance therapy could be considered as a viable non-surgical form of therapy. Bariatric surgery may be considered for the extremely obese patient (BMI > 35). Additionally, it should be determined if the patient is a candidate for surgical-orthodontic care, although this is uncommon.

Presurgical treatment planning

Most of the records necessary to perform definitive presurgical treatment planning may have already been obtained during the initial consultation visit. The results and recommendations of all consultations, obtained after the initial evaluation, should be reviewed. The current status of all significant medical conditions should then be assessed. A final determination can then be made regarding the patient's candidacy for surgery and a detailed discussion of the potential surgical, medical, and anesthetic risks of surgery should occur with the patient.

Complications of surgery in patients with MSDs have been well studied.⁵³ The most common complications observed following surgical correction of a MSD include: neurosensory changes of the lips and chin, infection, skeletal relapse, TMJ disorders, and dental injuries. Age is considered to be the strongest predictor of complications, while large skeletal movements seem to have greater relapse potential.⁵³ MMA patients typically display one or more of these predictors of complications, including older age, large surgical movements, and medical comorbidities that may negatively impact wound healing. Currently, it is unknown whether MMA patients actually experience more complications than MSD patients.

Due to the combined maxillary and mandibular movements and the large magnitude of facial advancement, it is recommended that a facebow transfer and mounting of dental casts on a semi-adjustable articulator be used to provide an accurate 3D positioning of the maxilla and mandible in relation to the TMJ. It is also recommended that model surgery be completed using a model platform to accurately measure the planned surgical movements of the maxilla and mandible, in three planes of space. The final surgical plan will be based on the patient's individual findings, including the presence or absence of a pre-existing dentoskeletal deformity, but a minimum of 10mm of mandibular and maxillary advancement is recommended to produce the most improvement in OSA. In the routine patient, who does not undergo concomitant orthodontic care, the maxilla will move an equivalent distance to the mandible, to maintain the patient's pre-existing occlusal relations.

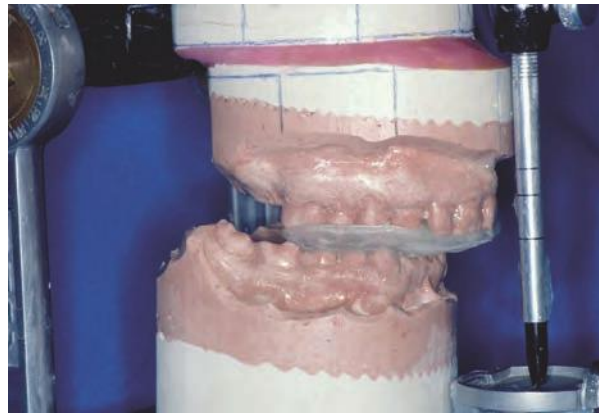


Fig. 47.4 Model surgery in preparation for maxillomandibular advancement showing interim splint after a 10mm advancement of the maxilla. (From Boyd SB. Management of obstructive sleep apnea by maxillomandibular advancement. *Oral Maxillofac Surg Clin North Am* 2009; 451, with permission.)

Following completion of the model surgery, an interim splint is constructed using the advanced maxillary cast referenced to the uncut mounted mandibular cast (Fig. 47.4). The interim splint will facilitate accurate antero-posterior and transverse positioning of the maxilla, since the recommended treatment sequence is to first perform the maxillary surgery. If the patient has an intact, stable dentition prior to surgery, it is unlikely that a final surgical splint will be necessary because the patient can be placed in a stable, reproducible occlusion following performance of the maxillary and mandibular osteotomies.

Surgical treatment

Anesthetic and medical management considerations

It is recommended that the surgeon and attending anesthesiologist discuss management of the airway, anesthetic techniques, and medical management of the patient prior to surgery to minimize the chance of perioperative complications.^{54–58} Fiberoptic nasopharyngeal intubation (possibly awake) with a RAE tube provides a secure airway and ample access to the surgical field. A tracheostomy is not routinely indicated to secure the airway, but is recommended where there is concern about the ability to safely perform nasopharyngeal intubation or where long-term postoperative airway management is required. Proper patient positioning and padding (e.g. gel pads) are important to reduce the risk of pressure ischemia, which may be increased due to size (BMI) of the patient, use of hypotensive anesthesia, and length of the procedure. Judicious intravenous fluid administration and use of intraoperative corticosteroids are helpful to diminish postoperative facial and parapharyngeal edema.

It is advantageous to use a modified hypotensive anesthetic technique during performance of the maxillary and mandibular osteotomies, to reduce blood loss, and to improve visualization of the surgical field.⁵⁹ The ability to achieve this level of reduction in blood pressure is dependent on the patient having a near normal blood pressure preoperatively and underscores the importance of adequately treating any hypertension that was identified at the consultation visit. In addition to hypertension, patients with OSA may have a history of ischemic heart disease, myocardial infarction, and possibly stroke, and in these individuals it is especially important to maintain adequate organ perfusion. Although it is uncommon that blood transfusion will be necessary intraoperatively, the patient is presented the option of donating 2 units of autologous blood, so blood will be available if needed.

Surgical technique and sequencing of care

The recommended sequencing of surgical care is as follows: (1) application of maxillary and mandibular arch bars; (2) total Le Fort I maxillary osteotomy; (3) bilateral sagittal split ramus osteotomies of the mandible; and (4) genial advancement if indicated.

A Le Fort I total maxillary osteotomy is performed using standard techniques with a modified step design (Fig. 47.5). The purpose of the step modification of the maxillary lateral wall osteotomies, is to facilitate bony interfacing and presumably enhance the stability of the maxillary advancement. After completion of the osteotomy, the maxilla is mobilized until it can be passively positioned forward to the planned surgical position, as verified with the interim splint. To facilitate complete mobilization, slow, deliberate “controlled force” is used, usually in conjunction with a Rowe forceps. Force is modified accordingly, to maintain adequate perfusion to the maxilla. Adequacy of perfusion of the maxillary soft tissues is visually monitored during the mobilization. The interim splint is then placed between the maxilla and mandible and the complex is wired into maxillomandibular fixation (MMF). The complex is then passively rotated superiorly to the proper vertical position, as confirmed by measurement to an external reference pin. The piriform rim is recontoured and the anterior nasal spine is reduced (Fig. 47.1) to minimize overprojection and widening of the nasolabial soft tissues, that may occur as a result of the large advancement of the maxilla.

The maxilla is then fixated by placement of 2.0 mm L-shaped bone plates at the piriform rim and

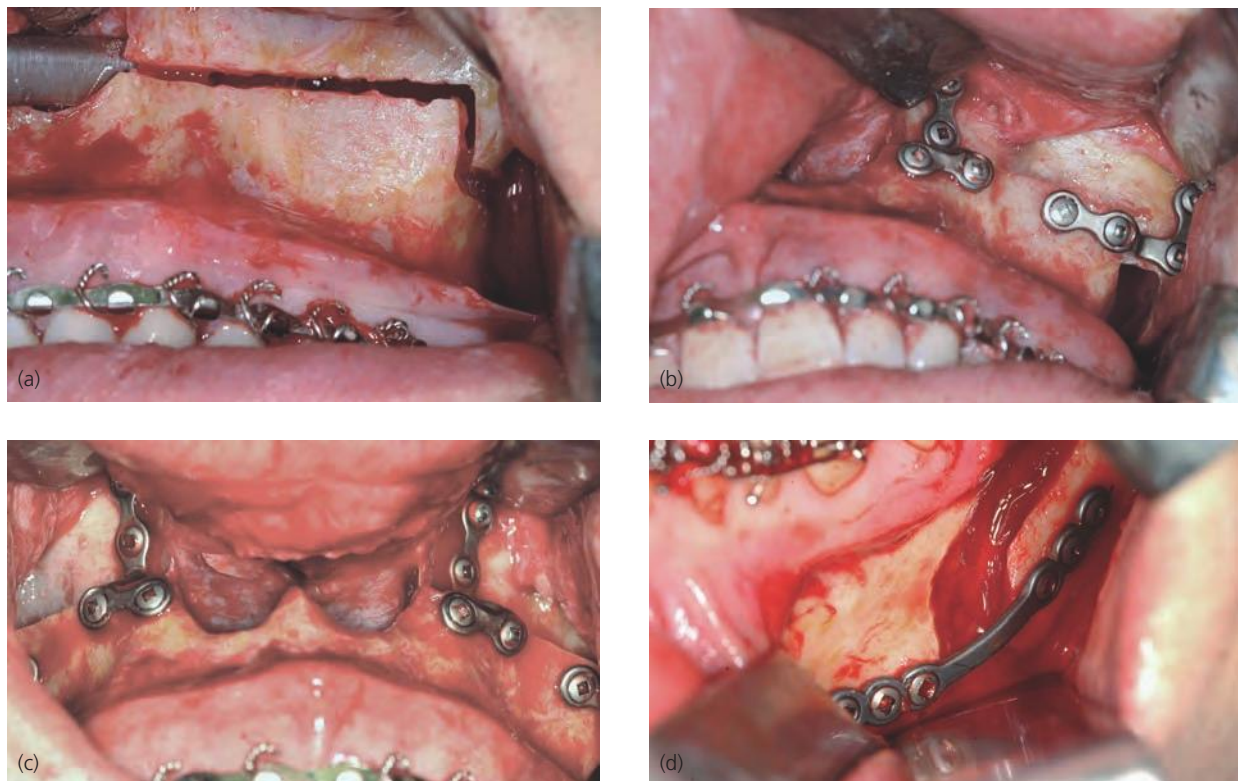


Fig. 47.5 Total LeFort I maxillary osteotomy with modified step design of lateral wall (a), placement of maxillary bone plates at piriform rim and zygomaticomaxillary buttress (b), reduction of anterior nasal spine and recontouring of piriform rims (c), and placement of mono-cortical bone plate fixation of the mandible, to minimize risk of injuring the inferior alveolar nerve (d). (From Boyd SB. Management of obstructive sleep apnea by maxillomandibular advancement. *Oral Maxillofac Surg Clin North Am* 2009; 452, with permission.)

zygomaticomaxillary buttress regions bilaterally (Fig. 47.5). The configuration of these plates produces a buttressing effect, which should enhance the stability of the large maxillary advancement together with adequate bone interfacing and good immediate stability of the maxilla. Using this technique, the author has observed very good long-term stability of the maxilla and has not found it necessary to place any bone grafts. The potential benefits of not having to perform a bone graft include: decreased operative time, elimination of any donor site morbidity, and earlier patient ambulation after surgery.

Following confirmation that the maxilla is in the proper anatomic position (as verified by the interim splint), the maxillary soft tissue wound is then closed. An alar base cinch suture (3/0 non-resorbable suture) is placed to reapproximate the alar bases at the preoperative position. A V-Y closure of the upper lip (at the midline) is then completed. These two techniques are designed to maintain proper anatomic position of the nasolabial tissues.⁶⁰

Bilateral sagittal split ramus osteotomies (BSSRO) of the mandible are then completed using standard techniques. The mandible is split using a slow deliberate method of controlled force to facilitate visualization of the inferior alveolar nerve and diminish the chance of injury to the nerve. Minimizing surgical trauma to the inferior alveolar nerve is especially important, since OSA patients are generally middle-aged and presumably have a decreased ability to recover from a nerve injury. Once the split is completed, care is taken to maintain soft tissue attachments so the entire hard tissue–soft tissue complex is advanced, for the purpose of increasing the posterior airway space. The mandibular osteotomies are then stabilized in the advanced position by either bone plates and monocortical screws or three bicortical screws (Figs 47.1, 47.5).

A genial advancement is not routinely performed, but if indicated will be performed following completion of the Le Fort I and BSSRO. A variety of methods has been used for genial advancement, ranging from a standard genioplasty to a more isolated genial tubercle advancement. The main objective of the procedure is to advance the genioglossal musculature for the purpose of advancing the tongue and presumably increasing the posterior airway space.

Postoperative care and monitoring

Once the surgical procedure has been completed and the patient is sufficiently awake to meet criteria for extubation, the nasopharyngeal tube is typically removed in the operating room. This protocol has the advantage of having both the treating anesthesiologist and surgeon present at the time of extubation, as well as all necessary equipment immediately available if reintubation is necessary. Using this protocol, it is very uncommon for a patient to require reintubation. After extubation and stabilization, the patient is

transported to the ICU for overnight monitoring. The patient's airway and associated medical conditions will need to be closely monitored. The patient is closely observed for any apneic events and oxygen supplementation is administered by face mask to maintain adequate oxygen saturation. Using this protocol, few apneic episodes are generally observed and adequate oxygen saturations can be maintained, so postoperative CPAP has not routinely been used.

It is not uncommon to observe postoperative hypertension and appropriate antihypertensive agents should be used to keep the patient at a normotensive state. For patients with pre-existing hypertension, or any other significant medical conditions, it is beneficial to develop a plan preoperatively, in conjunction with the patient's physician (cardiologist or primary care physician), for medical/pharmacological control of postoperative blood pressure. For the first postoperative night, pain is controlled through the use of incremental intravenous dosing of narcotic analgesics. Close observation of the airway occurs during administration of the analgesics as respiratory depression may occur at even low doses of narcotics in OSA patients.

Typically the patient will be stable enough to be transferred to a step-down unit on the first postoperative day. Pain control can be maintained by a patient-controlled anesthesia (PCA) pump or liquid medications administered orally. Normal postoperative recovery will be initiated including ambulation and consumption of a liquid diet, similar to a typical orthognathic surgery patient. It is recommended that night time oxygen saturations be monitored by pulse oximetry until the patient can maintain near normal oxygen saturations on room air while sleeping.

The typical hospital stay is 2–3 days. The patient will then be evaluated on an outpatient basis every 1–2 weeks for about 6–8 weeks following surgery. During these visits, wound healing, neurosensory function, skeletal stability, occlusion, range of mandibular mobility, and TMJ function will be assessed, and the patient will be instructed in wound care and rehabilitation of masticatory function. When healing is complete and the patient has regained normal jaw function, the arch bars will be removed under sedation and local anesthesia, in an outpatient clinic setting. A nocturnal polysomnography study, cephalometric radiograph, and photographs should be completed about 3–6 months following surgery to objectively evaluate treatment outcome (Fig. 47.6).

Pediatric considerations

For children, there are important special considerations in the evaluation, treatment planning, and surgical techniques that are different from the typical adult with OSA. During the initial consultation visit, it is important to obtain a comprehensive medical history. In the pediatric population, several congenital craniofacial syndromes are associated with OSA.



Fig. 47.6 Frontal facial (a), lateral facial (b), lateral cephalometric radiograph (c), and intraoral (d–f) images of patient shown in Fig. 47.2, at long-term evaluation 8 years following MMA. The patient had total resolution of OSA (long term post-MMA AHI = 0.2), increased posterior airway space, improvement in facial profile and maintenance of normal dental relations. Clinical examination showed normal sensation of the inferior alveolar nerves, normal range of mandibular mobility (60 mm) and normal temporomandibular function. The Epworth Sleepiness Scale showed no significant sleepiness (ESS = 4), and the SAQLI disease specific quality of life survey demonstrated a high positive effect of MMA and no significant treatment-related symptoms.

These syndromes include: Pierre-Robin sequence, Treacher Collins syndrome, hemifacial microsomia, and Nager's syndrome. Micrognathia is a common finding in many of these syndromes. Mandibular elongation by mandibular distraction osteogenesis (DO) has been shown to effectively treat micrognathia and concomitant OSA in children.⁶¹

Due to the underlying significant anatomic abnormalities in the craniofacial skeleton and upper airway in the young child with OSA, it is very beneficial to obtain CT imaging with 3D reconstructions for evaluation and surgical treatment planning. During the course of mandibular distraction, an interval 3D CT scan can be performed, to assess the magnitude and 3D characteristics of the distraction, and the concomitant changes in the upper airway.

The most common mandibular DO surgical technique includes bilateral mandibular distraction osteogenesis using an angle oblique osteotomy design.⁶¹ Following performance of the osteotomies, bilateral or multidirectional distracters are placed extraorally. Typically the distraction protocol includes a 3–7-day latency period followed by distraction at a rate of about 1.0 mm per day. A consolidation period of about 6–8 weeks occurs after completion of the mandibular distraction.

A recent meta-analysis showed that mandibular DO prevented the need for tracheostomy in 91.3% of neonates or infants and 97% of children showed resolution or improvement in their OSA symptoms.⁶¹

Acknowledgment

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Chapter 48

Distraction Osteogenesis

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Andrew Ow, and Li-wu Zheng

This chapter provides a comprehensive overview of the background history and development of distraction osteogenesis, along with a discussion of its possible practical applications in the future. The different methods of distraction, extraoral and intraoral, are described in detail, and the biological process of the distraction procedure is explained. The advantages of distraction are discussed, including: (1) no requirement for bone graft; (2) simultaneous expansion of surrounding soft tissue; (3) it is possible to repeat the surgery; (4) it is a simple technique with minimal blood loss. The wide use of the method in craniofacial cases and intraoral alveolar bone distraction is clarified.

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Introduction

Distraction osteogenesis is a clinical tissue engineering method of regenerating new bone by gradual stretching of divided bone segments with the use of a mechanical device. It has been called osteodistraction, callotasis, callus distraction, and bone lengthening in the literature. This technique has generated enormous interest among oral and maxillofacial surgeons because of the possibility of treating severe craniofacial deformities beyond the clinical limits of conventional orthognathic surgery. In contrast to the conventional orthognathic surgical techniques, distraction offers a few advantages including: (1) there is no requirement for bone grafts and hence no donor site morbidity; (2) it allows simultaneous expansion of the surrounding soft tissues; (3) it is feasible to repeat the distraction or surgery on the same site after distraction; and

(4) it is a simple technique with minimal blood loss that can be applied to children and infants.

History and development

This technique was initiated by surgeons in the late 1880s, and Codivilla of Bologna, Italy was the first to publish a case report in 1905.¹ He applied external traction following an elective osteotomy of the femur by hanging a weight at the end of the bed. In 1921, Putti² designed a unilateral device for femoral lengthening. He made use of large half-pins and an external telescoping tube with a compressed external spring. In 1927, Abbot³ modified the method and this became the accepted technique in distraction of long bones. He utilized a step osteotomy in conjunction with through and through pins attached to a frame on both

sides of the limb. The limb was slowly lengthened via a compressed spring in an external device. Due to a high complication rate of non-union and fibrous union, the technique gradually became obsolete.

It was not until 1954 that Dr Gavriel O. Ilizarov, a Russian orthopedic surgeon, revitalized the technique by researching on distraction osteogenesis for correction of post-traumatic deformities of the lower extremities.⁴ After World War II, a lot of Russian veterans were sent to Siberia for rehabilitation and management of problems such as osteomyelitis and malunion from war injuries experienced during the war. Dr Ilizarov developed a ring-shaped external fixator utilizing Kirschner wires, later called the “Ilizarov ring”. He used this device to treat patients with the technique of tension-wire external skeletal fixation. He admitted in his book that one of the patients wearing the Ilizarov ring turned the screws for uprighting the malaligned segment in the reverse direction and this resulted in lengthening of the leg without any complication. This accidental discovery of the possibility of achieving limb lengthening with his devices paved the way for developing many distractors achieving lengthening of different long bones. During the next 30 years, Ilizarov perfected this clinical technique of distraction bone lengthening osteogenesis.⁵⁻⁸ Through clinical experience and extensive animal experiments, he defined the factors contributing to consistent success by using a corticotomy with preservation of the periosteal and medullary blood supply to the bones, and bone expansion in small increments of 1mm per day in four rhythms. The Ilizarov technique started to become popular initially in Russia and later worldwide, particularly after Dr Ilizarov lectured on his successful experience at a surgical symposium in New York in 1988.

Development of distraction applications on cranio-maxillofacial skeleton

The feasibility of achieving distraction osteogenesis in intramembranous bones, such as the cranio-maxillofacial region, was first reported in 1972, when Snyder used a Swanson external fixation device to lengthen a canine mandible. In his experiment, Snyder surgically removed a 1.5cm segment of one side of the mandible.⁹ The bone was allowed to heal and a severe crossbite developed. This was corrected 10 weeks later by performing an osteotomy, applying an external fixator and gradually relengthening the mandible. After 14 days, the original occlusal relationship was restored. In 1976, Michieli and Miotti modified Snyder’s work by using an intraoral device and achieved similar success.¹⁰ In 1984, Kutseviak and Sukachev took the experiment further by lengthening a normal canine mandible 1.2cm using the Ilizarov principle.¹¹ These studies prompted Karp *et al.*^{12,13} at New York University to perform a detailed

histological analysis of the ossification process following mandibular distraction in a canine model, in order to confirm the stages of distraction healing in intramembranous bones; these bones are different embryonically from the long bones that ossify by endochondral ossification. This laboratory work eventually paved the way for the first four cases of human mandibular distraction published by McCarthy in 1992.^{14,15} The results of these studies were of great clinical significance because they proved that mandibular distraction can be achieved successfully with minimal risk of complications.

Biological process of distraction osteogenesis

Distraction osteogenesis consists of five sequential stages: osteotomy, latency, active distraction, consolidation, and remodeling. The inflammation and granulation tissue formation at the initial stages of distraction osteogenesis are similar to fracture healing until they are interrupted by traction forces. The collagen tissues are stretched when a traction force is applied to the bone segments. This traction force generates tension in the tissues that connect the bone segments, which stimulates new bone formation parallel to the vector of distraction.¹⁶

Osteotomy is the surgical separation of a bone piece into two segments. Discontinuity of a skeletal segment results in bleeding and formation of a blood clot. This triggers a biological process of bone repair known as fracture healing. This process involves recruitment of osteoprogenitor cells, followed by cellular modulation or osteoinduction, and establishment of an environmental template (osteoconduction). As a result, a reparative callus is formed within and around the ends of the fractured bone segments.

Latency is the period from osteotomy to the onset of active distraction. It represents the period allowing the reparative callus formation. The sequence of events occurring during the latency period is similar to that seen during fracture healing. Following the surgical separation of a bone into two segments, a cascade of events takes place.

Initially, as a result of bony disruption and fixation with a distractor, a hematoma forms between and around the bone segments. The hematoma is converted to a clot followed by an ingrowth of vasoformative elements and capillaries for the restoration of blood supply. The clot is replaced with granulation tissue consisting of inflammatory cells, fibroblasts, collagen, and invading capillaries (Fig. 48.1a).

Inflammation is followed by the soft callus stage, which is marked by a continuous ingrowth of capillaries into the callus. A minicellular network of growing capillary loops is formed in the medullary canal of both proximal and distal segments in the areas adjacent to the osteotomy line. Less differentiated, free circulating osteogenic cells are located inside the

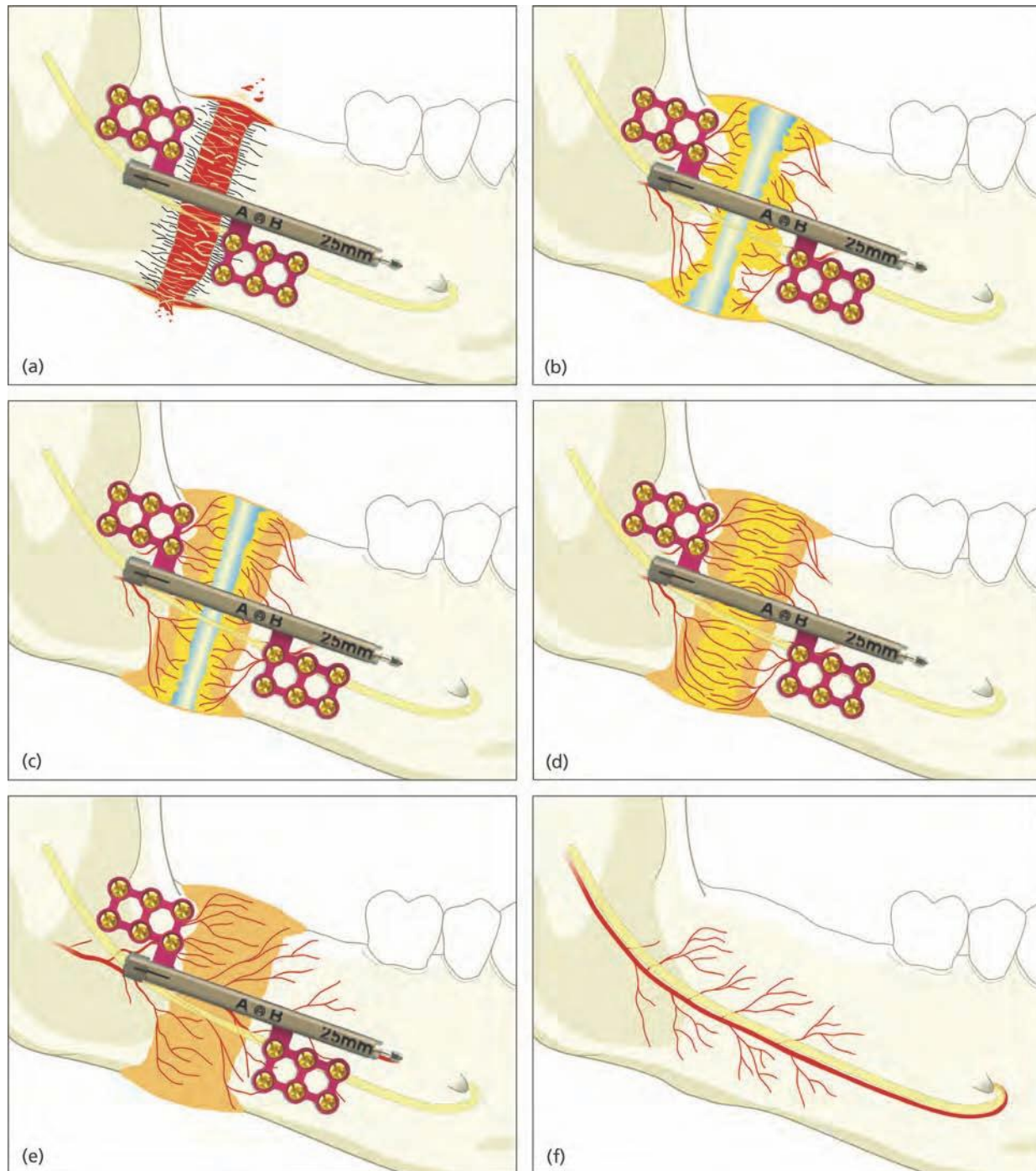


Fig. 48.1 The biological sequence of bone regeneration in distraction osteogenesis. (a) Hematoma is replaced by granulation tissue in latency period. (b) Progressive enlargement of gap during active distraction. (c) Osteogenesis with fine trabeculae at margin of distraction gap. (d) Extension of osteogenesis to the center of the gap in consolidation period. (e) Maturation of distraction regenerate in late consolidation period. (f) Remodeling of bone and re-establishment of inferior alveolar canal.

terminals of the newly formed capillaries. During the soft callus stage, granulation tissue is converted to fibrous tissue by fibroblasts. Cartilage also replaces the granulation tissue.

Active distraction is the period characterized by movement of the osteotomized bone segments by a mechanical distractor. Bone segments are gradually pulled apart, resulting in formation of new bony tissues within the progressively increasing intersegmentary gap (Fig. 48.1b).

The growth-stimulating effect of tension activates a cascade of biologic elements. This includes prolongation of angiogenesis with increased tissue oxygenation, and increased fibroblast proliferation with intensification of biosynthetic activities. Capillaries grow into the fibrous tissues, thereby extending the vascular network, not only toward the center of the gap but also toward the medullary canal of the adjacent bone segments. Capillary terminals actively invade the fibrous tissues, supplying them with

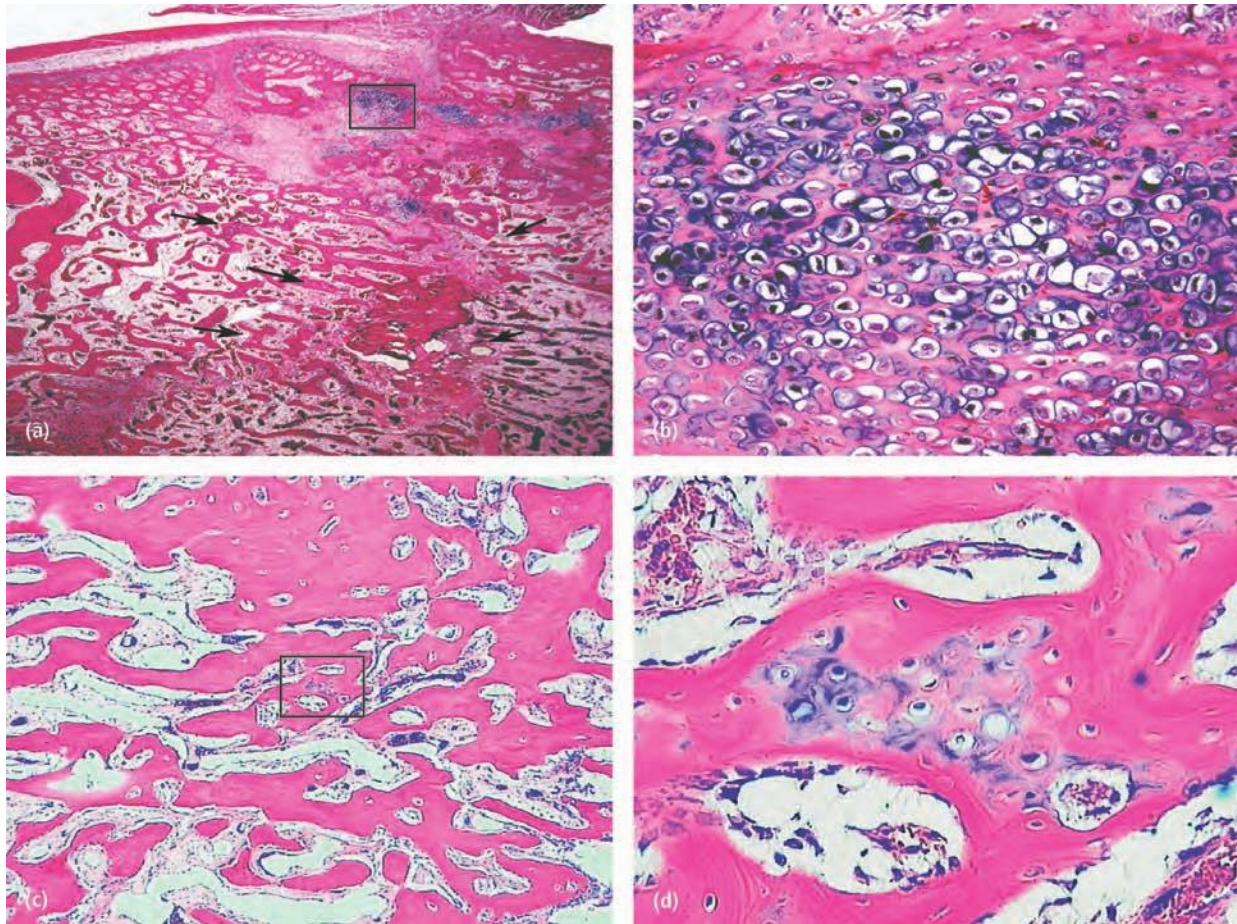


Fig. 48.2 The ossification pathways of distraction osteogenesis. (a) Distraction regenerate forms predominantly via membranous ossification (arrows) while endochondral ossification occurs occasionally in the peripheral and central area (frame). (b) At higher magnification of the frame in (a), a cartilage island is clearly seen. (c) Transchondroid bone formation. Focal region of chondrocytes (frame) surrounded by a mineralized matrix. (d) At higher magnification of the frame in (c), the cartilage cell foci are clearly visible.

mesenchymal cells that differentiate into fibroblasts, chondroblasts, or osteoblasts.

The osteoblasts, located among the collagen fibers, lay down osteoid tissue and eventually become enveloped as bone spicules, which gradually enlarge by circumferential apposition of collagen and osteoid. Primary trabeculae begin to form. Osteogenesis is initiated at the existing bone walls and progresses toward the center of the distraction gap (Fig. 48.1c).

Consolidation is the period between cessation of traction force and removal of the distraction device. It represents the time required for completing the mineralization of the distraction regenerate. After distraction ceases, the fibrous interzone gradually ossifies and one distinct zone of woven bone completely bridges the gap (Fig. 48.1d). As the regenerate matures, the zone of primary trabeculae significantly decreases and later is resorbed completely (Fig. 48.1e).

Remodeling is the period from the application of full functional loading to the complete remodeling of the newly formed bone. During this time, the initially formed bony scaffold is reinforced by parallel-fibered lamellar bone. The cortical bone and marrow cavity are restored. Remodeling of the Haversian system normalizes the bony structure into cortical and cancellous bone. Finally, the structure of newly formed

bony tissue is comparable to and indistinguishable from that of the pre-existing bone (Fig. 48.1f).

There are two basic modes of mineralization of the distraction regenerate. Endochondral ossification means bone mineralization is achieved via a cartilaginous intermediate. This is the mechanism responsible for bone mineralization in the long bone. Intramembranous ossification means direct bone mineralization without a cartilaginous intermediate and is a specific mechanism responsible for mineralization of cranio-maxillofacial bones. Distraction regenerate forms predominantly via intramembranous ossification (Fig. 48.2a), whereas endochondral ossification occurs occasionally in the periphery or any ischemic tissue areas. (Fig. 48.2b).⁷ Beside the two principal bone formation pathways, a third bone formation method, transchondroid bone formation, can occasionally be observed during the bone healing process of distraction osteogenesis.¹⁷ In this form of mineralization, focal regions of chondrocytes are surrounded by a mineralized matrix, and then directly transformed into bone rather than by the usual endochondral pathway (Fig. 48.2c). The matrix of this chondroid bone is more similar morphologically to bone than cartilage, but the cells are indistinguishable from chondrocytes (Fig. 48.2d). Factors resulting in low

oxygen tension within the distraction regenerate, such as unstable fixation and ischemia, may cause development of a cartilaginous intermediate during distraction osteogenesis.

Mandibular distraction

Indications for mandibular distraction osteogenesis

The initial application of mandibular distraction osteogenesis was mainly directed towards lengthening the ramus and body of the mandible in micrognathia secondary to congenital conditions such as hemifacial microsomia or Treacher Collins syndrome. It was also used in relieving airway distress in neonates suffering from congenital conditions such as Pierre Robin sequence. Its success in treating congenital deformities led to its application in adults where orthognathic surgery would traditionally have been used. These included cases of developmental class II mandibular hypoplasia and patients with obstructive sleep apnea (OSA). Mandibular distraction osteogenesis has also been used to treat transverse mandibular width discrepancy, temporomandibular joint (TMJ) ankylosis (by transport distraction), and moderate to severe alveolar ridge deficiency (by alveolar widening or increase in height). The common indications for mandibular distraction osteogenesis are listed in Table 48.1.

The current applications of distraction osteogenesis for the mandible include: lengthening of the mandibular body and ramus, transverse expansion of the symphysis, and bone transport for reconstruction of the mandibular body or condyle. The development of mandibular distraction has been largely guided by the capability of the new distractor designs. A large array of devices for different applications is commercially

available. The mandibular distractors can be broadly categorized into two main groups – extraoral and intraoral. Extraoral distractors are attached to the mandible via percutaneous pins and connected to the distractor outside the skin, whereas intraoral distractors are concealed inside the oral cavity. In some designs, the activation rod of the intraoral distractors exits through the skin to allow more convenient activation.

Extraoral mandibular distraction

Extraoral distraction osteogenesis evolved from the mini orthopedic devices used for limb lengthening. The early development of craniofacial distractors started with linear bone movement. With the initial success in clinical applications for mandibular body lengthening, the development shifted to achieve two- or three-dimensional mandibular distraction. The benefits of the new distraction devices are their capability in achieving: (1) angular adjustment to attain bi-directional bone lengthening; and (2) multiplanar bone movement, particularly in lateral facial expansion. Recently, modifications include: the use of titanium metal to reduce the weight of the distractor, which is particularly important for infants; and a composite design of titanium and bioresorbable polymer for the fixation plates and screws.

Extraoral distraction by orthopedic devices

In 1992, McCarthy used the Hoffman Mini Lengthener (Stryker Leibinger, Kalamazoo, MI, USA) to distract the mandibles in four children who presented with craniofacial anomalies.¹⁵ The appliance was initially designed for hand reconstruction, and was attached to the osteotomized bone segments with two pairs of pins. In his series, a maximum lengthening of 24 mm was achieved. With the integration of postsurgical orthodontics, good long-term results were obtained. Using a similar device, Klein and Howaldt were also able to achieve satisfactory results in 1995 in nine patients with mandibular hypoplasia.¹⁸

In 1993, Perrott and co-workers used another lengthening device (Synthes, Paoli, PA, USA) to perform mandibular widening and soft tissue expansion in a patient with hypoglossia–hypodactylia syndrome.¹⁹ This distractor was a mini orthopedic device for leg lengthening, which consisted of two double-pin clamps connected to a linear telescopic distractor. A symmetrical 10 mm mandibular lengthening with expansion of the overlying soft tissue was achieved over 30 days.

The first multiplanar mandibular distraction was reported by Pensler *et al.* in six patients using an Orthofix Mini Fixator (Orthofix, Inc., Richardson, TX, USA) in 1995.²⁰ This device was based on a system designed for reduction and fixation of long bone fractures. The distractor was composed of a sliding double-pin clamp attached to an activation rod and

Table 48.1 Indications for mandibular body and ramus distraction osteogenesis.

Congenital	
Hemifacial/craniofacial microsomia	
Goldenhar syndrome	
Pierre Robin sequence	
Treacher Collins syndrome	
Nager syndrome	
Other congenital micrognathia	
Developmental	
Class II mandibular hypoplasia	
Obstructive sleep apnea	
Acquired	
Trauma	
TMJ ankylosis	

a rotating double-pin clamp connected to the distractor by a ball-and-socket joint, which facilitated multiplanar distraction as required by the patients' deformities. The activation was at a rate of 0.25 mm four times a day. The average bone lengthening was 18.3 mm. All patients showed marked improvement in oral functions including the occlusion, airway, and esthetics.

It is noteworthy that all the devices described above were evolved from the available orthopedic rigid fixation systems. Despite the ability to resist mandibular segment displacement from mastication or muscle tension, high precision is required for positioning of the devices to avoid any undesirable bony movement during activation. The distractors are considered by most maxillofacial surgeons to be bulky and are limited mainly to mandibular body lengthening.

Extraoral distraction by mandibular distractors

Klein and Bitter¹⁸ introduced their own design of extraoral device for mandibular lengthening modified from the Hoffman Mini Lengthener. This Unidirectional Mandibular Distractor (Normed Medizin-Technik GmbH, Tuttlingen, Germany) was composed of a geared device connected to a rotating clamp and a sliding clamp for better adaptation to the mandible. Each clamp holds a pair of transcutaneous osseous pins for fixation (Fig. 48.3a).

In 1996, Molina and Ortiz-Monasterio reported their technique and distractor, modified from McCarthy's experience.²¹ Their technique used an oblique buccal corticotomy on the lateral cortex of the mandible from the gonial angle to the retromolar region. They modified the traditional distractor design to a semi-rigid fixation system. The Molina Unidirectional Distractor (KLS Martin Group, Tuttlingen, Germany) was composed of one intraosseous stainless steel pin inserted on either side of the corticotomy segments (Fig. 48.3b). The pins were connected to the distractor, which was composed of two pin-holding plates joined by an activation screw capable of linear elongation. There are three patterns available for different age groups and maximal distraction lengths for each age group are recommended: infant (28 mm), children (43 mm), and adults (53 mm). A 5-day latency period was recommended before activation of the device at a rate of 1 mm per day. The pins, which were parallel during the insertion, were expected to become divergent as the distractor was activated because the activation rod was made of softer stainless steel that would be bent in response to pressure. According to Molina, the curvature of the distraction rod would enhance the remodeling of the underlying bone, resulting in a curvilinear elongation of the mandible at the angle region. The progressive divergence of distractor produces more elongation of the structures located laterally rather than medially, and hence results in transverse improvement of the facial con-

tour, which is of particular benefit for correction of hemifacial microsomia.

Molina and Ortiz-Monasterio also introduced the use of their Bi-directional Distractor (KLS Martin Group, Tuttlingen, Germany) in patients with deformities involving both the mandibular body and ramus (Fig. 48.3c). Two lateral corticotomies were made, one in front of and one at the gonial angle to allow distraction in the horizontal and vertical planes respectively. The distractor was connected by three transosseous pins. The central pin was fixed to the mandibular segment in between the two corticotomies to act as a fixed pivot for independent vertical and horizontal distraction. The other two pins were fixed to the body and ramus respectively for attachment to the activation rods. There are two sizes available, allowing a maximum of either 56 mm or 76 mm lengthening. From their case series of 106 patients receiving either uni-directional or bi-directional distraction, good esthetic and functional results were achieved with no report of skeletal relapse.²¹

Further modifications of the bi-directional devices provided angular adjustment between the body and ramus distraction vectors during activation. One example is the Bi-directional Mandibular Distractor (Normed Medizin-Technik GmbH, Tuttlingen, Germany), which is composed of a middle joint piece, from which two geared distraction rods extend in different directions (Fig. 48.3d). This design enables independent control of the vertical and horizontal components of distraction. In addition, a more controllable position of the mandibular angle is achievable. Klein and Howaldt reported their experience with this device in 18 patients with various degrees of mandibular hypoplasia.²² The amount of bone lengthening ranged from 7–45 mm horizontally and 7–50 mm vertically.

In complex craniofacial deformities, such as Purzanky type IIb or III hemifacial microsomia, the mandibular deformities can involve horizontal, vertical, and transverse planes. A three-dimensional (3D) surgical correction is therefore necessary to restore the facial symmetry. To achieve an independent lengthening of the mandibular body and ramus combined with gradual angular correction in the situation described above, a multiplanar distraction device is essential to achieve 3D correction. The Multi-directional Mandibular Distractor (Normed Medizin-Technik GmbH, Tuttlingen, Germany) is a pioneering European-designed model modified from the earlier bi-directional version (Fig. 48.3e). This 3D device is composed of two geared distraction rods with one free movable rider. Both rods are connected in the middle by a special joint with attachments to two additional intraosseous pins, which allow independent callus lengthening. The central piece has two universal joint mechanisms allowing each rod to have independent 3D vector control.

The Multi-Guide Mandibular Distraction Device (Stryker Leibinger, Kalamazoo, MI, USA) was an

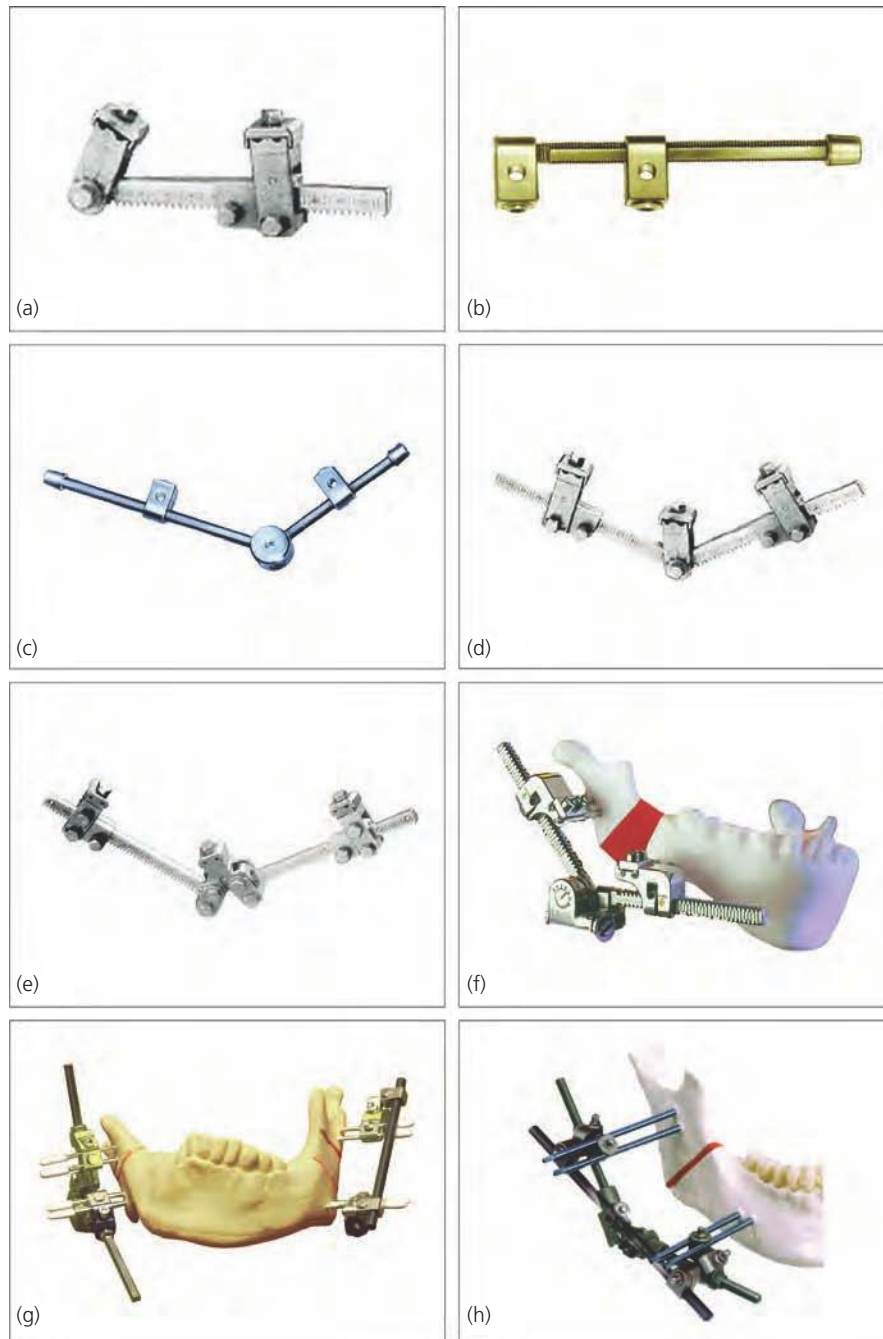


Fig. 48.3 Examples of extraoral mandibular distractors. (a) Unidirectional Mandibular Distractor (Normed). (b) Molina Uni-directional Distractor (KLS Martin); (c) Molina Bi-directional Distractor (KLS Martin). (d) Bi-directional Mandibular Distractor (Normed). (e) Multi-directional Mandibular Distractor (Normed). (f) Multi-Guide II Mandibular Distraction Device (Stryker Leibinger). (g) External Multi Vector Distractor (Synthes). (h) 3D Xternal Distraction System (KLS Martin).

American-designed multi-planar distraction device introduced by McCarthy in 1998. This device was made of stainless steel and consisted of two activation rods with sliding clamps connected in the middle by a universal hinge. Each activation rod could achieve linear bone lengthening of 15 mm. There were two additional activation screws for independent centers of rotation of the distracted callus in vertical and transverse plane of up to 90°. As a result, bone regeneration could be manipulated three-dimensionally during the activation process.^{23,24} In order to improve the dimensions of activation, the Multi-Guide II Mandibular Distraction Device

(Stryker Leibinger, Kalamazoo, MI, USA) was marketed in 2000 by extending the activation rods to 20 mm and 35 mm (Fig. 48.3f).

Despite the superior ability in callus adjustment during activation, it is obvious that all the multiplanar distractors share the same limitation as the uni- and bi-directional distractors: a significant increase in weight and size, which may cause symptoms such as discomfort or neck pain during activation and consolidation periods. As a result, all the recent models have switched to a lighter-weight model made of titanium and interchangeable parts for customization.

The Multi Vector Mandible Distractor (Synthes Maxillofacial, Paoli, PA, USA) is a titanium self-assembly of two distraction arms attached to a 3D hinge for multiplanar movement (Fig. 48.3g). Fixation is achieved by four transosseous pins connected to the distractor arms by a pair of holding clamps. The distractor arms are available in different lengths, ranging from 15–85 mm. Angular adjustment could be achieved up to 90° in either direction, and transverse adjustment is feasible up to 32° towards and 16° away from a patient. Through different combinations of assemblies, the distractor can be customized to any individual's need, and it can be transformed from a single vector device for linear bone lengthening, to a multivector device of achieving 3D manipulation of the distracted callus. In combination with a reconstruction plate, this device could be transformed for the purpose of bone transport in reconstruction of segmental mandibular defects. Lastly, a consolidation rod made of ultra light-weight carbon fiber is available for replacement of the distractor. Altogether these modifications enable more precise control of the three vectors and enhancement of patient comfort.

The 3D Xternal Distraction System (KLS Martin Group, Tuttlingen, Germany) is a newly introduced product to provide gradual multidirectional lengthening and stabilization of bone segments (Fig. 48.3h). This light-weight titanium, low-profile device is an assembly of interchangeable distraction rods, allowing a maximum of 85 mm lengthening, and a 3D hinge for vertical and transverse vector adjustment. It allows vertical angular adjustment from -6° to +98°, and transverse angular adjustment of 20° in either direction. The device can be customized to perform bone transport, and a carbon fiber rod is also available to replace the bulky distractor body.

Intraoral mandibular distraction

The pioneer models for intraoral mandibular distraction were based on modifications of the available orthodontic expansion appliances, and miniaturization of extraoral distractors.²⁵ Based on the principles of the palatal expander developed by Bell and Epker,²⁶ Guerrero published a report of the first intraoral tooth-borne appliance for distraction of the mandibular symphysis in 1991.^{27,28} The device is a custom-made orthodontic Hyrax appliance for transverse widening. In 1994, McCarthy and co-workers developed a bone-borne device for intraoral distraction of the mandibular body.²⁹ The Uniguide Mandibular Distraction Device (Stryker Leibinger, Kalamazoo, MI, USA) is indeed a miniaturized version of their external model, which consisted of two clamps attached to the bone via pairs of pins connected by a telescopic distraction rod. At about the same time, Wangerin and Gropp developed an Intraoral Titanium Mandibular Distraction Device (Medicon eG, Tuttlingen, Germany), which was the first distractor

using miniplates rather than intraosseous pins for fixation.

The introduction of intraoral devices greatly improved patients' acceptance of mandibular distraction. Despite the ability to hide the devices inside tissues and the absence of facial scars, there are limitations to the development of the intraoral distractors due to miniaturization of the devices and restricted mouth opening in some patients. Further development of intraoral distractors has evolved along three main routes: (1) designs based on the anatomical location; (2) designs for universal adaptation on any part of the mandible; and (3) custom-made distractors.

Anatomically, a mandible is composed of two halves that are fused at the midline forming a symphyseal curve. Each mandibular half is made up of a horizontal body and a vertical ramus, forming the gonial angle. Articulation to the cranium is by the condyle located on the posterior upper end of the ramus. Mandibular intraoral distractors have been designed based on these anatomical characteristics and are further elaborated as described below.

Mandibular ramus distraction

In mandibular ramus distraction, the vector of bone lengthening is usually vertical. It is commonly indicated in patients with short mandibular ramus such as hemifacial microsomia. Owing to the limited access, the activators will be either exposed intraorally using a double hinge joint, or transcutaneously at the submandibular or the retromandibular region.

Wangerin and Gropp developed a Vertical Mandibular Distraction Device (Medicon eG, Tuttlingen, Germany)³⁰ for intraoral lengthening of the ascending ramus in 1996. This device was made of titanium and consisted of a rectangular distraction cylinder with two T-shaped miniplates. To facilitate the activation, a gear was added to the distraction cylinder, which was then connected by a universal joint to the activation pin perpendicular to the distractor body. There were two different sizes of this distractor body available to enable a maximum vertical elongation of 15 mm and 20 mm, respectively.

Diner and Vazquez introduced an intraoral distraction system (Stryker Leibinger, Kalamazoo, MI, USA) for uni-directional ramus lengthening in 1997.³¹ This device was a self-assembly of different combinations of distractor frames and rods. The distractor frame included a rectangular framework, and two fixation clamps were secured to the bone with transosseous pins (Fig. 48.4a). The frames were available in two sizes allowing 18 mm and 28 mm bone lengthening, respectively. The clamps were connected to each other with two sliding rods for stabilization of rotational forces. The distraction rod consisted of a threaded portion and a smooth portion connected to each other by a double hinge. The rods were available in different lengths ranging from 85–125 mm to meet

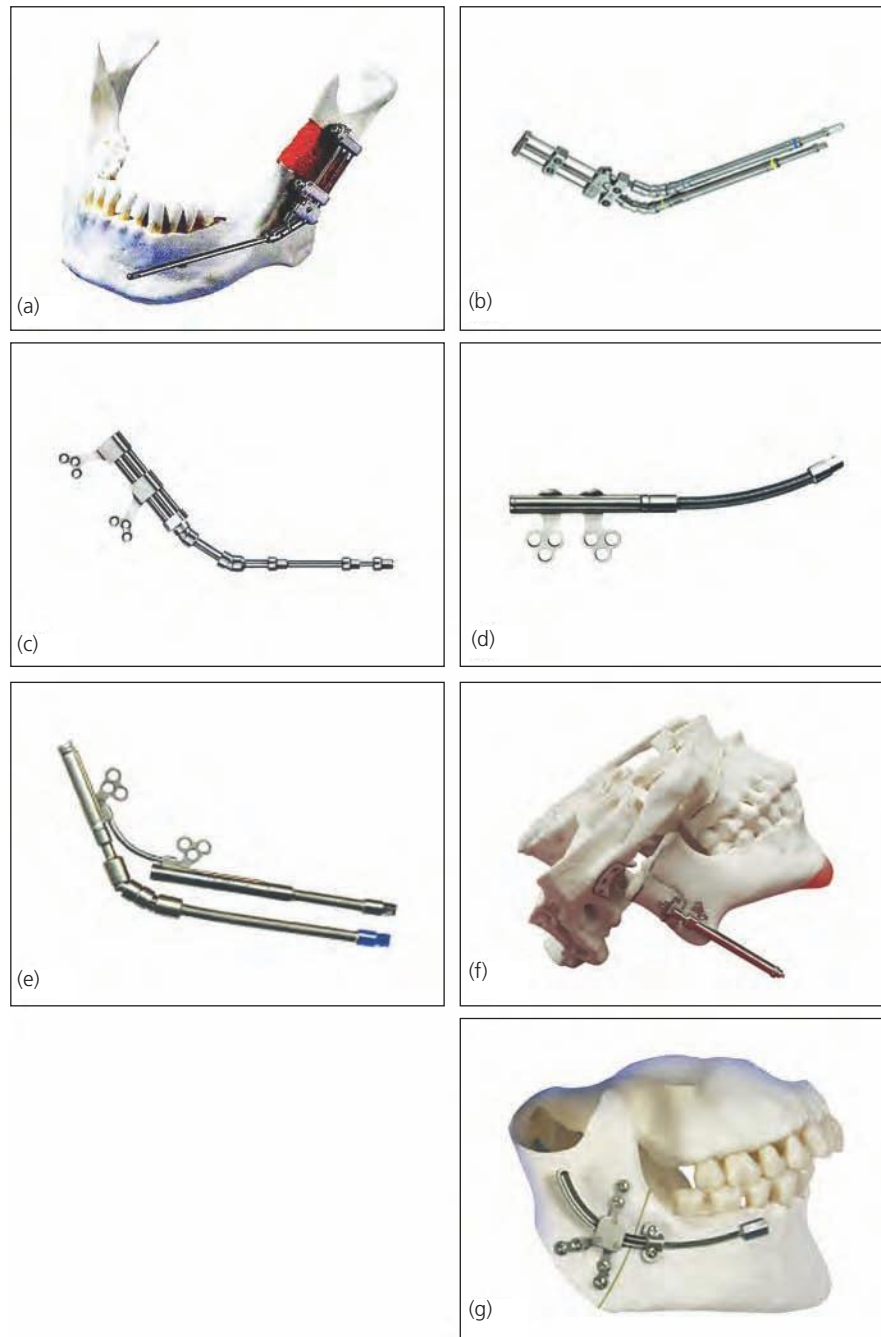


Fig. 48.4 Examples of intraoral mandibular ramus distractors. (a) Uni-directional Distraction System (Stryker Leibinger). (b) Bi-directional Distraction System (Stryker Leibinger). (c) Intraoral Mandibular Ramus Distractor (KLS Martin). (d) Zurich Pediatric Ramus Distractor (KLS Martin). (e) Wood Zurich Intraoral Distractor (KLS Martin). (f) Mandibular Distractor (Synthes). (g) Logic Mandibular Distractor (Osteomed).

the requirements of different age groups and sizes of the oral cavity while maintaining the rod exposed above the mucosa, but inside the oral cavity. From their report on nine young patients, the amount of ramus elongation achieved ranged from 12–28 mm. In a latter modification, a bi-directional device was introduced (Fig. 48.4b). This model was composed of a distractor frame fitting to two detachable rods in order to perform angulation adjustment of 50° and independent bone lengthening of both the mandibular ramus and body basing on a single osteotomy at the gonial angle. The maximum bone lengthening, 20 mm, was considered insufficient for most mandib-

ular ramus lengthening. Although the two rods are color coded, patients can find them confusing and mistakes in distraction of the wrong rod can occur.

In 1998, Hoffmeister introduced a low-profile intraoral distractor – the ascending Mandibular Ramus Distractor (KLS Martin, LP, Jacksonville, FL, USA).³² This device is composed of a threaded unidirectional distraction rod (15–25 mm) and two Y-shaped micro-osteosynthesis plates. The activation is achieved by rotating a double hinge activator, which allows better location relative to the mandibular ramus. In addition, the activator rod could be shortened after activation to improve patient comfort (Fig. 48.4c).

At about the same time, Sailer introduced the Zurich Pediatric Ramus Distractor (KLS Martin, LP, Jacksonville, FL, USA) for uni-directional lengthening of the mandibular body and ascending ramus. This is a miniaturized device composed of a cylindrical body enclosing a threaded distraction rod with two osteosynthesis plates attached. The plates are relatively shorter than in Hoffmeister's design, and are available in the form of cloverleaf or Y-shaped. Owing to the shorter length of connection between the plate and body, fixation of the plate can be located on the anterior ramus or turned around the fixation plate at the retromolar region. There are three maximal distraction lengths available: 15 mm, 20 mm, and 25 mm. Activation is via a semi-flexible rod that can be bent for a curvilinear placement inside the labial sulcus without causing ulceration of the labial mucosa (Fig. 48.4d).

The Wood Zurich Intraoral Distractor (KLS Martin, LP, Jacksonville, FL, USA) is a modified design introduced by the same manufacturer for bi-directional distraction. This distractor is composed of two cylinders containing a threaded distraction rod (Fig. 48.4e). One contourable mesh can be attached to each cylinder, and they are joined by a fixed curvilinear rod. There are two activators for lengthening the mandibular body and ramus up to 20 mm.

The Synthes Mandible Distractor (Synthes Maxillofacial, Paoli, PA, USA) is a single vector distractor designed mainly for ramus elongation (Fig. 48.4f). It is composed of two miniplates attached to a telescopic distraction rod. The stainless steel distraction body is enclosed inside a sleeve to minimize possible soft tissue entrapment. The distractor comes in only one length allowing up to 30 mm of distraction. There are left and right models available based on the foot plate configuration to facilitate anterior or posterior positioning of the screws. Owing to the considerable length and the lack of any hinge or flexible component, the distractor placement is usually semiburied with the activator exposed externally at the submandibular region. As well as vertical ramus elongation for correction of a short ramus, this distractor has been successfully used for transport distraction in condylar reconstruction.³³ In the second-generation device, the distractor has been modified to be a titanium ramus device with a detachable upper titanium plate to enhance distractor removal with the possibility of leaving the plate behind fixed to the mandibular ramus.

The Logic Mandibular Distractor (Osteomed, Dallas, Texas, USA) was developed based on the principle of the logarithmic properties of mandibular growth (Fig. 48.4g).^{34,35} In this low-profile device, the distraction rod is curved to allow a fixed curvilinear trajectory that mimics the natural growth curve of the mandible. There are three types of device available in this distraction system, which allows mandibular lengthening from 24–52 mm. Fixation is by self-tapping screws and activation is via a flexible wire that

provides access through the oral mucosa for ease of activation. During consolidation, the activation wire can be cut to facilitate submersion of the distractor which reduces patient inconvenience.

In 2004, Schendel illustrated the application of a Logic distractor in a case of hemifacial microsomia.³⁶ In this case, the ramus osteotomies were achieved by bilateral sagittal splits, which permitted immediate bone lengthening and correction of any bone misalignment if a counterclockwise rotation of mandible were indicated to correct open bite. The use of a curved distractor enables a logarithmic path of activation, which can create a more normal mandibular plane angle and movement as well as minimizing possible development of anterior open bite after distraction. In addition, detachment of the activation rod allows the body to be submerged to enhance patient comfort. The Logic JR Pediatric Distraction System has subsequently been introduced for use in children and infants in whom poor compliance would be expected during intraoral activation. This distractor design is similar to the adult version except activation is through a detachable flexible cable exposed at the postramal region.

Mandibular body distraction

Mandibular body distraction is usually indicated for advancement of a retruded mandible. Generally speaking, the design of any mandibular body distractor is similar to the corresponding ramus distractor, and activation is usually via an intraoral route. In infants and young children, surgeons prefer to place the activation rod exiting at the upper cervical region for active distraction due to lack of cooperation and the high mucosal infection rate around the activation rod.

Following the successful application in mandibular widening, Guerrero and Bell designed the DynaForm Intraoral Distractor (Stryker Leibinger, Kalamazoo, MI, USA), which is a universally applicable device for mandibular distraction (Fig. 48.5a). The device consists of a body with a distraction screw and four flexible arms. The distractor body is available in different lengths ranging from 7 mm up to 30 mm. Fixation is achieved by bone-borne, tooth-borne, or hybrid using either fork-end or screw-end terminals, which accept 2 mm screws or 0.024 inch stainless steel wires attaching to either the mandibular bone segment or teeth. The great flexibility of the distractor arms allows good intraoperative control of the distractor. This also minimizes the risk of inferior alveolar nerve damage from screw fixation. Using the fork-end terminals, distractor removal can be simplified just by cutting the arms and pulling the forked ends of the device out, leaving the fixation screws in the bone.

Apart from mandibular body lengthening and expansion, this distractor has also been used for intraoral bone transport combined with a reconstruction

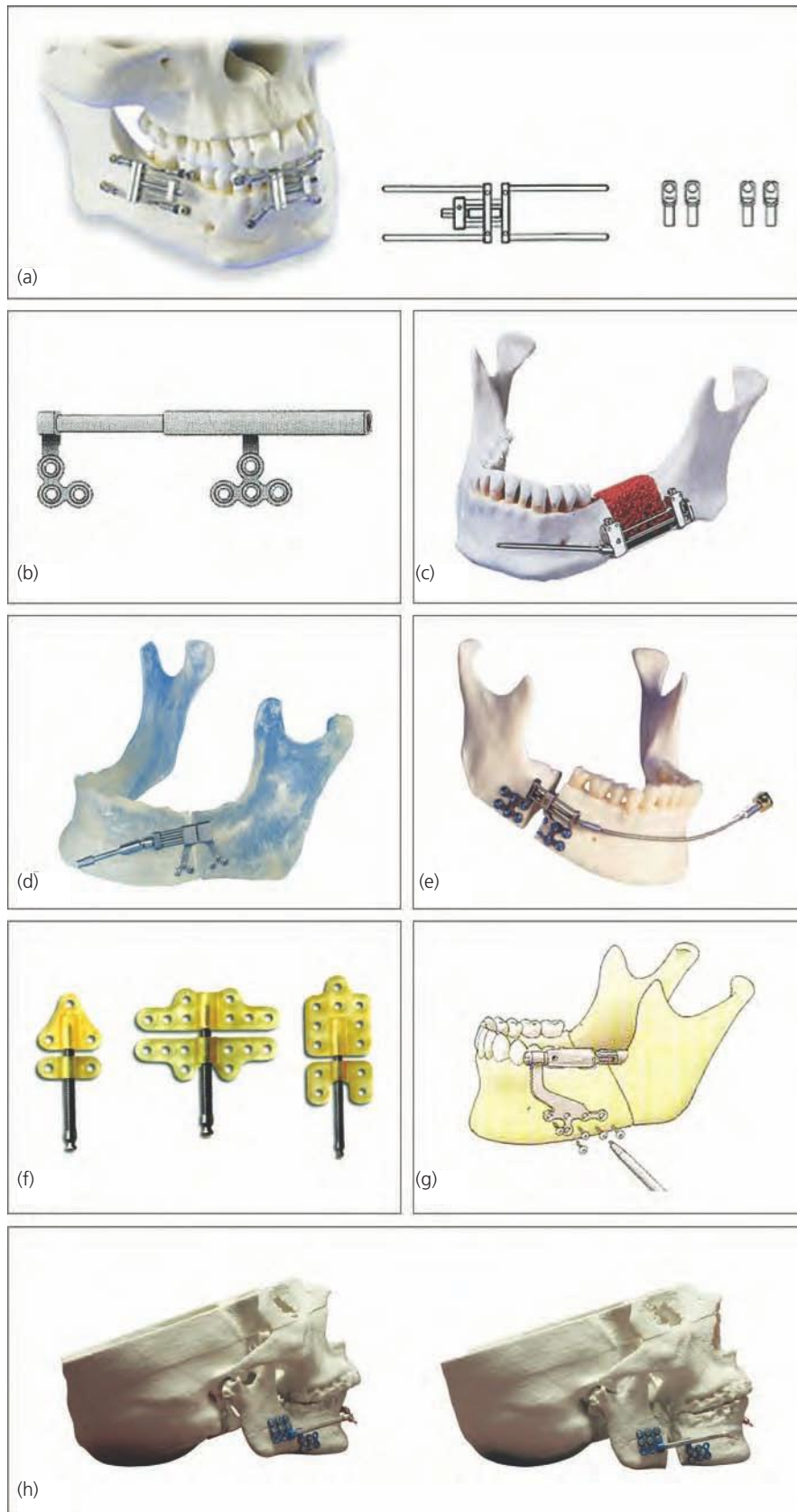


Fig. 48.5 Examples of intraoral mandibular body distractors. (a) Dynaform Intraoral Distractor (Stryker Leibinger). (b) Horizontal Mandibular Distractor (Medicon eG). (c) Intraoral Uni-directional Distractor (Stryker Leibinger). (d) Horizontal Distractor (KLS Martin). (e) Mandibular Distractor (W. Lorenz). (f) Resorbable Mandibular Distractor (W. Lorenz). (g) Mandibular Distraction – Dynamic Osteosynthesis System (Surgi-Tec). (h) CMF Distractor (Synthes).

plate in mandibular reconstruction.^{37,38} Despite the linear nature of regenerated bone from transport distraction, Guerrero reported the use of a two-phase transport disc distraction for reconstruction of the anterior mandible simulating the regeneration of a natural curvature.³⁹

In 1995, Cohen introduced a system of miniature distractors that can be universally applicable for the craniofacial skeleton, including the mandible. The Modular Internal Distraction System (Stryker Leibinger, Kalamazoo, MI, USA) is an assembly of two mini Wurzburg 3-D Mesh plates and a universal distractor body. The distractor body is available in two sizes allowing a maximum lengthening of 15 mm and 30 mm, respectively. The body is attached to a flexible activation cable, which can be bent to minimize postoperative irritation of the surrounding mucosa from intraoral activation. The cable can alternatively be exposed at less conspicuous sites, such as the sub- or postmandibular regions for extraoral activation. Owing to the highly contourable 3D mesh plates and the flexible activator, this distraction system can be easily adapted anywhere on the craniofacial complex as well.

In 1998, Razdolsky developed a series of tooth-borne and hybrid ROD distraction devices (Oral Osteodistraction, LP, Buffalo Grove, IL, USA), of which the anterior activated distraction mechanism can be attached to stainless steel crowns or miniplates by removable attachments.⁴⁰ In addition, he designed a special laboratory instrument allowing individually programmed fabrication of the distraction body along a predetermined axis of distraction based on individual occlusal records.

Following fixation of the stainless steel crown and/or plate, the distractor body is temporarily removed to enable the corticotomy cut to be made. Three types of distraction system are available for application in the mandible. The ROD-1 distractor is a completely tooth-borne appliance designed for interdental distraction in class I or class II malocclusion with crowding of the anterior teeth. The ROD-2 distractor is a hybrid device indicated for class II malocclusion with severe mandibular hypoplasia and minimal crowding of the anterior teeth. Fixation of the ROD-2 device is by miniplate and screws attached to the ascending ramus and through stainless steel crowns to the mandibular body. The ROD-3 distractor is a tooth-borne appliance designed for mandibular widening. In 1999, Dessner *et al.* reported the successful application of this distraction system for five patients with retrognathic mandible.⁴¹

Apart from the vertical distractor, Wangerin and Groppe also developed the Horizontal Mandibular Distractor (Medicon eG, Tuttlingen, Germany) for lengthening the mandibular body. This distractor is a modification of their original design (Fig. 48.5b), which is composed of a square-shaped cylinder (20–25 mm) with two miniplates (L- and T-shaped) on either end. From their 3-year report on 23 patients

undergoing both vertical and horizontal distraction, the amount of bone lengthening achieved was in the range of 7.5–25 mm.⁴²

Around the same time, Diner and Vazquez introduced another design of intraoral distractor (Stryker Leibinger, Kalamazoo, MI, USA) for uni-directional mandibular body lengthening. The design is almost identical to its ramus version, and is composed of an assembly of a distractor body and a distractor rod (Fig. 48.5c). There are two distractor bodies available for either 18 or 28 mm body lengthening. The difference to the ramus version is on the connection design of the activator, which is a single hinge joint. In a latter modification, it allows: (1) fixation by means of screws or titanium miniplate to adapt to the individual's anatomical variation; and (2) the distractor rod can be detached following activation to improve patient comfort. However, the activation length is relatively shortened (maximum: 23 mm). The bi-directional device was introduced a few years ago to increase the flexibility of the callus adjustment during activation, and hence further reduce the development of anterior open bite.

The Horizontal Distractor (KLS Martin, LP, Jacksonville, FL, USA) is a low-profile device designed by Hoffmeister. The device is generally similar to the ascending mandibular ramus distractor, and is composed of a threaded unidirectional rod and two Y-shaped micro-osteosynthesis plates (Fig. 48.5d). The difference from the ramus device is that the activator is a smooth rod of shorter length with a single hinge joining the threaded rod. The distractor is available in different length ranging from 10–20 mm. During fixation, the plates are adapted and fixed near the lower border of mandible. It is not uncommon for the distractor body to be exposed intraorally together with the activation rod.

Despite its name proposing indications for children and the mandibular ramus, the Zurich Pediatric Ramus Distractor (KLS Martin, LP, Jacksonville, FL, USA) can be applied for mandibular body lengthening in adults as well. Owing to the short connector length, the plate could be fixed to the upper part of the mandibular body instead of the lower, facilitating total submersion of the distractor body into the soft tissues. Despite the flexible activation rod, the rigid smooth rod connected to the distractor through a single hinge axis has also been introduced in latter modifications. Three designs are available to enable maximal bone lengthening ranging from 15–25 mm.

The Mandibular Distractor developed by Lorenz (W. Lorenz Surgical, Jacksonville, FL, USA), was designed solely for linear mandibular body lengthening. The device includes four miniplates connected to the distractor body of which one or two plates can be cut. The body is then attached to a flexible rod for activation (Fig. 48.5e). The distractor is available in different lengths from 5 mm up to 20 mm. It includes a special activation screw driver with an audible

“click” sound for clear determination of the amount of advancement. During the consolidation period, the flexible shaft can be detached to improve patient comfort. In addition, a resorbable distraction system was introduced. This design is composed of a metallic distraction screw joined with two resorbable plates (Fig. 48.5f). It is indicated in pediatric patients (less than 2 years old) with mandibular hypoplasia. There is only one surgical procedure and the removal of the distraction screw is a simple in-office procedure. The plates usually undergo spontaneous resorption within 1 year.

In 1998, Mommaerts introduced a horizontal distractor using the ascending ramus as anchorage.⁴³ His design was based on the concept that the ascending ramus provides: (1) a volume of bone that can receive large horizontal screws in the retromolar area, between the internal and external oblique lines; and (2) an area which is not prone to disuse resorption. The Mandibular Distraction – Dynamic Osteosynthesis System (Surgi-Tec NV, Brugge, Belgium) is composed of a single horizontal posterior fixation screw implant, a vertical hinge that copes with the lateral force vector in the condyles, a telescopic distraction module, and an anterior fixation unit that is fixed with monocortical screws (Fig. 48.5g). There are two distraction modules available to enable a maximum of 17 mm and 21 mm linear bone lengthening. From his initial clinical experience of 35 cases, Mommaerts cautioned that the placement of the distractor module and the posterior fixation screws should be close to the dental arch and away from any lateral occlusal interference and lower labial sulcus. In addition, the fixation screws should be long enough to avoid any slippage of the posterior fixation unit. From radiographic investigation using cephalometric radiographs, Hendrickx *et al.* noted the proximal segment was anteriorly rotated by about 3.3°, while the distal segment was posteriorly rotated after the lengthening procedure.⁴⁴ As a result, the angle of mandible was pushed back half the distance of the advancement of the anterior mandible.

In view of the broad age group indicated for distraction, it is difficult to define a particular size that fits all and this is the basis of the new modular design of mandibular distraction. Synthes and Medartis produced modular mandibular distractors at a similar time to enable surgeons to choose the most suitable components for individual cases (Fig. 48.5h). There is also improvement of the flexible activating rod design and it is easier to maintain hygiene around the mucosal exit. The latest improvement in rod design is the detachable rod design that enables the distractor to be buried inside the mucosal bed without the commonly infected distractor rod.

Significant esthetic and occlusal improvement can be achieved with mandibular distraction as illustrated in a clinical case of severe class II division II malocclusion receiving distraction of the posterior mandibular body (Fig. 48.6).

Mandibular symphyseal distraction

Mandibular symphyseal distraction is indicated for widening of the mandible, in which the amount of expansion exceeds that which can be achieved with conventional orthodontic or surgical treatment. Mandibular symphyseal distraction is also indicated in several orthodontic scenarios, such as severe anterior mandibular crowding, unilateral scissor bite, or impacted anterior teeth. However, the site for housing the distractor is limited and the amount of bone gained by widening is small. Fixation of the device could be tooth-borne or bone-borne.

The first generation of intraoral distractors was modified from the surgical-assisted rapid palatal expander.^{27,28} Thereafter, application of the DynaForm Distractor (Stryker Leibinger, Kalamazoo, MI, USA) in mandibular widening was reported.⁴⁵ This distraction system includes two compact distractor bodies capable of 7 mm or 12 mm maximal lengthening, which can be housed within the symphyseal region (Fig. 48.5a). At least two of the four flexible arms can be cut and bent to the proper length and shape according to the specific anatomic structure of the mandible. Fixation of the device can be either bone-borne or hybrid. At the time of removal, no incision is required. The arm can be simply pulled and the fork-ended terminal will release from its stabilization screw, which can be left behind without any need to open the wound. This has effectively converted the distraction process into a one-stage surgical procedure rather than the traditional two-stage surgery.⁴⁶

Tomcat and co-workers introduced two types of paramedial mandibular distractors – the Symphysis Distractor (Fig. 48.7a) and the Berger Distractor (Fig. 48.7b) (KLS Martin, LP, Jacksonville, FL, USA) – for correction of transverse mandibular deficiency associated with crowding of the anterior teeth in patients with class I or II malocclusion. The distractor is composed of a cylindrical body and a longitudinal gliding hole. Fixation is by miniplate and screws. The differences between the two distractors are the plate configuration and the length of distraction (15 mm for the Symphyseal Distractor and 10 mm for the Berger Distractor). The activation site is at the front to improve patient comfort. To ensure a simultaneous activation on both the alveolar and basal bone, this distractor has two occlusal splints incorporated to cover the buccal segments from the canine to the molar. The buccal part of the splint is attached to the upper miniplate of the distractor whereas the lower miniplate is fixed to the basal bone. The overall design is aimed to provide equal force to both the teeth and the basal bone and to avoid any occlusal interference and any TMJ problems during activation.⁴⁷ On the other hand, it is worth noting that the presence of this device may obliterate the space and interfere with re-attachment of the mentalis muscles.

In 2001, Mommaerts reported the use of a bone-borne Transmandibular Distractor (Surgi-Tec NV,

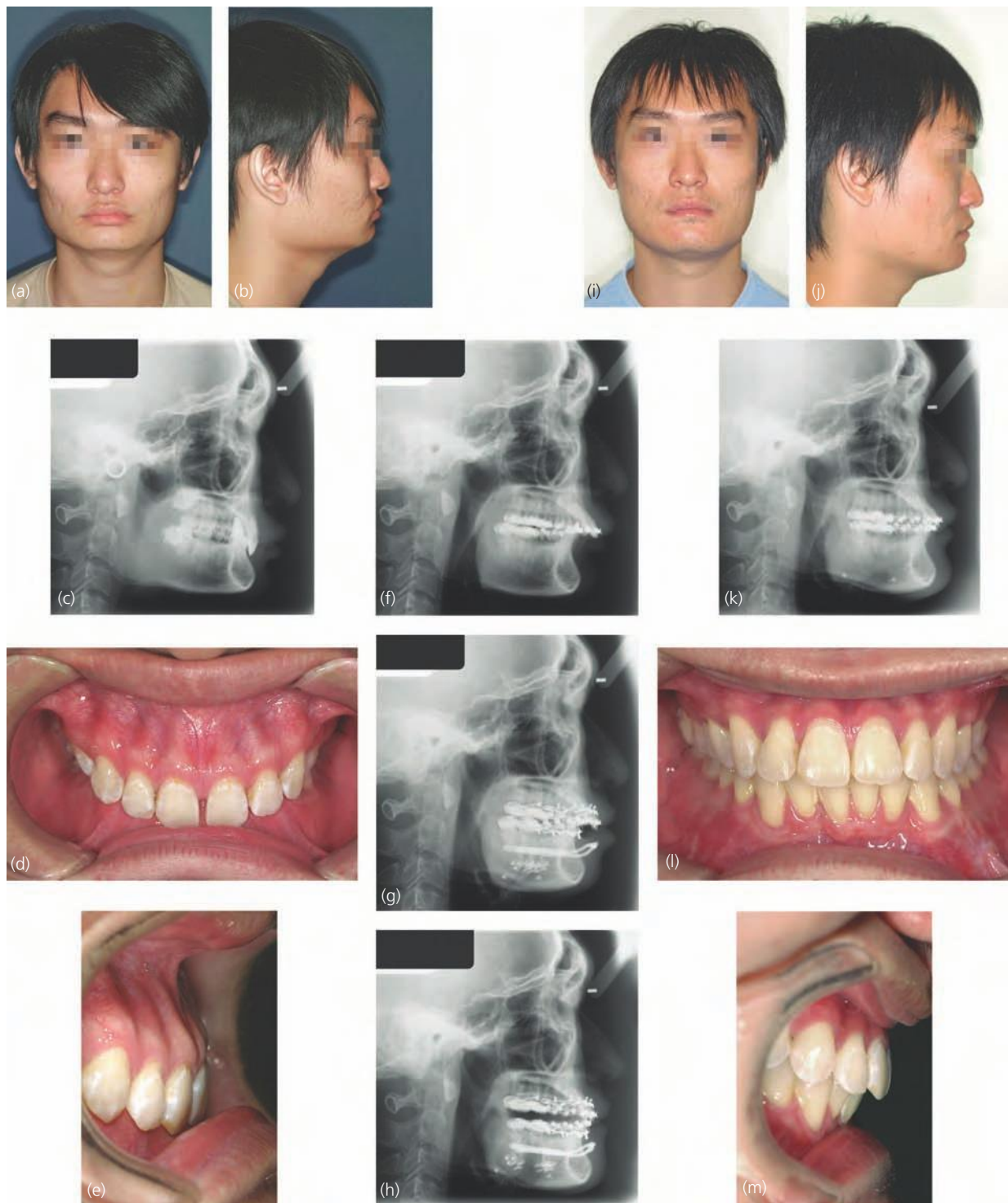


Fig. 48.6 A clinical case of mandibular body distraction. (a) Preoperative frontal view shows reduction in the lower facial third. (b) Preoperative lateral view shows hypoplasia of the mandible and chin. (c) Preoperative lateral cephalograph shows class II division II malocclusion. (d, e) Oral views show severe deep bite. (f) Lateral cephalographs show maxillary and mandibular teeth are decompensated. (g) Internal distractors fixed on each side of the mandible. (h) Class I occlusion achieved. (i, j) Postoperative frontal and lateral views show a balanced face. (k) Improvement in the facial profile. (l, m) Stable occlusal outcome.

Brugge, Belgium) for anterior mandibular widening.⁴⁸ This device is made entirely of pure titanium. It is composed of two vertical footplates connected by two threaded parallel distraction rods and a pair of off-set extensions, which can be bent to improve the plate adaptation (Fig. 48.7c). The extension allows the distraction rods to be exposed intraorally for activa-

tion. In addition, it allows a limited area for reattachment of the mentalis muscles. Two main distractor types are available in this system, which allow maximum expansion of 12 mm or 20 mm. Several subtypes are also available for better adaptation of the individual's chin contour.

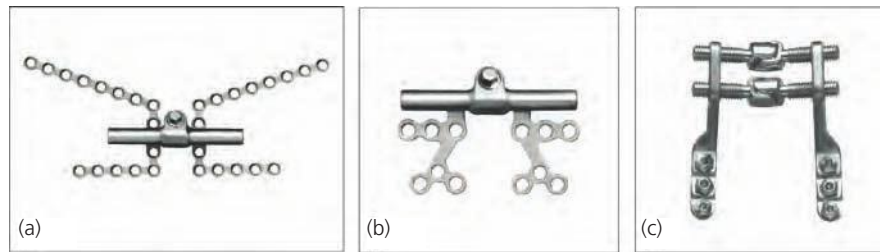


Fig. 48.7 Examples of mandibular symphyseal distractors. (a) Symphyseal Distractor (KLS Martin). (b) Berger Distractor (KLS Martin). (c) Transmandibular Distractor (Surgi-Tec).

Craniofacial distraction osteogenesis

With the advances of mandibular distraction, laboratory studies were performed to investigate the efficacy of distraction osteogenesis in other regions of the craniofacial skeleton. The first experiment on midface was not initiated until 1993, when Rachmiel *et al.* began midfacial distraction on a sheep model.⁴⁹ An external device was able to successfully advance the midface by 36 mm at the nasofrontal area and 43 mm at the lateral aspect of the maxilla. New bone regeneration was confirmed radiographically, clinically, and histologically. In 1995 Staffenberg demonstrated that the midface could be advanced using external distractors without the creation of maxillary osteotomies in growing animals.⁵⁰

Indications for craniofacial distraction osteogenesis

Distraction osteogenesis has been advocated in cases of severe mid-face hypoplasia requiring advancements greater than 10 mm. These have mainly included syndromic cases such as Apert, Crouzon, and Pfeiffer syndromes. Due to the large movement required, conventional surgical treatments of such deformities have been associated with instability and the need for bone

Table 48.2 Indications for craniofacial, midface, and maxillary distraction osteogenesis.

Congenital	
Apert syndrome	
Crouzon syndrome	
Pfeiffer syndrome	
Other craniosynostosis	
Nasomaxillary hypoplasia	
Cleft maxillary hypoplasia	
Developmental	
Maxillary hypoplasia	
Acquired	
Maxillary relapse after orthognathic surgery	
Trauma	

grafting. As such, craniofacial distraction osteogenesis is advantageous as it allows a large magnitude of movement without the need for bone grafting. It also allows the vector of movement to be adjusted in the horizontal and vertical planes. Similarly, distraction osteogenesis has been applied at the Le Fort I level, especially in cases of cleft maxillary hypoplasia or even in cases of severe developmental maxillary hypoplasia. Other applications of distraction osteogenesis at this level include correcting transverse width discrepancies and moderate to severe alveolar ridge deficiencies (alveolar distraction). The common clinical indications for craniofacial, midface, and maxillary distraction osteogenesis are listed in Table 48.2.

External maxillary and midface distraction

In 1996, Molina and Ortiz-Monasterio⁵¹ reported using an external facial mask resembling an orthodontic forward traction headgear following a Le Fort I osteotomy. However, Polley and Figueroa⁵² noted that an orthodontic facial mask with elastics was not sufficient to achieve the desired amount of forward movement. They developed a rigid external fixation system (RED; KLS Martin, Tuttlingen, Germany) (Fig. 48.8a) for maxillary advancement mounted on a halo frame fixed to the temporal portion of the skull. This serves to distract the midface forward or downward after maxillary osteotomies by anchoring to the maxillary dental arch via an occlusal splint. In a 10-year-old child presented with severe maxillary hypoplasia as a result of bilateral cleft lip and palate, Polley and Figueroa were successful in achieving a maxillary advancement of 15 mm with no complications or discomfort. They concluded that the device was simple to use and the scalp pins did not cause any problems. After 1 year, the position of the maxilla and occlusion remained stable. Similar external distractors with different color head frames but with the same mechanics were produced by Lorenz, called a blue system, and Synthes, called a gray system (Fig. 48.8b).

Instead of pulling the midface forward as with the previous mentioned midface distractors, Molina⁵³ designed a unilateral orbito-malar distractor (KLS Martin, Tuttlingen, Germany) (Fig. 48.8c) that pushes the facial skeleton forward and this can be used in conjunction with a Le Fort III osteotomy. The self-

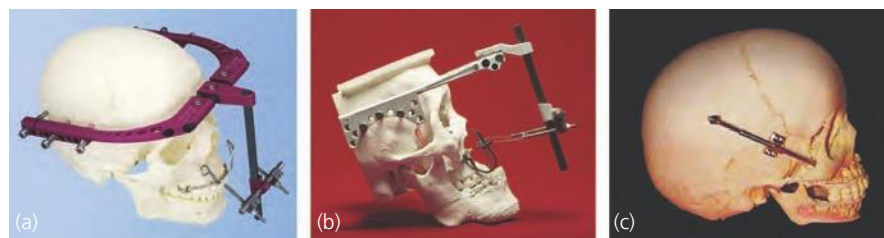


Fig. 48.8 Examples of extraoral midface distractors. (a) Rigid External Distractor (KLS Martin). (b) External mid-face distractor (Synthes). (c) Molina Orbito-malar Distractor (KLS Martin).

contained rod is smooth and facilitates function and comfort. The activation portion of the rod exits percutaneously within the hair-bearing skin behind the ear and distraction up to 25mm is achieved. The anterior point of the device has a pivot point that enables flexibility in placement to the malar bone at the frontozygomatic process.

Internal maxillary and midface distraction

In 1995, Cohen *et al.*⁵⁴ introduced a system of miniature distractors that could be customized for use anywhere in the craniofacial complex. They initially reported on the use of these devices in a 4-month-old infant with unilateral craniofacial microsomia and anophthalmia. Facial moulages of the infant were taken to aid in the design of the device. A modified Le Fort III osteotomy with internal orbital osteotomies and a mandibular osteotomy were performed. The distraction devices were placed to correct the sagittal and vertical maxillary deficiency, expand the orbit and increase the length of the mandibular body. The vectors, chosen independently and the devices custom modified, enabled multiple distraction to proceed simultaneously. Cohen⁵⁴ further refined his miniature distraction devices, now called the Modular Internal Distraction (MID) system (Styker Leibinger, Michigan, USA) (Fig. 48.9a). Two distractor frames are available to provide 15mm or 30mm of advancement. The frames are attached to 1.7mm Wurzburg three-dimensional micromesh plates of varying sizes using 1.6mm screws. A flexible activation cable exits percutaneously either at the pre- or postauricular skin, through the scalp, or intraorally. It is recommended that a complete osteotomy be performed with a latency period of 5–7 days, followed by 1mm per day of distraction and a consolidation period of 8–12 weeks.

Sailer introduced the Zurich Paediatric Maxillary Distractor (KLS Martin, Tuttlingen, Germany) (Fig. 48.9b), which has two plates, one of which was attached to the maxillary tuberosity and posterior alveolus bone, and the other to the zygomatic buttress.⁵⁵ In a group of adolescents and adult patients, Bauman *et al.*⁵⁶ was able to achieve a mean advancement of 6.4mm (range 5–8mm) when measured at the A point. They noted that the intraoral distractors were well tolerated by all patients. Lauwers *et al.*⁵⁷ reported a case of severe maxillary hypoplasia in a

16-year-old cleft patient treated by maxillary advancement. Two intraoral distractors (Zurich Pediatric Maxillary Distractor, KLS Martin, Tuttlingen, Germany) were placed for a high Le Fort I osteotomy. After 12 months of postoperative follow-up, no bony relapse was detected and the occlusal result was stable.

Razdolsky introduced a ROD-4 distractor, which comprises a tooth-borne device attached to the distraction screw by a lock and key design.⁵⁸ This facilitates the insertion of a tooth-borne component preoperatively. A single case report has been published so far and a distraction of only 4mm was achievable in a non-cleft patient.

In 2004, Cheung *et al.*⁵⁹ reported the use of an intraoral bone-borne distractor (Synthes, Pennsylvania, USA) (Fig. 48.9c) in 15 cleft lip and palate patients. They found that the advanced maxilla was stable at 1 year follow-up. This type of maxillary distractor can be customized to meet the anatomical needs of the patients, based on preoperative treatment planning. It has four distractor lengths that allow 10mm, 15mm, 20mm, or 25mm advancement. Three anterior and posterior footplate heights are available to accommodate the pediatric and adult population. The distractors are made from stainless steel. They may be attached either directly to the maxilla or indirectly via a maxillary occlusal splint.

Gateño *et al.*⁶⁰ developed a new Le Fort I internal distraction device and was successful in advancing the maxilla of three cleft lip and palate patients with severe maxillary hypoplasia who needed advancement in excess of 12mm. The internal distraction device consists of an upper and a lower bone plate. The upper plate is shaped like a “U” and is installed above the Le Fort I osteotomy while the lower plate is shaped like an inverted “U” and is installed below the Le Fort I osteotomy. The anterior and posterior limbs of the plates are secured with screws to the pyriform and zygomatic buttress respectively. The distracting screw is oriented parallel to the sagittal plane and placed in the maxillary sinus. An activating rod is attached to the distracting screw with a universal joint and exits the oral cavity through the buccal mucosa.

Chin *et al.*⁶¹ designed an implantable internal titanium distraction device to achieve midface advancement. They custom designed their own internal device because they believed that patients would

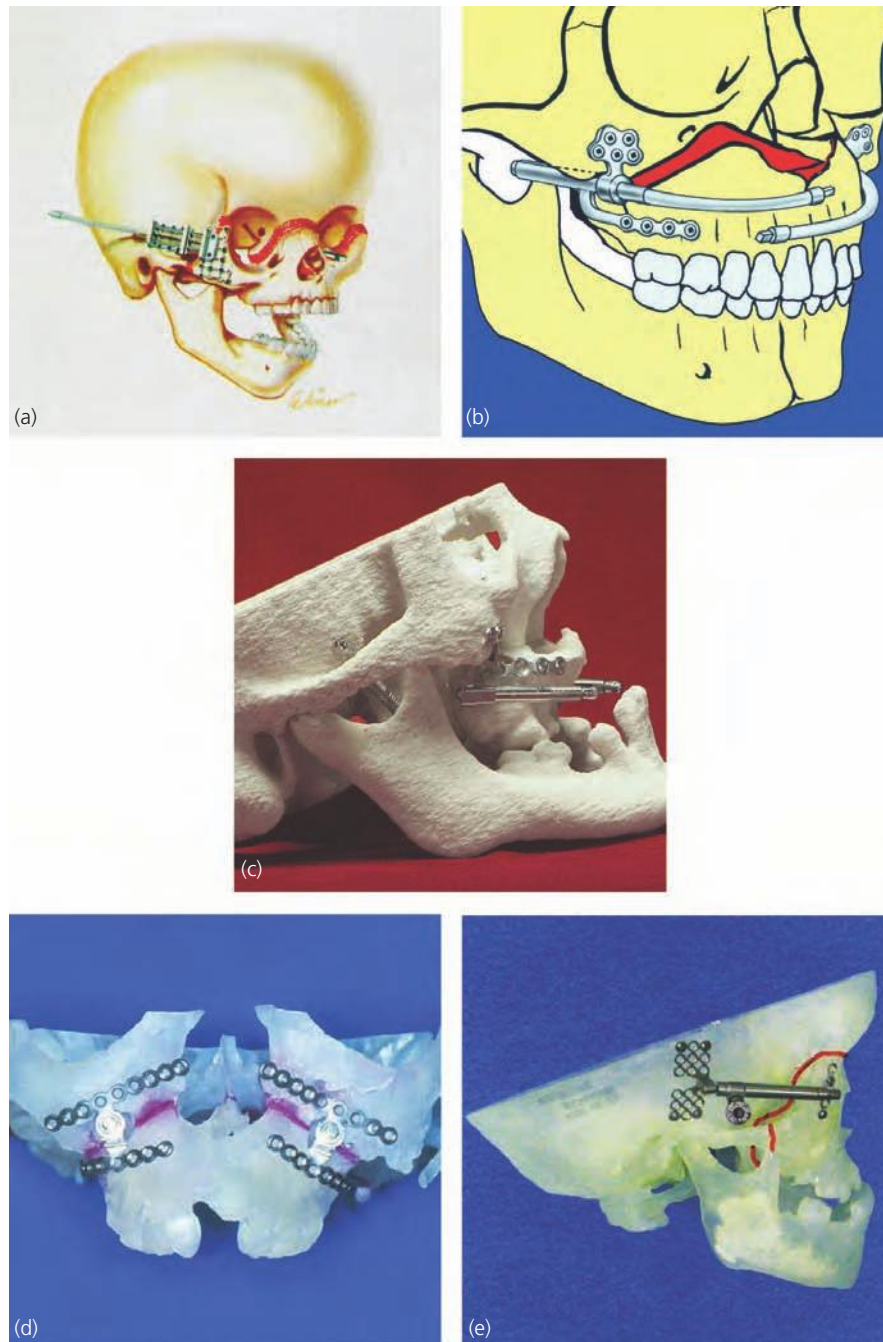


Fig. 48.9 Examples of intraoral midface and maxillary distractors. (a) Modular Internal Distractor (Stryker Leibinger). (b) Zurich Pediatric Maxillary Distractor (KLS Martin). (c) Intraoral Maxillary Distractor (Synthes). (d) Nadjmi Trans-sinusoidal Distractor (KLS Martin). (e) Riediger Midface Distractor (KLS Martin).

have a better acceptance and ease of integration. Models of the skeleton are milled from computed topographic data to plan the surgery and determine the vector of the internal distraction bone-borne device. The force is transferred from the temporal bone posteriorly to the midface through the lateral orbital rim and malar complex. Chin and Toth's surgical approach departed from the principles of Ilizarov by not observing a latency period but began activation immediately before closing the surgical site. They reported that 4–12 mm of distraction in the midface was achieved in their group of patients.⁶¹

An ingenious distractor using the maxillary sinus to hide the distractor has been marketed by Martin as the Nadjmi Trans-sinusoid Distractor (Fig. 48.9d). The distraction screw is fitted into the maxillary sinus. The upper and lower plates are fixed above and under the Le Fort I osteotomy line respectively. The head of the activator is put through the soft tissue just behind the upper lip.

Martin developed the Riediger Midface Distractor made of medical-grade titanium (Fig. 48.9e).⁶² It consists of a distraction cylinder with an internal expansion system and a lateral activation rod. A

bendable plate with four holes is connected at the posterior end. The lateral activation rod is constructed perpendicular to the distraction cylinder and is accessible through a small cutaneous incision in the preauricular region.

A case of maxillary distraction with an internal Synthes device at a Le Fort I level in combination with mandibular orthognathic surgery in a cleft lip and palate patient following by external rhinoplasty is illustrated to show the significant improvement both in esthetics and occlusion (Fig. 48.10).

Zygomatic distraction

Zygomatic deficiency is commonly seen in children with congenital deformities, such as craniofacial microsomia and Treacher Collins syndrome.

Previously, these patients were treated by recreating a zygomatic arch with either vascularized or non-vascularized bone grafts.⁶³ Many of these grafts either resorbed or failed to grow properly, and required serial grafting at a later age. Due to these problems, attempts have been made to augment the zygomatic complex by using the technique of distraction osteogenesis. In 1992 Phillips *et al.* reported the first animal zygomatic distraction using an external distractor on a canine model.⁶⁴ In 1994 Glat *et al.* confirmed that distraction of the zygoma could achieve good quality bone regenerate.⁶⁵ Molina *et al.*⁶⁶ reported the first successful distraction of an entire zygomatic complex using an internal distraction device that exited at the postauricular region. With this technique, they were able to advance the deficient zygoma forward as much as 10 mm.

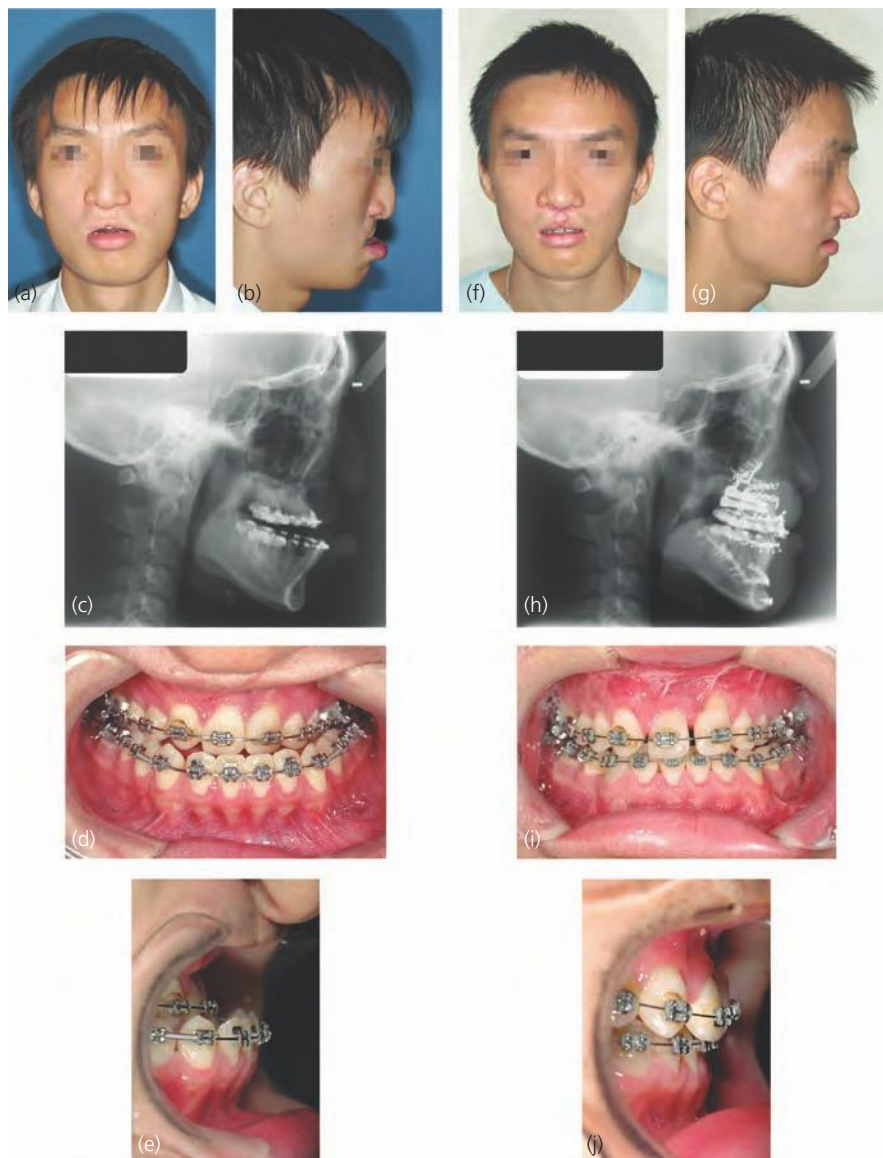


Fig. 48.10 A clinical case of intraoral maxillary distraction on a cleft lip and palate patient. (a) Preoperative frontal view shows incompetent lips and flaring of the lips. (b) Preoperative lateral view shows protrusion of the lower lip and retruded maxilla. (c) Lateral cephalograph confirms hypoplasia of the maxilla and dentoalveolar hyperplasia of the mandible. (d) Class III malocclusion. (e) Teeth are decompensated. (f, g) Postoperative improvement in facial profile. (h) Intraoral maxillary distractors fixed on the maxilla. (i, j) Class I occlusion achieved.

Cranial distraction

Osteodistraction techniques have also been applied to the cranium. Persing *et al.* reported in 1986 on the possibility of coronal suture mobilization with a spring device on a rabbit craniostosis model.⁶⁷ They confirmed that advancement of the skull base across the frontosphenoidal suture was achievable by distraction in two cases.⁶⁸ This is particularly useful in patients suffering from craniostosis by reducing the complication of infection associated with immediate frontal osteotomy advancement.

Remmler *et al.* later applied a circumferential external distraction device to a rabbit craniostosis model.⁶⁹ A circumferential suturectomy was performed and this distractor advanced the craniofacial skeleton. Tschakaloff *et al.* reported that they could achieve coronal suture distraction with a subcutaneous device that was activated internally.⁷⁰ Muhlbauer⁷¹ reported in 1995 a group of patients with Apert syndrome, who underwent distraction osteogenesis of the midface and orbits for the correction of exorbitism and midface hypoplasia. He made use of bilateral screws lodged into the body of the zygoma and fixed posteriorly to the cranium. The device was placed percutaneously in the hair-bearing scalp above the ears. Results were reported to be excellent and were well documented by photographs and 3D CT scans.

Maxillary palatal distraction

Transverse expansion of the maxilla was first described in 1860.⁷² The combined surgical and clinical orthodontic treatment for maxillary expansion by a Hyrax appliance with anchorage on teeth became the standard rapid maxillary expansion method.⁶⁶ In 1999 Mommaerts⁷³ introduced a TransPalatal Distractor (TPD – Surgi-tech NV, Brugge, Belgium), the first bone-borne distractor successful in attaining maxillary transverse expansion by distraction osteogenesis. This device has interchangeable modules, which allow large expansion to be achieved even in narrow palates. Mommaerts emphasized that there is no loss of anchorage since the abutment plates are fixed on the palatal bone. This has later been trans-

formed into a 3-in-1 device to reduce the chance of component disassembly and to facilitate insertion (Fig. 48.11a). Recently, the Rotterdam Palatal Distractor and Magdeburg Palatal Distractor (KLS Martin, Tuttlingen, Germany) were developed (Fig. 48.11b, c). The Rotterdam Palatal Distractor is based on the mechanical principle of a car jack.⁷⁴ On activation, the nails of the abutment plates penetrate the bone and stabilize the device hence no screws are necessary for fixation of the device. This device is available in two sizes. Type A is designed for extremely narrow palates particularly in syndromal patients, whereas type B is for normal palates. Klaus and Zahl⁷⁵ reported use of the Magdeburg distractor. This device has to be fixed with screws to the palatal bone and can be applied submucosally or epimucosally. This device has proven to be useful in patients with acquired deformity. The Smile distractor was also recently developed by Titamed (Fig. 48.9d). This distractor is composed of two abutment plates and a cylindrical module. Conical, self-tapping osteosynthesis screws with hexagonal heads are used to fix the abutment plate. Different cylinder lengths are available to achieve expansion from 8–20 mm.

Alveolar distraction osteogenesis

Alveolar distraction osteogenesis (ADO) has the benefit of simultaneously increasing bone dimension, either in width or vertically, and the volume of surrounding soft tissues, particularly the attached gingiva of the alveolar ridge. Since its first report in 1996, ADO has been well recognized to have consistent evidence of bone augmentation clinically, histologically, and radiographically, thus making it a predictable, safe, and reliable surgical procedure for vertical bone augmentation with low incidence of major complications.^{76–83} The first report of ADO published in 1996 was of ADO applied to treat a vertically deficient alveolar ridge.⁸⁴ Since then it has been extensively used as one of the treatment modalities for bone augmentation in treating alveolar bone deficiency of both the mandible and maxilla prior to implant treatment. ADO is starting to gain popularity in reconstructing bone defects following tumor surgery. Favorable results of bone augmentation have been reported in cases involving vascularized flaps such as from the fibula.⁸⁵

Indications for alveolar distraction osteogenesis

The recommended clinical indications for ADO include:⁸⁶

- moderate to severe atrophy of edentulous ridge;
- segmental deficiency of the alveolar ridge that may compromise the esthetic and functional aspects of implant placement;

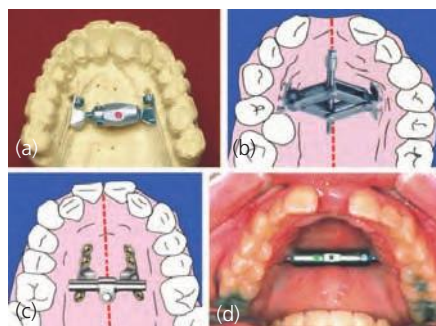


Fig. 48.11 Examples of palatal distractors. (a) TransPalatal Distractor (Surgi-Tec). (b) Rotterdam Palatal Distractor (KLS Martin). (c) Magdeburg Palatal Distractor (KLS Martin). (d) Smile Distractor (Titamed).

- narrow alveolar ridge;
- ankylosed submerged teeth such that orthodontic movement is not feasible;
- osseointegrated implant that is at a higher gingival line than adjacent teeth.

Besides the indications proposed above, Garcia *et al.*⁸⁷ recommended vertical ADO whenever the ratio of required crown height to bone height available for implantation is greater than one.

The advantages of ADO in comparison to conventional surgical bone augmentation techniques are that ADO is minimally invasive, there is a shorter period for rehabilitation with implant, the bone graft procedure is eliminated thus avoiding donor site morbidity, augmentation is more physiologic with natural bone regeneration simultaneous with soft tissue expansion, and there is a lower rate of complications.^{76,77,80–82,88–93}

However, ADO also has several disadvantages, such as the long duration of treatment, involving two surgical procedures, it is considered to be technically complex, and it is also difficult to perform in a limited alveolar space.^{77,88,92,94,95}

Vertical alveolar distraction

The most common vertical distractor used is TRACK design (KLS Martin Group, Tuttlingen, Germany) with two titanium miniplates connected by a central activation rod, either 10 or 15 mm in length. The problem of lack of stability, particularly from the lingual pull by the tongue muscle and palatal pull by the tight palatal mucosa, has prompted a modified design with the addition of a stabilization plate at right angles from the other plates at the base of the cylindrical rod (Fig. 48.12a). Alternatively, Chin designed a distractor called LEAD (Stryker Leibinger, Kalamazoo, MI, USA), with the rod passing through the alveolus and separating the miniplates, thus achieving distraction. A distractor was purposely developed for vertical distraction of the edentulous ridge by Groningen (KLS Martin Group, Tuttlingen, Germany) composed of two microscrews for activation and a position screw to minimize rotation of the fragment. Medartis and Synthes have successfully incorporated an angulating device to the distractor. In the Synthes design, there is a small green screw at the bottom of the activating rod to adjust the vector angulation (Fig. 48.12b, c). In the Medartis design,

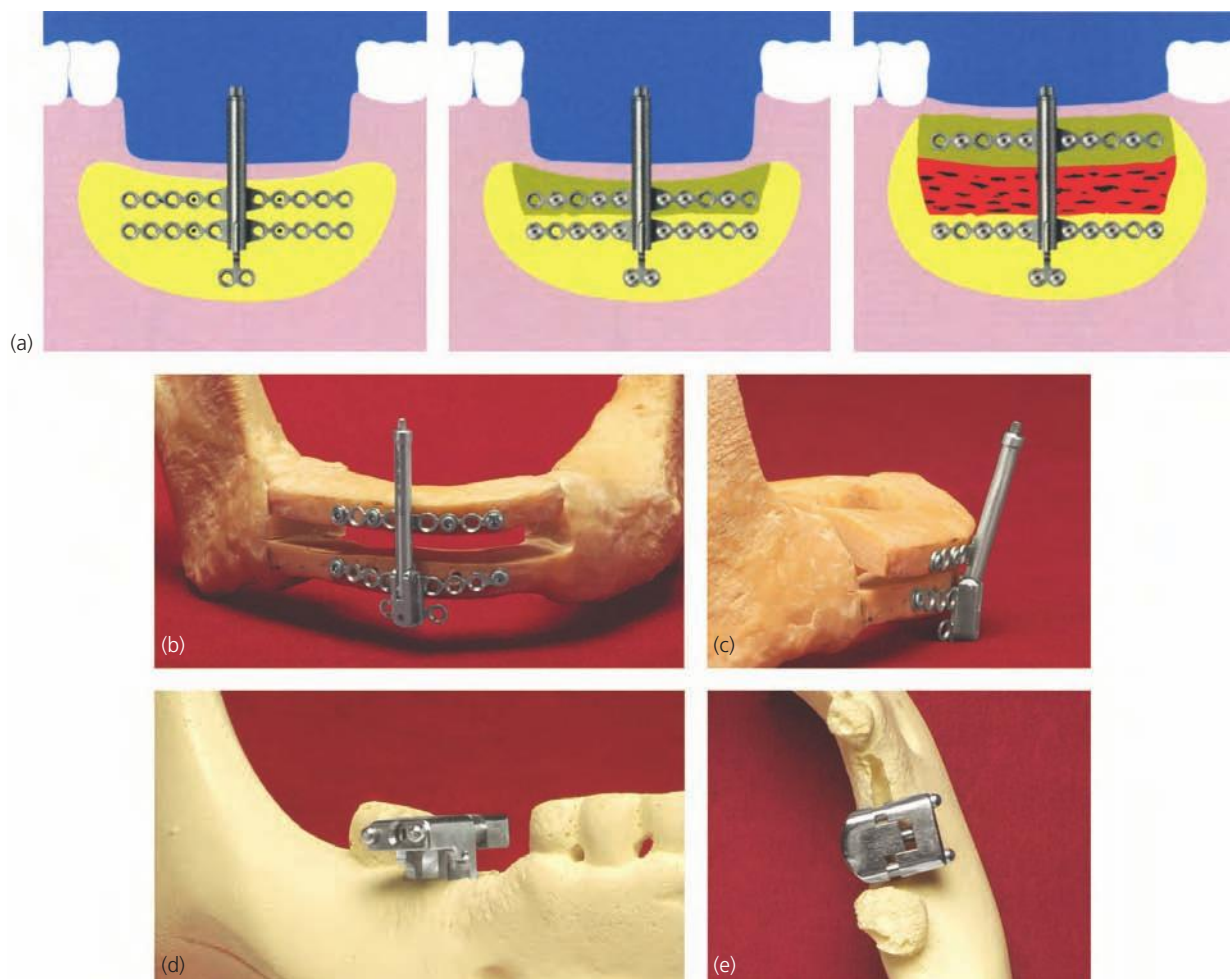


Fig. 48.12 Examples of alveolar distractors. (a) Alveolar ridge TRACK Distractor (KLS Martin). (b) Alveolar Distractor (Synthes). (c) Alveolar Distractor with vector angle adjusted (Synthes). (d) Crest Expander for transverse expansion of ridge (frontal view; Surgi-Tec). (e) Crest Expander (bird's-eye view; Surgi-Tec).

there is a thin rod running parallel together with the activating rod; this enables postoperative distraction of the upper fragment if needed. Surgi-Tec developed a robust vertical distractor that also allows simultaneous heightening as well as transverse movement of the distracted fragment. However, the vertical distraction length is limited to 10mm at a time and the tissue needs to be reopened for anything more than 10mm (up to 20mm).

Vertical ADO can be performed under either local anesthesia, intravenous sedation, or general anesthesia. The procedure involves an intraoral incision at the buccal vestibule, without lateral releasing incisions. Subperiosteal dissection should be performed to obtain adequate visibility of the underlying bone, but no mucoperiosteal dissection toward the alveolar crest and on the lingual side is required to preserve adequate blood supply for the osteotomized bone segment.

The intraoral distractor should be adapted to the planned site before the osteotomy. The bone segment can be completely separated from the basal bone using an oscillating saw or a surgical bur. The distractor is then fixed with mini-screws to both the basal bone and the osteotomized segment.

The osteotomized segment should first be activated by the distractor to check the direction of distraction and freedom in movement. Once movement in the line of distraction has been checked, the osteotomized segment should be repositioned with bone-to-bone contact until it is ready for the active distraction phase.

Transverse alveolar distraction

There are only two transverse alveolar distractors present on the market. One was developed in Japan and one by Surgi-Tec from Belgium. The Alveo-Wider from Japan consists of a malleable titanium mesh placed on the labial cortical plate and activation is by a small screw passing through the mesh and buccal plate to reach the lingual plate. Since the lingual plate is stable, on activation the buccal plate will be pushed laterally thus creating a distraction gap for bony regeneration. The mesh and micro screw are then removed at the time of implant placement. In contrast, the Crest Expander from Belgium is a flat device with four prongs sticking out at the bottom (Fig. 48.12d). The device is inserted following a similar osteotomy to that previously described but inserted from the crestal side rather than the buccal side (Fig. 48.12e). The low profile is advantageous to avoid any occlusal interference from the opposing teeth.

Transverse ADO or alveolar widening can be done either in one or two stages. For both procedures, it is normally done under local anesthesia where a crestal mucoperiosteal incision is made followed by buccal vertical mucoperiosteal incisions placed anterior and posterior to the distraction zone.

The crest itself is minimally exposed and a round bur is used to make a small trough along the crest. Bone cuts are made through the trough, and through the anterior and posterior vertical incisions with minimal stripping of mucoperiosteum using a sagittal micro saw or piezoelectric ultrasonic bone cutter. The use of piezoelectric bone cutting tools is particularly convenient for splitting of the thin alveolar ridge with minimal loss of residual bone.

An osteotome is normally used crestally and a "green-stick" fracture is produced in the buccal plate. The distractor is tapped into place before the wound can be approximated with sutures. For a two-stage procedure, the bone cuts are made during the first visit and the distractor will then be placed at the second stage. As in other distraction procedures, patients will undergo latency, distraction, and consolidation periods as described in the previous section. Removable prostheses are not allowed in the distracted areas until the distraction device is removed. It was also suggested that the ADO protocol should consider an overcorrection of more than 25% within the limits of the applied surgical protocol.⁹⁰

The duration of the consolidation phase seems to be the major factor that determines the duration of the overall treatment period and primary stability of implants placed at the augmented region.⁸⁰ Studies by Saulacic and Turker indicated that a period of 12 weeks is sufficient for bone maturation and for the site to be ready for implantation.^{80,81} However, another study suggested that implants could be placed in a distracted region within 4–8 weeks postoperatively because distraction osteogenesis heals faster than the time required for autogenous bone graft.⁸²

In the systematic review by Saulacic *et al.*,⁸⁰ the mean osseointegration period of 469 implants (dental implants and prosthetics treatable distraction implants) was 4.59 ± 1.34 months (range 3–8 months) with an overall success rate of 97%. ADO can provide a limitless vertical bone augmentation restricted only by the length of the vertical distractor design. A report on the ability of ADO to increase the width of the alveolar ridge in a transverse direction has been published by Laster.⁸² In that study, alveolar width in the range 4–6mm was successfully gained. The authors have successfully performed 20mm fibular distraction for heightening of mandibular defects.

It has been reported that ADO was successfully performed to gain both width and height to allow placement of 14mm implants in an initially narrow maxillary ridge.⁹⁶ A study conducted by Enislidis showed a high survival rate (95.7%) of implants following ADO.⁹² This was also supported by studies done by Chiapasco *et al.* and Jensen *et al.* with 100% and 90.4% implants survival rate, respectively.^{77,78} Recently, Uckan *et al.* also demonstrated that the survival rates of implants in bone regenerated by intra- and extraoral distraction were comparable, reaching 88% and 94%, respectively.⁹³

Although bone resorption in ADO was reported during the peri-implant stage, the incidence was consistent with implants placed in normal bone. The survival and success rates of implants were also comparable to cases in normal bone.⁸⁸ It was demonstrated that the distraction regenerate has a better ability to maintain the regenerated bone gained when compared to guided bone regeneration.⁷⁷ Thus, ADO is better in terms of achieving a long-term prognosis in maintaining bone gain while having similar peri-implant resorption to normal bone.

In a study to evaluate patients' perceptions of the technique, it was concluded that ADO had a high degree of acceptance among patients. The technique was rated as a much better procedure than conventional bone graft surgery among patients who had undergone both surgical procedures.⁹⁵

Temporomandibular joint distraction

Temporomandibular joint (TMJ) reconstruction is usually performed following removal of pathologies/diseases which affect the normal anatomic relationship of its core components: mandibular condyle, articulating disc, and glenoid fossa. The most common pathology is TMJ ankylosis, while other conditions which necessitate TMJ reconstruction include: advanced joint arthritis, degenerative changes, and benign neoplasm. The mandibular condyle is commonly involved in all the above conditions, and often needs to be excised. As a result, rebuilding of the condyle is regarded as one of the most important aspects in TMJ reconstruction.

The concept of TMJ distraction is that a neocondyle can be created in the ramus bone after loss or resection of the original condyle. A bony segment is made on the posterior mandibular ramus and then moved gradually towards the corresponding glenoid fossa guided by a distractor device in order to restore the normal anatomical relationship. This concept was first presented by Stucki-McCormick in 1997⁹⁷ and

was supported by an animal study, in which the neocondyle created could provide satisfactory biomechanical properties for normal functional loading.⁹⁸ Today it is a well accepted treatment modality for adult patients with good long-term stability and TMJ function.³³ In children, despite promising short-term results, current clinical evidence is limited by the relatively short follow-up period with respect to the long potential growing period of the mandible.⁹⁹ Nevertheless, a recent animal study has demonstrated the growth potential of the neocondyle created by transport distraction, particularly when the neocondyle is under functional stimuli.¹⁰⁰

The main goal of condylar distraction is to create a neocondyle in good anatomic relationship with the glenoid fossa. Unlike maxillary and mandibular distraction, in which the final distraction segment can be modified slightly by occlusal elastics, the final position of the neocondyle is highly dependent on the distraction vector. Good precision is necessary during the planning stage. 3D surgical planning using a stereo-model is therefore highly recommended. The advantages of stereo-model planning include:

1. Allows surgical simulation for resection of pathology and assessment of the extent of defect.
2. Helps the design of the distraction trajectory and the position of the distractor, by providing clear anatomic information of the surrounding vital structures.
3. Give good visualization of the glenoid fossa to estimate any further adjustment required for good housing of the future neocondyle.
4. Makes further 3D planning possible in situations when correction of additional dentofacial deformity is required apart from the TMJ reconstruction; in these cases combination of the stereo-model and dental casts is necessary.
5. Enables fabrication of vector-locating splint and titanium glenoid fossa for transfer of the planning information to the surgical environment.
6. Provides an excellent tool for surgeon-patient communication.

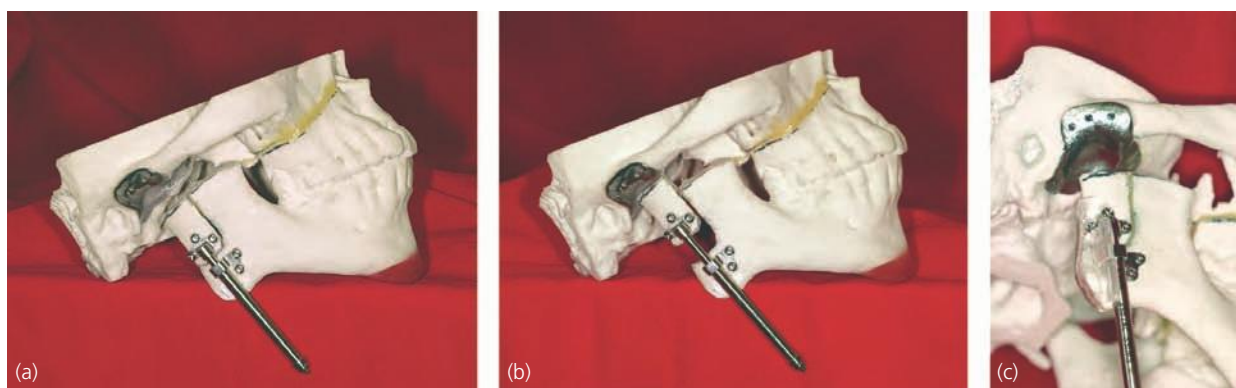


Fig. 48.13 Temporomandibular joint distraction. (a) L-osteotomy of posterior ramus, gap arthroplasty with titanium fossa and location of unilateral mandibular distractor (Synthes). (b) Simulation of the condylar distraction from activation of the distractor. (c) Localization of distractor with the aid of vector-locating splint.

On the stereo-model, a reverse-L osteotomy is designed in the posterior mandibular ramus behind the mandibular foramen. The dimension of the osteotomy segment should allow sufficient area of screw placement for fixation of the distractor on both ends (Fig. 48.13a). Sometimes minor shaving along the gonial angle is required to provide better adaption of the distractor arm. A titanium glenoid fossa can be fabricated if there is any risk of perforating the thin bony fossa. Upon completion of the distraction simulation (Fig. 48.13b), a vector-locating splint is fabricated³³ to register the distractor's position and angulation over the mandibular ramus and the gonial angle region (Fig. 48.13c).

Similar to presurgical planning, the placement of the distractor is usually the last stage of the surgery. While a preauricular approach is indicated for the excision of pathology affecting the TMJ, a submandibular approach is indicated for the placement of the distractor. Surgical access to the mandibular ramus for distractor placement is gained by a 3cm submandibular incision around the gonial angle. The preadapted distractor is placed on the lateral mandibular ramus, with the location being guided by the vector-locating splint. Temporary fixation is achieved with one screw placed on each end of the distractor. The outline of the osteotomy cut can be marked with a surgical bur before the distractor is removed. A reverse-L osteotomy is then completed using a surgical bur followed by an osteotome. Minimal mobilization is advisable in order to avoid any excessive detachment of the medial pterygoid muscle from the osteotomized bone segment. The distractor is oriented to the planned position and fixed with two to three screws on each side, guided by the vector-locating splint. The distractor is activated and tested before the wound is closed, to ensure that the transported segment is activated smoothly.

A 7-day latency period is recommended before the distractor is activated. A daily activation rate of 1 mm can be divided into two to four rhythms per day. A check radiograph is recommended 3–5 days after the initial activation to confirm that the osteotomy segment has been properly separated from the ramus. During the activation phase, the patient is encouraged to perform regular jaw exercises. The completion of activation is determined: (1) clinically, by checking for any sign of posterior open bite developing on the activation side; and (2) radiographically, using a panoramic radiograph to check whether the distracted bone has reached the desired docking location. At the end of the activation phase, the distractor is rewinded 1–2mm backward to avoid direct bone contact between the distracted bone segment and the glenoid fossa. A panoramic radiograph should be taken to confirm the position of the transport segment docking in the glenoid fossa.

During the consolidation phase, patients should be reviewed regularly until radiograph signs of bone refilling in the distraction gap. The distractor should

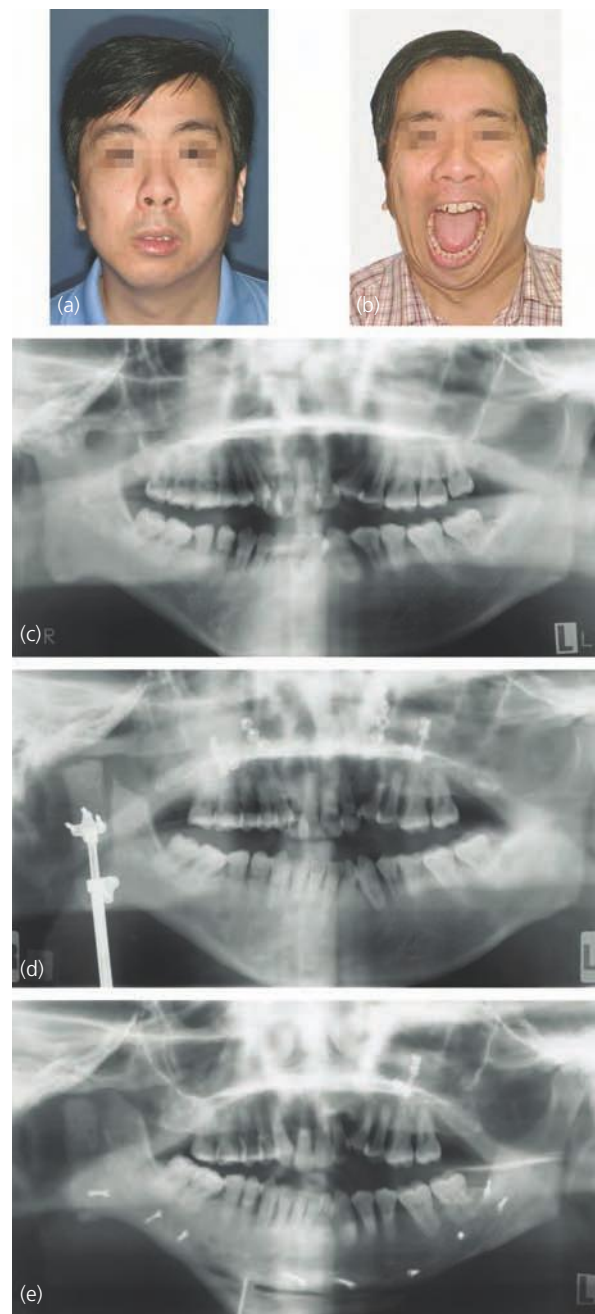


Fig. 48.14 A clinical case of temporomandibular distraction on a patient with TMJ ankylosis. (a) Preoperative frontal view shows inability to open the jaw. (b) Postoperative frontal view shows 50mm mouth opening. (c) Preoperative panoramic radiograph shows enlargement and fusion of right condyle. (d) Panoramic radiograph shows activation of the distractor. (e) Radiograph taken after removal of distractor shows bone regeneration along the distraction gap.

preferably be removed under general anesthesia. The most convenient way of gaining access to the distractor is via an intraoral approach using the previous incision line. An endoscope using a transbuccal route can also help to confirm bone formation in the distraction gap and to localize the screws during removal. The distractor is removed via the oral route, and the transcutaneous opening is then refreshed and closed primarily. A classical case of TMJ ankylosis

with reconstruction of the condyle by distraction is illustrated in Fig. 48.14.

Bone transport distraction

Extraoral devices for mandibular bone transport

The feasibility of bone transport in mandibular reconstruction was first presented by Costantino in 1990. Following a number of animal experiments proving successful regeneration of a bone segment bridging the defects, Costantino presented the first clinical report of bone transport for reconstruction of a segmental defect in 1995.¹⁰¹ An extraoral appliance was used, which was composed of a semi-circular external frame attached by two pairs of pins to the mandibular segments and a transport tram connected to the bone transport segment.

Using a distraction lengthening device attached to a reconstruction plate (Synthes, Paoli, PA, USA), Block presented four cases of bifocal distraction osteogenesis for mandibular reconstruction.¹⁰² A 7-day latency period was allowed before activation of 0.5 mm twice daily. The findings showed satisfactory

bony reconstruction without the need for bone graft, and successful recreation of the alveolar ridge and oral mucosa regeneration. In 1997, Fedotov reported the result of transport distraction using a semicircular apparatus on 22 patients with post-traumatic defects varying in size from 10–180 mm.¹⁰³ Depending on the size of the defects, the approach in bone transport would vary from monofocal, bifocal, or even trifocal distraction. In cases of trifocal distraction, two transport discs were created and distracted towards each other until they met. Further distraction can compress the transport discs at the docking sites with each other achieving body union forming a continuous mandibular bridge.

In 1997, Klein reported the use of a Frankfurt Craniofacial Distraction System (Normed Medizin-Technik GmbH, Tuttlingen, Germany) in six patients for reconstruction of mandibular defects.¹⁰⁴ This distractor was composed of a U-shaped gear rod for connection to the individual distractors and stabilizers (Fig. 48.15a). Fixation was achieved with transosseous pins. A 5-day latency period was allowed before activation of the device at the rate of 1 mm per day. Once the transport segment reached the docking site, a second surgery was required to clear the soft tissue from between the transport and normal bony

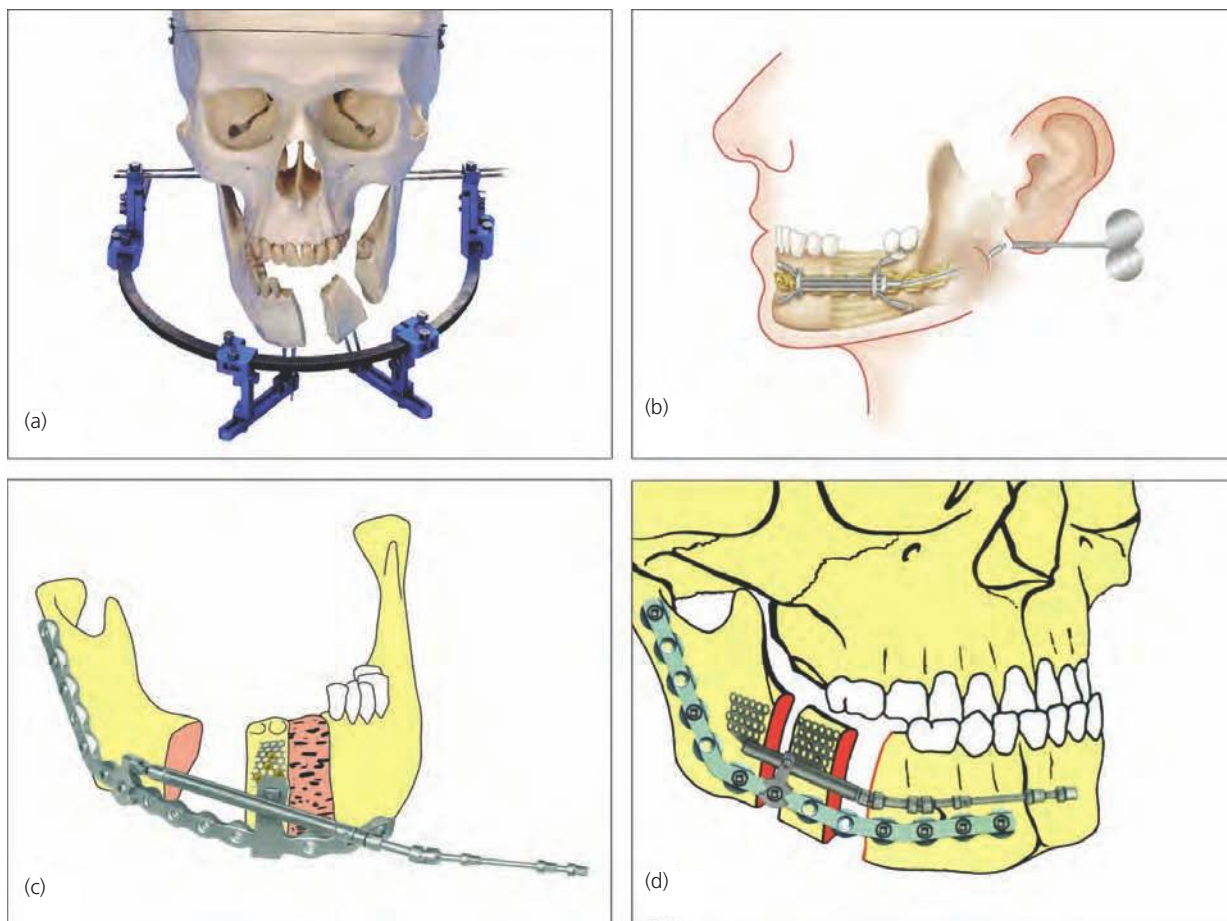


Fig. 48.15 Examples of mandibular bone transport distractors. (a) Frankfurt Craniofacial Distraction System (Normed). (b) DynaForm Intraoral Distractor with reconstruction plate (Stryker Leibinger). (c) Herford Transport Distractor (KLS Martin). (d) Threadlock Transport Distractor (KLS Martin).

segment before further activation to achieve compression union and the subsequent mandibular reconstruction without bone grafting.

It is noteworthy to point out that the above devices for mandibular bone transport are all made of bulky U-shaped external stainless steel rigid frames, which form the path of bone transport simulating the natural curvature of the mandible. Surgeons quickly became aware that the distraction regeneration always ends up in a linear bony bridge irrespective of the distraction pathway. When a transported disc of bone is moved along a curve, the collagen fibers within the distraction site always follow a straight line pattern regardless of the curve on which the transport took place.³⁸ Therefore, recent extraoral bone transport devices are based on a combination of customized assemblies of multiplanar distractors and reconstruction plates. Both the Multi-Vector Mandibular Distractor (Fig. 48.3g) and 3D Xternal Distraction System (Fig. 48.3h) can achieve a linear activation of up to 85 mm. In addition, the use of a light-weight carbon fiber rod greatly enhances patient comfort during the consolidation period and frees the distraction body for application in a different patient.

Intraoral distractor for mandibular bone transport

The clinical application of an intraoral distractor in bone transport was first described by Guerrero *et al.* in 1995¹⁰⁵ using the DynaForm distractor in combination with a reconstruction plate (Fig. 48.15b).

In 2004, Herford reported the use of a plate-guided distractor in four patients for reconstruction of a segmental mandibular defect.¹⁰⁶ The Herford Transport Distractor (KLS Martin, Jacksonville, FL) is an intraoral device that is secured via a titanium mesh to a reconstruction plate with a screw on one end and a transport bone segment on the other (Fig. 48.15c). A series of distractors for a maximal range of bone movement from 30–60 mm is available in this system. The superior portion of the mesh can be secured to the transport bone segment whereas the inferior portion is adapted around the plate. A screw-activated internal design combined with multiple swivel joints enables the transport segment to move along the track of a reconstruction plate to achieve distraction of a curved segment from the midline of the mandible all the way to the ramus. In addition, the same manufacturer also provides the Threadlock Transport Distractor which allows reconstruction of a straight mandibular continuity defect in the range of 30–50 mm (Fig. 48.15d).

Maxillary transport distraction

Liou developed a distractor for alveolar cleft transport (Fig. 48.16a). The Liou Distractor (KLS Martin Tuttlingen, Germany) can be used for the following

circumstances: bilateral or unilateral wide alveolar cleft or oronasal fistula in bilateral cleft lip and palate patients; maxillary alveolar bony defect due to trauma; creating interdental edentulous space to relieve maxillary dental crowding; and maxillary lengthening for maxillary hypoplasia. The device is available for left or right alveolar clefts in 15 mm and 20 mm sizes. In patients with posterior maxillectomy defect, a bone-borne transport distractor was designed by Cheung *et al.* (Fig 48.16b).¹⁰⁷ The custom-made transport distractor is based on a stereo-model generated from CT scan data. This device is made of stainless steel and was designed and manufactured by the industrial center at the Hong Kong Polytechnic University. The design is similar to the fourth-generation distractor used in their pilot study on monkeys.⁷⁵ The distractor consists of a cylindrical distractor body and three extension arms with two stabilization arms and one transport arm. The distractor can generate up to a 15 mm distraction path and is activated by a flexible distraction rod connected to the front end of the distractor body (Fig. 48.16c). The two stabilizing arms extend to the zygoma body and the pyriform bone, respectively. The transport arm is fixed to the transport segment by a threaded screw and there are four screw holes designed in serial direction for fixation to the bone transport segment. On activation the distractor can move the transport arm linearly along the distractor body via a distractor rod rotated in a clockwise direction. The distractor arms are prebent and simulated to the planned position on the patient's stereo-model.

Complications

The hypothesis that distraction osteogenesis is an uncomplicated procedure has been undermined by the reported complications that occurred among patients who underwent distraction in the maxillofacial region. The rate of published complications in distraction osteogenesis can vary from 27.7% to 40%.^{79,108} In general, complications can occur in four different stages: intraoperative, latency, active

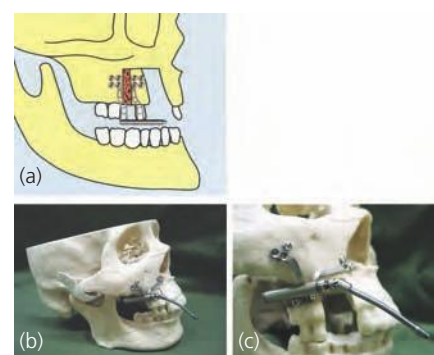


Fig. 48.16 Examples of maxillary bone transport distractor. (a) Liou Distractor (KLS Martin). (b) Cheung's Posterior Maxillectomy Transport Distractor. (c) Maxillectomy distractor under activation.

Table 48.3 Possible complications reported at different stages of distraction osteogenesis.

Stages	Types	Complications
Intraoperative	Device	Excessive length of the threaded rod Fracture Unstable placement
	Non-device	Excessive bleeding Fracture of the transport segment Incomplete fracture Jaw fracture Nerve transection
Latency period	Device	Loosening of distractor
	Non-device	Epistaxis Infection Wound dehiscence Skin irritation Diplopia Hypoesthesia Blindness
Active distraction	Device	Incorrect vector direction of distraction Breakage or loss of the distractor Osteitis around screws
	Non-device	Dishisence of the mucosa by the transport segment Wound dehiscence Skin fenestration Infection Pain during active turning of distractor Malnutrition Premature consolidation Temporomandibular joint pain
Consolidation	Non-device	Fibrous union Delayed wound healing Malunion Relapse Persistent nerve damage Infection Condylar resorption Hypertrophic scar

distraction, and consolidation (Table 48.3). These complications can be either device related or non-device related. The persistence of complications may be short or long term.

The activation rod has been reported to be one of the main sources of complications, presenting commonly as facial scars or recurrent infection around the rod exit (Fig. 48.17). These may result in disappointing outcomes for patients and surgeons.^{93,95} Other major complications such as fracture of proximal or basal bone were minimal. Long-term complications, including blindness, diplopia, TMJ pain, permanent nerve damage and neurological alteration, have been reported in the literature.^{59,79} Idiopathic condylar resorption of the TMJ related to mandibular distraction may occasionally occur resulting in anterior open bite and sleep apnea (Fig. 48.18).

**Fig. 48.17** Common complications related to distractors. (a) Facial scar from extraoral distractor retention and traction. (b) Intraoral infection around an activation rod.

In vertical ADO, complication rates vary from 66–100%.^{92,109–111} However, vector displacement has become one of the most frequent unpredictable complications that has unfavorable impact on dental implant placement.^{93,110} This may be due to mylohyoid and genioglossal muscle traction at the floor of the mouth, shape of the mandible, and lingual periosteum that attaches to the osteotomized segment acting as a hinge.¹¹⁰ Enislidis *et al.* reported that 37.8% of 45 distraction regions suffered from soft tissue dehiscence.⁹² Perdijk reported that fracture and decreasing residual bone height are the most common complications associated with ADO in atrophic mandibles, and these normally occur within the first postoperative year.¹⁰⁹

There are non-surgical issues that need to be taken into consideration when deciding whether a patient is suitable for this procedure. Financial consideration, which involves the cost of the distractor, and patient and family member's compliance to activate the distractor also play a major part in contributing to successful surgical outcome. Different complications may lead to different consequences. For example, intraoperative complications may lead to an extended surgery time, thus increasing the patient's morbidities. Vector problem may lead to occlusal interference and instability. In severe complications of vector malalignment, leading to anterior open bite, the patient may require reoperation for adjustment of occlusion.

It is important for surgeons to understand the causes of these complications so that they know how to prevent and treat them when necessary. Prophylactic antibiotic treatment and adequate mucosal closure can help to minimize infection around the distraction wound. Wound dehiscence can be minimized by smoothing the sharp edges of the transported fragment. If wound dehiscence occurs postoperatively despite the above measures, resuturing of the soft tissues should be done as early as possible to prevent the spread of infection and necrosis of the distracted bone. Neurosensory deficits can be minimized by correct execution of the osteotomy and placement of fixation screws. Resorption of the transported fragment can be minimized by an overcorrection of distraction or the use of elastic to unload the TMJ. Fractures of the transported fragment or basal bone can be minimized by correct use of osteotomies without undue force. Distraction should be stopped and the fracture treated with osteosynthesis if a fracture is identified during the operation.

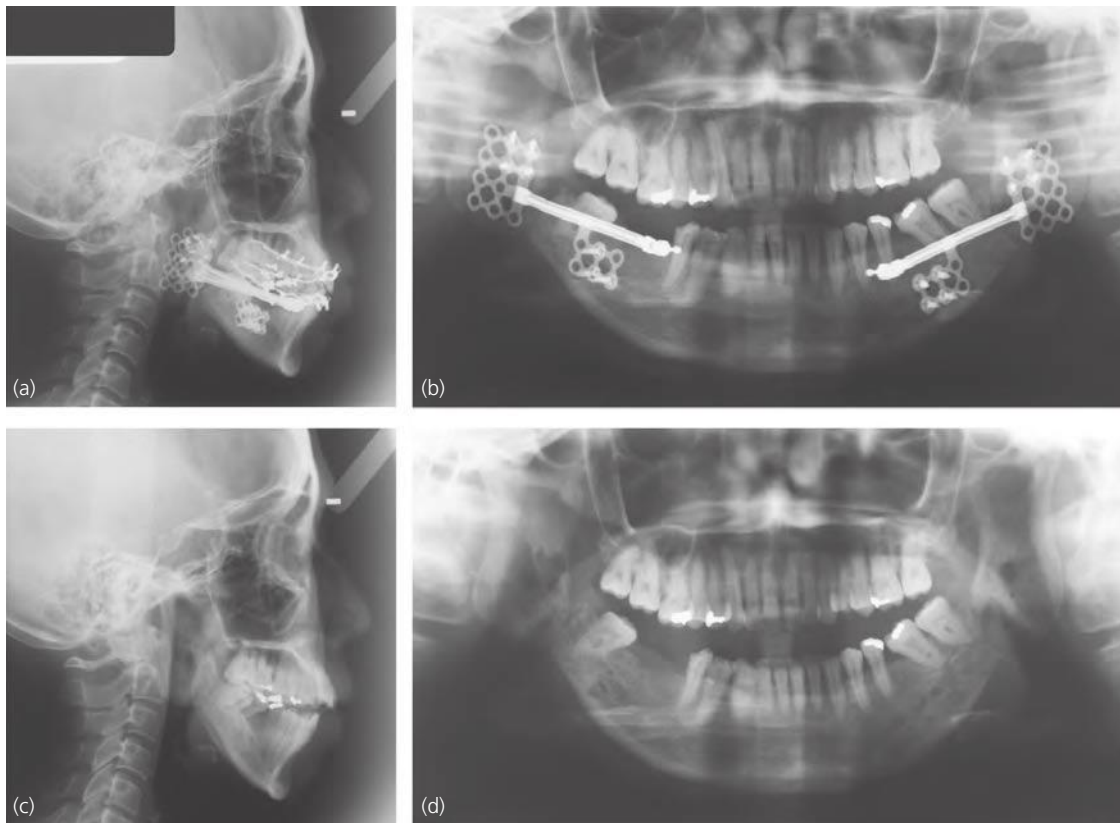


Fig. 48.18 Fibrous union and relapse of occlusion from mandibular distraction in a case with previous infection around distractor rod. (a, b) Apparently satisfactory healing and occlusion from intraoral mandibular distraction. (b, c) Relapse of occlusion to class II open bite shortly following removal of mandibular distractor.

Device-related complications may also cause serious unfavorable results. These include consolidation delay, fibrous union, and malunion. These can be largely prevented with an appropriate distraction rate and vector, correct stabilization of the distractor, and complete segmental osteotomy. Other conditions such as deviation from the correct distraction vector can be minimized with prior evaluation of the mucosal thickness and vestibular or lingual muscle insertion. Proper evaluation of the occlusion and avoidance of bone segment interference can prevent device fracture. If it occurs, immediate stoppage of distraction and fragment repositioning should be performed.

When unfavorable outcomes develop, the treatment strategy should be changed according to the severity and type of complications. Further delay of the distractor removal until consolidation can be attained in cases with large distraction and in medically compromised patients. In serious conditions, such as open bite from vertical ramus distraction, distraction surgery may need to be repeated with the consent of the patient.

Research and development in maxillofacial distraction

Distraction osteogenesis was originally applied to craniofacial syndromes in children and in clinical

cases beyond the biological limits of conventional orthognathic surgery. It was soon realized that distraction cannot replace conventional orthognathic surgery because distraction cannot achieve impaction, constriction, or retrusion of the bony segments. Through the experience of surgeons working in the cranio-maxillofacial region, the scope for clinical applications has increased and indications as well complications with this technique have been gradually defined.

There has been an explosion of distractor designs available on the market in the last 17 years. Further development is limited by the intermittent mode of distraction activation and the mechanical age may soon be replaced by biological modulation of distraction for compromised tissues and hosts. Emerging results of distraction from some new research directions are further elaborated below.

Administration of growth factors to enhance bone healing

Bone healing during distraction osteogenesis can be affected by many factors including the type of osteotomy, timing and rate/rhythm of distraction, stability of fixation, age of the patient, and underlying disease. Although there have been efforts to improve osteotomy techniques and stability of fixation, the overall treatment of bone lengthening still takes a long time.

The patient has to tolerate wearing a distractor for several months until the lengthened segment can bear the mechanical load. From the clinical perspective, surgeons in maxillofacial and orthopedic surgery would like to be able to reduce the lengthy treatment course.

Reduction in the consolidation period or increases in the distraction rate are possible options for reducing the treatment time. The biological environment created by distraction at a routine rate is superior to that with a rapid distraction rate.¹¹² A higher rate of distraction impairs angiogenesis and inhibits the expression of osteogenic growth factors, such as bone morphogenic proteins 2 and 4, leading to poor bone quality (Table 48.4).^{112,113} Great efforts have been made by researchers and clinicians to promote bone formation during distraction osteogenesis by local and systematic administration of angiogenic and osteogenic growth factors or cytokines, including bone morphogenic protein (BMP), transforming growth factor beta (TGF- β), platelet-derived growth factor (PDGF), vascular endothelial growth factor (VEGF), and fibroblast growth factor (FGF). Among all these growth factors and cytokines, BMPs play the most important role in bone healing and regeneration by inducing the osteogenic differentiation of mesenchymal stem cells, and have a synergistic effect with the angiogenic growth factor, VEGF. On a rabbit model of mandibular lengthening, recombinant human (rh) BMP-2 has been confirmed to enhance bone ossification at both routine and rapid distraction rates (Fig. 48.19). The addition of rhBMP-2 is able to compensate for the rapid distraction rate in distraction osteogenesis.¹¹⁴ The administration of growth factors and cytokines has demonstrated a promising way to accelerate bone healing resulting in a reduced treatment course. The effective delivery method, cost effectiveness, and biological safety still need further investigation.

Resorbable distractor

The parallel development of resorbable surgical materials and the application of distraction osteogenesis have led to merging of these technologies in the development of single-stage resorbable distractors. Interest was stirred in resorbable materials with the realization that mixing two different types of poly-

mers would result in markedly different resorption characteristics depending on the proportions of each polymer. These polymer macromolecules, when properly formulated, can serve a structural function and are then gradually degraded and resorbed by the body. Cohen *et al.*¹¹⁵ published the initial work using a maxillary distraction device made of a resorbable polymer in 2000, and this was followed with a series of new devices allowing one-stage distraction of the craniofacial skeleton. Resorbable mandibular distractors, when combined with advances in resorbable materials and distraction, may enable predictable distraction with a single operation.

The currently used polymers are not as strong as the equivalent volume of titanium. Therefore, resorbable devices tend to be thicker and bulkier. Rather than bending as is done with titanium plates, the resorbable materials are thermally sensitive and can be molded into a 3D shape after they are heated in a sterile water bath. This experience has led to the recognition that successful application of resorbable distraction requires modifications of the techniques used in the application of metallic distraction.

Continuous distraction osteogenesis

The quality of the newly formed bone by mechanical traction is highly related to the rate (the amount of bone lengthening in millimeters per day) and rhythm (in how many steps each day should the rate be achieved) of distraction. For a given rate of distraction, a higher rhythm of distraction has been confirmed to improve bone formation. Automatic distraction systems that can activate a high frequency or so-called continuous distraction provide a promising alternative in distraction osteogenesis. Micro-motor, micro-hydraulic cylinder, portable syringe, and infusion pump have been used to drive the distractor.^{116–119} Bone healing is accelerated after continuous distraction when compared with intermittent distraction. A major problem hindering the wide clinical use of automatic distraction systems is their size and weight.

A recently developed miniaturized automatic driver using the core of a clock, consisting of a high-torque movement circuitry, a step motor, and a gearing system, has been used to achieve an extremely

Table 48.4 Qualitative analysis of BMP-2 and -4 expression during rabbit mandibular distraction osteogenesis at different times.

Time of sacrifice	Osteoblastic cells		Fibroblastic cells		Vascularized fibrous matrix	
	Routine distraction	Rapid distraction	Routine distraction	Rapid distraction	Routine distraction	Rapid distraction
Day 1	++	+	++	+	++	–
Day 7	+++	+	++	+	++	+
Day 14	++	+	++	+	+	+

–, no positive staining; +, less than one third of cells positive; ++, one third to two thirds of cells positive; +++, more than two thirds of cells positive

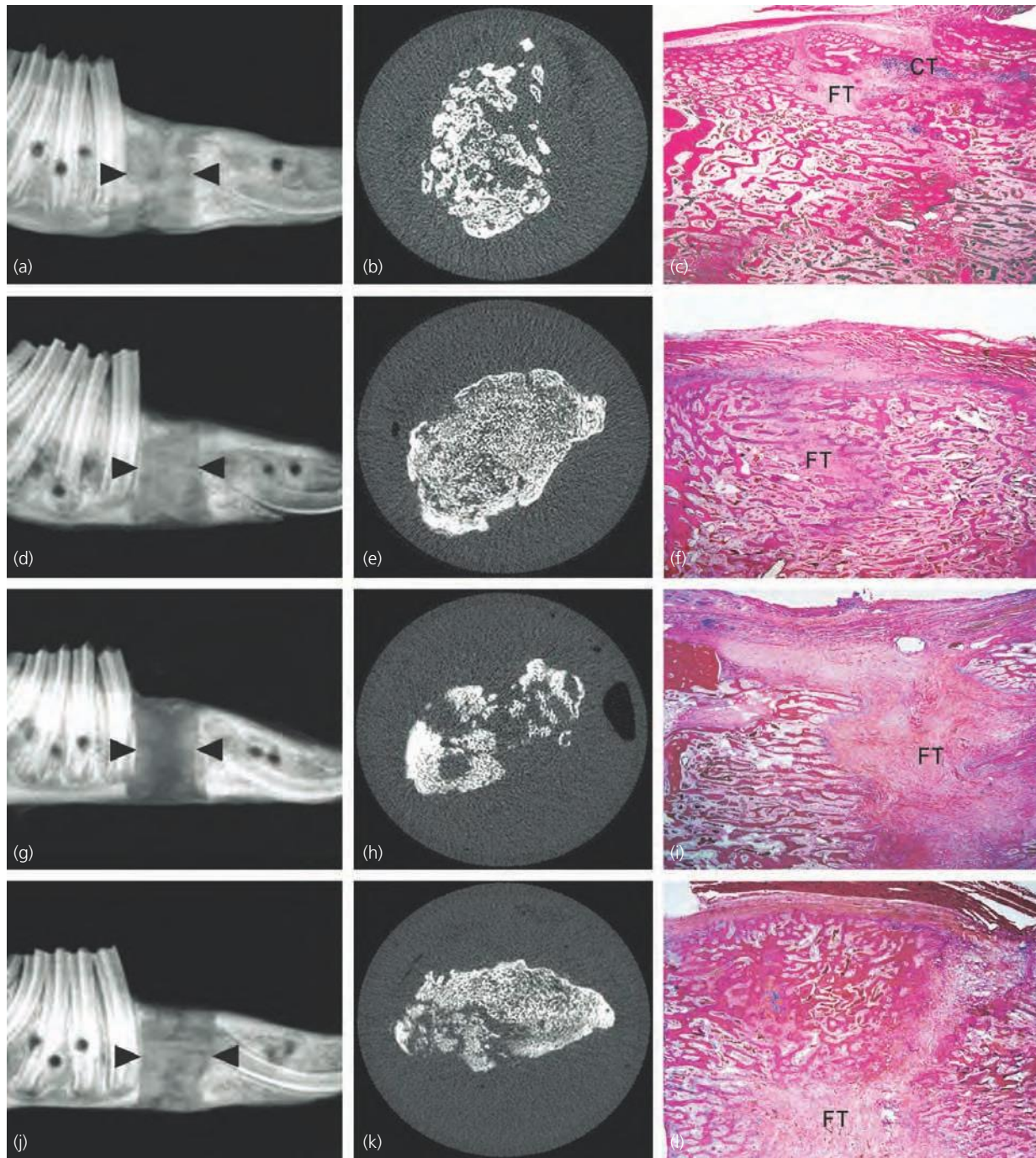


Fig. 48.19 The effect of rhBMP-2 injection on distraction regenerates under normal and rapid rates of distraction. Under routine rate distraction, plain radiograph, micro CT, and histological examination show obviously more bone formation in the BMP injection sides (d-f) than in the control sides (a-c). Under rapid rate distraction, bone formation is also significantly accelerated in the BMP injection sides (j-l) than in the control sides (g-i). (CT = cartilaginous tissue; FT = fibrous tissue.)

high frequency distraction of eight steps per second (Fig. 48.20).¹¹⁵ This automatic driver is an independent system and does not need a specially designed distractor. Using the specially designed mounting mechanism at the end of the flexible shaft, the automatic driver can be easily attached on most clinical distractors. The rate can be decreased or increased by adjusting the speed of the circuitry. This automatic driver not only demonstrates a great potential for further clinical application, but also provides a very useful tool to study the vascularization and molecular-biological modulation of high-frequency

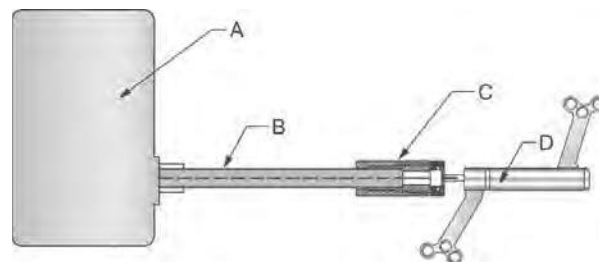


Fig. 48.20 Technical drawing of an automatic distraction system. A, driving unit; B, flexible shaft; C, mounting mechanism; D, universal distractor design. Torque generated by the driving unit is transmitted to the activation rod of a distractor through the flexible shaft and mounting mechanism.

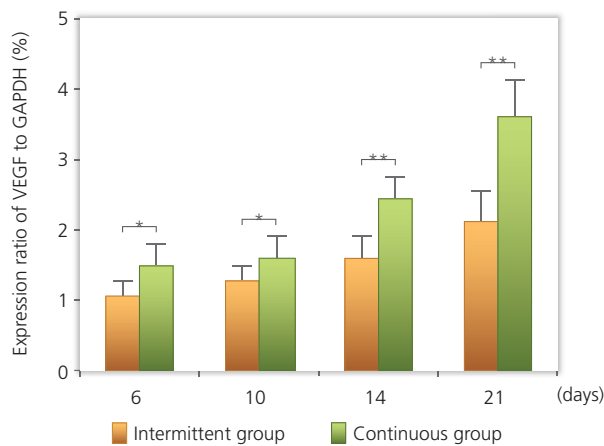


Fig. 48.21 Increased expression of vascular endothelial growth factor (VEGF) from continuous distraction when compared with intermittent distraction by real time PCR. *P < 0.05; **P < 0.01.

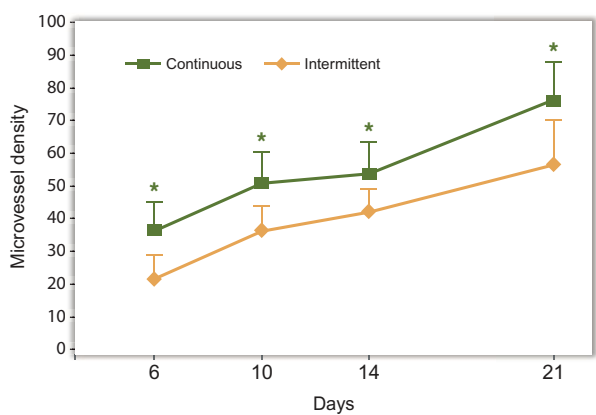


Fig. 48.22 Difference between microvessel density (vessels/mm²) in intermittent and continuous distraction in mandibular distraction regenerates. *P < 0.05; **P < 0.01.

Table 48.5 Histological evaluation of bone healing in the rabbit mandibular distraction regenerates according to the bone-forming indices (n = 40).

	Day 6		Day 10		Day 14		Day 21	
	Intermittent	Continuous	Intermittent	Continuous	Intermittent	Continuous	Intermittent	Continuous
Bone union	0	0	0	0.8 (0.45)*	1.2 (0.45)	2.6 (0.55)*	2.6(0.55)	3.8 (0.84)*
Bone maturity	0	0	0	1.6 (0.55)*	2.0 (0.71)	3.6 (0.55)*	3.4(0.55)	4.2 (0.84)
Marrow quality	0	0	0	0	0	1.6 (0.55)*	1.2 (0.45)	2.6 (0.55)*

Means (SD) for bone-forming indices. *P value of <0.05 was considered significant.

distraction. Study has shown that high-frequency distraction upregulates gene expression of angiogenic growth factors, such as VEGF (Fig. 48.21) resulting in enhanced angiogenesis (Fig. 48.22) and osteogenesis (Table 48.5).

Currently, surgeons can only examine the distraction progress and the mechanical condition of the distractor in person, which means frequent clinical appointments with the patients. It will save much time and trouble if the doctor can check the data feedback from the distraction system and adjust the treatment protocol at a distant site. With the possible availability of a remote-controlled distraction system, capable of transmitting information through the internet, surgeons could supervise patients more easily and provide instant treatment modification if needed. The automatic driver system in combination with remote-control technology would provide a change in the future application of distraction osteogenesis.

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Chapter 49a

Craniofacial Syndrome Patients – Reconstructive Surgery

Peter Tarnow

The chapter is an overview of reconstructive surgery in craniofacial syndrome patients. Craniofacial surgery deals with congenital deformities, trauma, and tumors of the craniofacial skeleton and the surrounding soft tissues. This chapter will present the symptoms and treatment of craniosynostosis and craniofacial syndrome patients focusing on cranial and upper facial surgery. The type and timing of surgical interventions are discussed, as well as the importance of dedicated team work.

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Introduction

The term craniofacial syndrome refers to patients born with a pattern of disease that involves the craniofacial skeleton. This may involve the premature closure of sutures in the cranial vault, the cranial base, and in the midface, leading to growth disturbance, abnormal function, and distorted appearance. Many carry identifiable genetic markers that are often used in diagnosis. Similar terms, which may be used interchangeably, are craniofacial dysostosis or craniofacial dysostosis syndromes. Craniofacial syndromes are rare and often hereditary, the more common among these are the syndromes described by Saethre–Chotzen,

Crouzon, Apert, and Pfeiffer, which are named accordingly.

Craniofacial surgery is a collection of advanced techniques for reconstructive and restorative surgery for patients with craniofacial malformations. The French plastic surgeon Paul Tessier is considered by many to be its father,¹ having pioneered many novel techniques which are still used today. In these patients, craniofacial surgery aims to improve function, development potential, and appearance by repositioning the bones of the face and skull through osteotomies as well as manipulation, reconstruction, or augmentation of soft tissues. Scarring is minimized using incisions that are easily hidden. The techniques and philosophies have now spread around the world and numerous centers of craniofacial surgery have been established to take care of and treat these patients. Since its inception craniofacial surgery has expanded and evolved tremendously. Techniques have been refined, indications standardized across the world, and more consistent outcomes achieved. Craniofacial surgeons also provide comprehensive treatment of craniofacial trauma, malignancy, and periorbital surgery, and often perform facial esthetic surgery.

A team approach to the treatment of these patients and their families significantly improves outcomes and cannot be emphasized enough.² The days of isolated surgeons dabbling in craniofacial surgery are over. A specialized surgical team must include craniofacial surgeons, neurosurgeons, oral surgeons, anesthesiologists, ophthalmologists, and otolaryngologists. Dentists, orthodontists, pediatricians, and allied medical professionals all contribute significantly. All must have an interest in craniofacial surgery and be able to provide long-term commitment to the regular follow-up of patients and their families. All should contribute to a longitudinal treatment plan which maximizes the growth potential and development of the child as well as functional and esthetic outcomes. It is important to recognize that the end result of surgery may not be apparent for many decades after any intervention and that long-

term follow-up and accurate record keeping are required. Decisions regarding the timing of interventions are critical as the operations influence growth outcomes. For example, the child's ability to quickly reossify calvarial defects during the first year of life can be taken advantage of by timing any skull osteotomy surgery at this age to allow rapid healing. On the other hand, early interventions in the upper and lower jaws may diminish their growth potential substantially by causing restrictive scarring and may cause long-term secondary deformity.

Diagnosis and preoperative evaluation are based on clinical examination. Three-dimensional computed tomography (CT) scans are routinely used for diagnosis, planning, and follow-up, but a range of diagnostic tools may help each patient depending on the specific situation.

Craniosynostosis

Craniosynostosis is a major feature of most craniofacial syndromes, and a comprehensive understanding of it is necessary to provide adequate treatment. Craniosynostosis is the premature fusion of one or more cranial sutures which results in growth disturbances of the skull. The growth is restricted perpendicular to the fused suture, as first described by Virchow in 1851.³ It is a relatively common disorder with a birth prevalence of about 1/2000–2500 newborns.⁴ Craniosynostosis will lead to morphologic skull and face deformities, but can in addition affect intracranial pressure with psychological or functional consequences like learning disabilities, visual and hearing impairment or loss, or even death.

At this time the treatment of craniosynostosis still remains surgical despite significant progress in the understanding of genetic and molecular biology involved in the malformations.⁵ Craniosynostosis syndromes are often a result of mutations in fibroblast growth factor receptor genes FGFR-1, FGFR-2, and FGFR-3, or mutations in transcription factors TWIST and MSX2.⁴ The fate of cranial sutures is, however, a complex process involving the dura mater, periosteum, sutural mesenchyme, and bone. Molecular signaling by different growth factors and their receptors, bone morphogenetic proteins and their inhibitor noggin, and extracellular matrix components all take part in cranial suture biology.⁵

Operative procedures to treat craniosynostosis are usually carried out during the first year of life. There are many different operative techniques employed around the world, and techniques continue to evolve. Early surgical procedures included the commonly performed linear craniectomy which was thought to release the normal growth of the underlying brain and allow perpendicular expansion of the skull. Experience has shown that this did not occur and that the surgically created suture rapidly closed. Later experiments with the interposition of a polyethylene

film⁶ were equally disappointing. Occasionally, such an operation could be successful if performed during the first months after birth with mild or incomplete craniosynostosis. Later cranioplasty techniques achieved better, more consistent results by more extensive operations using combinations of static and dynamic techniques. Whilst more successful, these operations carry a much higher risk of complications, longer hospital stays, and increased need for blood transfusion. In the last decade, however, there has been a renewed interest in minimizing procedures, and techniques using springs,⁷ distraction,⁸ helmets, and endoscopy⁹ are being employed.

Sagittal synostosis

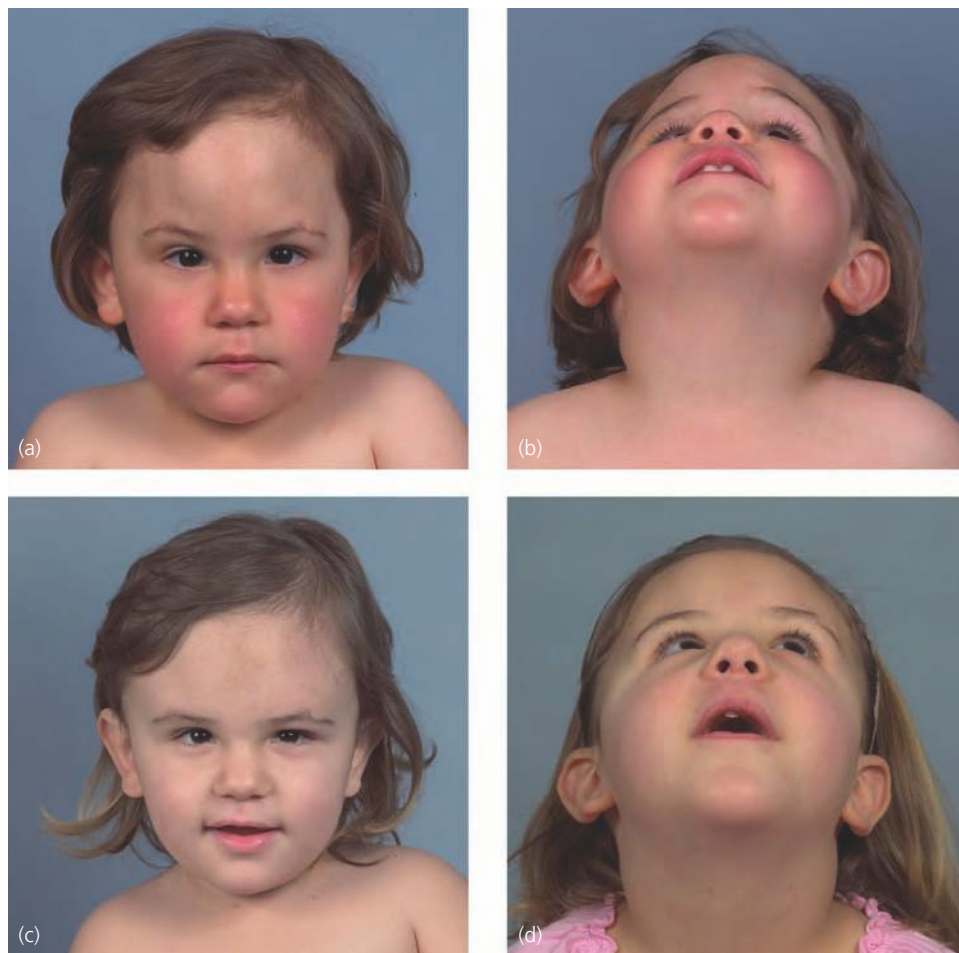
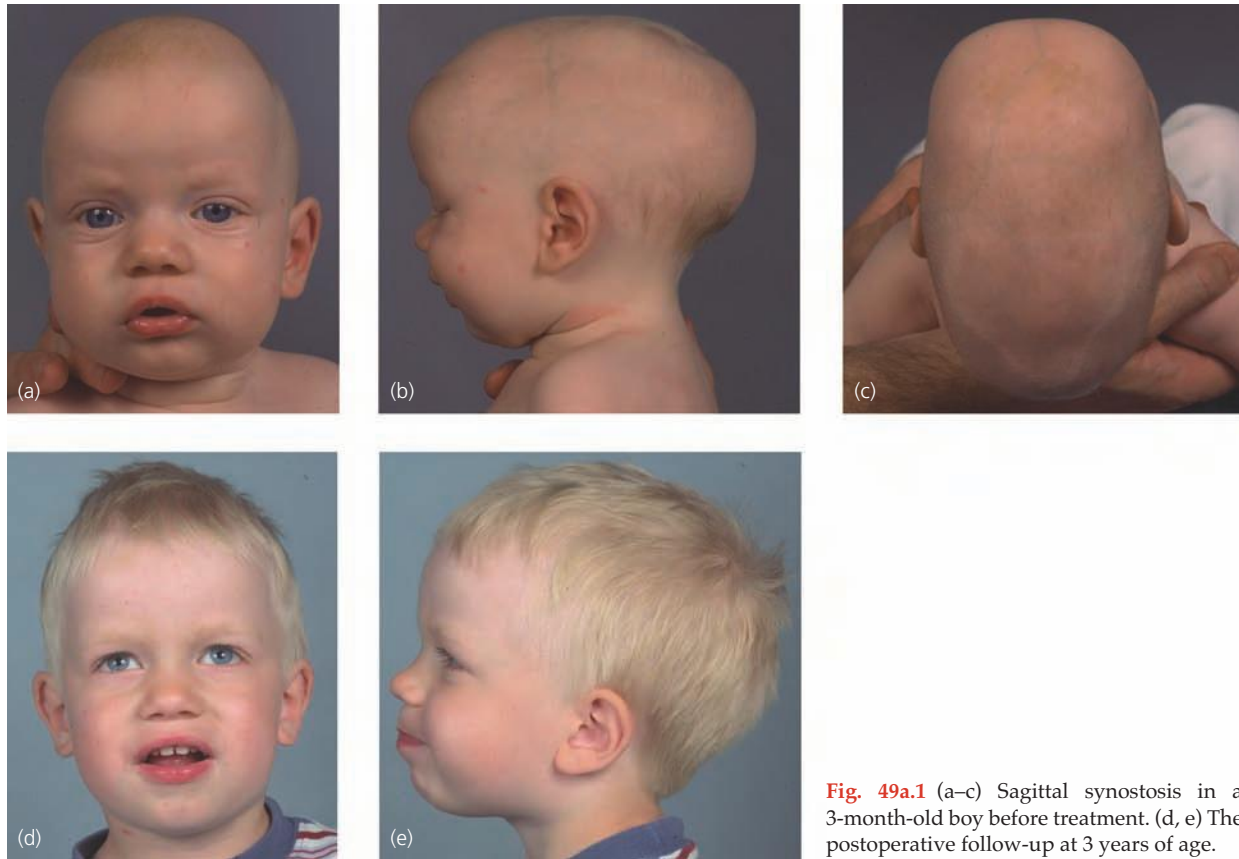
The most common craniosynostosis is sagittal synostosis. The deficient growth results in a long and narrow skull, with the skull forming the shape of a boat keel or "scapha" when viewed from above (scaphocephaly) (Fig. 49a.1). Different patients may be more or less affected, from a slight elongation and a ridge of the suture to a severely deformed skull with frontal bossing, hypertelorism, and a long and pointed occiput.¹⁰ Surgery of sagittal synostosis might be a strip craniectomy during the first months or widening and shortening of the skull with a cranioplasty (Fig. 49a.1).¹¹ Results of a suture release can be enhanced using spring techniques.⁷ Most authors believe better results occur through early intervention, which of course is dependent on an early recognition, diagnosis, and patient referral¹² in the first months of the child's life.

Metopic synostosis

Synostosis of the metopic suture gives a pointed and narrow forehead and a compensatory widening of the back of the head. The closed suture is ridged and the skull looks triangular from the top, leading to the descriptive name trigonocephaly. A narrow distance between the eyes (hypotelorism) and epicanthal folds may be seen. The incidence is 1/15000 live births with a male predominance.¹³ There is also a concern regarding cognitive impairment in this population.^{14,15} The surgical approach is to make a rounder and wider forehead, increase intracranial volume anteriorly, as well as to reposition the orbits (Fig. 49a.2). The hypotelorism may be further improved or normalized by the use of springs.¹⁶

Unicoronal synostosis

Unicoronal synostosis or frontal plagiocephaly may be either left- or right-sided. The facial midline will deviate. The forehead on the affected side does not grow normally and stays flat. The orbital rim is dislocated upwards and backwards. On the opposite side the forehead has a compensatory bossing and the orbital roof is pushed downwards.¹⁷ If untreated, the



fronto-orbital dysmorphology will worsen with time. The suture is sometimes palpable as a ridge and the anterior fontanel is triangular,¹⁸ with the base on the affected side. Surgical correction aims at correcting the asymmetry of the frontal bone and superior orbital ridges (Fig. 49a.3).^{19,20} Ocular strabismus is common with unicoronal synostosis.²¹

Bicoronal synostosis

When both coronal sutures fuse prematurely, the skull cannot grow normally in the sagittal direction. The shape of the skull will be wide, short, and tall (brachycephaly) (see Fig. 49a.6). The forehead will show an upper bossing and the orbits are shallow with a repositioned supraorbital bar.²² Bicoronal synostosis may be an isolated disorder, but is sometimes a part of a craniofacial dysostosis syndrome. The suture may be an easy palpable ridge. In addition to the obvious skull shape appearance, X-ray examination shows an upwards displacement of the greater sphenoid wings bilaterally in the frontal projection. Surgery will aim at normalizing the shape of the forehead, reducing height and width of the skull, and allowing anterior–posterior elongation. This has been done using different techniques, such as the “floating forehead”, static or dynamic cranioplasty.^{23–25}

Lambdoid synostosis

Lambdoid synostosis is a rare form of craniosynostosis. It can affect one or both sides. If bilateral, the condition may seriously affect the intracranial space. In unilateral lambdoid synostosis the affected side of the occiput is flat with an ipsilateral occipitomastoid bossing and a contralateral parietal and frontal bossing. The skull shape looks like a trapezium both from above and behind.²⁶ The skull base on the affected side will assume a lower position compared to the unaffected side as visible on X-ray examination. The overall deformity of the skull resulting from untreated premature lambdoid synostosis is significant. Surgery will restore volume and convexity on the affected side.²⁷

Positional plagiocephaly

Positional plagiocephaly refers to the condition where a plagiocephaly is not associated with a premature craniosynostosis. Positional plagiocephaly is caused by external pressure on the soft infant skull. This may be a result of a sustained unbalanced position of the infant’s head. It is more common in premature infants due to the soft skull. A significant rise in positional deformities has been seen in the last decade due to supine positioning of children to prevent sudden infant death syndrome (SIDS). Since the recommendations of the “back to sleep” campaign a fortunate decrease in SIDS has been seen, however this has been accompanied by an increase in positional skull deformities. Infants who are placed in the supine position will sometimes lie on the same side of the back of the head. The occipital area will flatten on this side, further provoking the head to fall into the same position. If untreated, a compensatory bossing of the ipsilateral forehead will take place, further accentuating the deformity.²⁸ These conditions are treated by altering the positioning and by using a soft pillow.²⁹ The use of helmets may reverse the deformity more rapidly. The sooner the treatment is started the faster the recovery.³⁰ Surgery is best avoided in these children, with an exception for extreme cases not responding to conservative treatment. Prevention is the best treatment.

Craniofacial dysostosis syndromes

Craniosynostosis occurs as part of the craniodysostosis syndromes and commonly affects the cranial base and midface as well as the cranial vault. In addition to this restricted growth of the skull and the face, there are also different skeletal deformities and other organ malformations associated with these syndromes. There are more than 100 such syndromes described in the literature; most of them are very rare. They are of genetic origin, usually inherited as an autosomal dominant trait. Mutations are mostly found in the fibroblast growth factor receptor genes.³¹ Since there is no medical or genetic cure available yet,

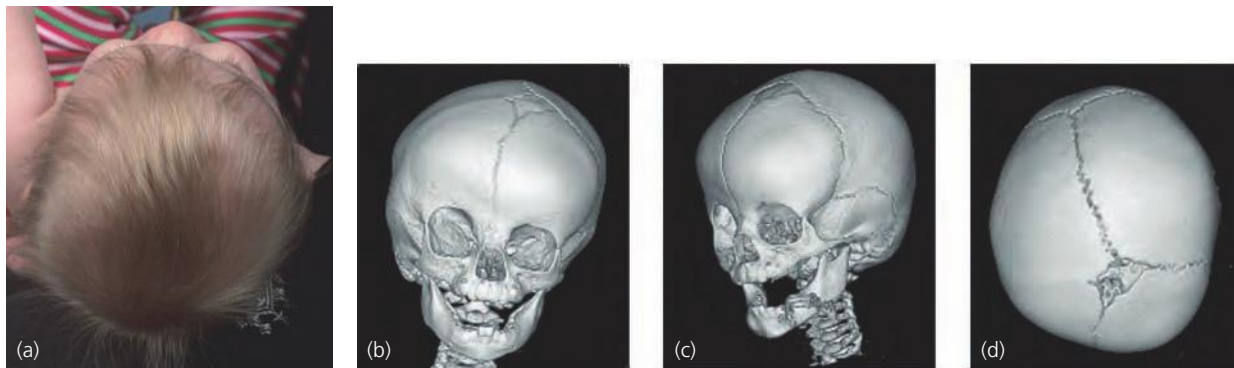


Fig. 49a.3 (a) Unicoronal synostosis of the right side with flattening of the ipsilateral forehead. (b–d) CT scan shows a fused right-sided coronal suture with forehead and orbital asymmetry with a deviation of the metopic suture.

surgery provides the best treatment for these patients. A typical treatment plan will span over 20 years in most patients. The timing and choice of reconstructive procedures vary in different parts of the world, but the main indications for surgery are generally agreed upon.

Treatment in infancy

A child with a craniofacial dysostosis syndrome is often recognized at birth or shortly after due to the obvious clinical manifestations. Many craniofacial dysostosis syndromes result in deficient growth of midfacial structures, and the child's airway may have an increased resistance to airflow. As infants breathe through their nose obvious breathing difficulties will appear in these children leading to nocturnal apneas and snoring, and, if severe, hypoxia. Severely affected individuals may need a secured airway through a nasopharyngeal airway or a tracheostomy.

A restricted airway will also affect breast-feeding. Since the child cannot breathe normally through the nose, breast-feeding can be an impossible or difficult task for the child, resulting in feeding problems and failure to thrive. A nasogastric tube or a gastrostomy may solve these problems during this period, which can extend for years.

When cranial sutures are not patent, the brain may not have enough space for its rapid growth during the first year of life. This will lead to a raised intracranial pressure. If untreated, this raised pressure can cause behavioral and cognitive disturbances, deafness, blindness, or even death. Intracranial hypertension is a difficult diagnosis to make in this population. Invasive intracranial pressure (ICP) measurements can be made, but there are no defined limits of a harmful pressure in these children. A radiograph will reveal a "beaten-copper" appearance of the cranial vault. A CT scan may show similar bone deficiencies, and small or absent liquor cisterns. Ophthalmologic examination reveals a papillary edema of the optic nerve.³²

Accumulation of cerebrospinal fluid (CSF) within the cranium, hydrocephalus, is closely associated with craniofacial dysostosis syndrome patients. This is in contrast to non-syndromic, mono-sutural craniosynostosis, where it is rarely seen. The diagnosis of hydrocephalus is difficult, since a large number of craniofacial dysostosis patients will present with a ventriculomegaly that is not progressive and not shunt-dependent. In addition the hydrocephalus may be masked by a simultaneous craniostenosis causing a high intracranial pressure and small ventricles. The intracranial volume will be compromised by the craniostenosis at the same time as the hydrocephalus will try to expand the intracranial volume. Thus, repeated clinical follow-ups using magnetic resonance imaging (MRI) or CT scan and fundoscopy both before and after cranial surgery will be neces-

sary for a correct diagnosis of the shunt-dependent hydrocephalus.

The etiology of progressive hydrocephalus remains controversial. It may be the result of a constriction of the posterior fossa with a resulting increase in CSF outflow resistance. Another theory is that the venous outflow from the cranium is constricted leading to impaired CSF absorption.

Ventriculoperitoneal shunting is the treatment of choice. It is a surgical treatment with a not insignificant morbidity and should be reserved for those patients having a truly progressive hydrocephalus. These patients have to be monitored for over- or undershunting.

There are several kinds of visual impairment that affect craniofacial syndrome patients. Optic atrophy, amblyopia, corneal scarring, strabismus, and refractive disorders are common findings. Furthermore, malformations of brain tissue, eye, extraocular muscles, and orbit may occur in a wide spectrum. Optic atrophy with deteriorated vision or blindness can be present already at birth or will develop as a result of increased intracranial pressure. Fortunately, the surgical expansion of craniostenosis will release the pressure on the optic nerve and may hinder the progress of optic atrophy. In patients at risk of raised intracranial pressure, there is a need for repeated, careful ophthalmologic assessment using fundoscopy to detect a raise in intracranial pressure and prevent irreversible visual problems.

Amblyopia is a permanent loss of vision due to a suppressed eye that has not been stimulated due to a squint. Strabismus is common among these patients, and early treatment may restore binocular vision. Refractive problems are very common and need attention and correction with proper glasses.

When exorbitism is present in a child with shallow orbits, the eyelids sometimes cannot close properly, which may lead to corneal drying, ulcers, and scarring. The unprotected eye may also be damaged by trauma. In severe cases the eye may dislocate anteriorly requiring emergency repositioning and tarsorrhaphy.

Crouzon syndrome

The cranial and upper facial features of Crouzon syndrome are craniosynostosis, exorbitism, and a hypoplastic midface. The craniosynostosis is usually bicoronal, but may affect other sutures alone or in combinations (Fig. 49a.4).³³ Intracranial volume can be threatened by craniosynostosis, not allowing enough space for the growing brain. The exposed eyes will have a higher risk of becoming traumatized and dry out, and the resulting keratitis can cause scarring of the cornea and visual impairment. In addition, there is often a complex divergent strabismus.³⁴ Raised intracranial pressure and exposed eyes necessitate early intervention to gain intracranial volume, reshape the skull, and protect the eyes.

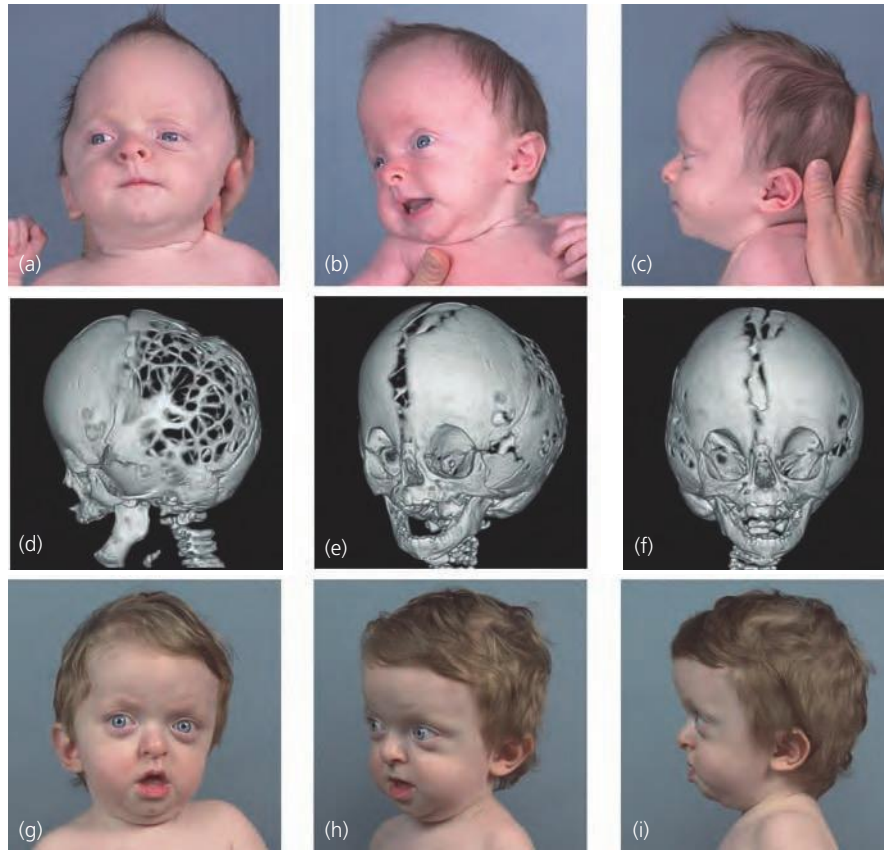


Fig. 49a.4 (a–c) Crouzon syndrome with brachycephaly but mild midface hypoplasia in a boy at 2 months of age, preoperatively. (d–f) CT scan showing extreme cranio stenosis with the brain growing through the cranial bones. (g–i) The same boy at postoperative follow-up after cranial expansion, here at 1 year of age.

Cranial vault surgery is performed during the patient's first year. The author prefers an early intervention when the child is about 4–6 months old. The reasons to perform early surgery are that the bone is more pliable, secondary deformities will be less, and the use of springs to enhance results will be possible.⁷ Other authors prefer to wait until 9–10 months of age to possibly lessen the need for repeat early cranial vault surgery. The surgery is performed by the standard coronal zigzag incision. Fronto-orbital advancement and reshaping is done, with narrowing of the broad skull shape. Particular attention is paid to the supraorbital complex, which usually has a concave deformity. This maneuver will also add some protection to the eyes. The occiput may simultaneously be advanced backwards either statically or by the use of springs. In some patients additional surgery such as ventriculoperitoneal shunting for hydrocephalus, lateral tarsorrhaphies, or repeat cranial expansion may be necessary.

During childhood the Crouzon patient will suffer from the effects of midface hypoplasia, including a compromised airway (sleep apnea), exposed eyes, speech difficulties, or from psychosocial problems relating to their distinctive appearance. Planning corrective surgery for this in childhood is controversial and many authors try to delay interventions for as long as the child and parents will allow. Facial growth will inevitably suffer from surgery, and while short-

term amazing results can be achieved these tend to relapse and disappear completely when the rest of the child grows into adolescence. This is something to be aware of when performing extensive surgery during childhood in Crouzon and other craniofacial dysostosis patients. The surgeon planning any intervention must be looking after the child and family as a whole and be thinking 20 years into the future, with each intervention building on the last. There is no plan that will apply to all patients, and the craniofacial surgeon must always consider the individual and their family, with his or her unique dysmorphology, and make a treatment plan that will best fit their needs.

Surgical treatment of the midface hypoplasia may be performed by a Le Fort III osteotomy, a monobloc osteotomy, a facial bipartition, or other combinations of osteotomies. Distraction osteogenesis techniques are used to bring bone segments further than with the use of traditional static techniques.³⁵ Long-term results have shown that despite surgery, relapse in adolescence may require additional or repeated procedures to achieve a satisfactory outcome (Fig. 49a.5). Growth of facial structures appears to be inhibited as part of the underlying syndrome, and surgery during childhood may negatively affect the remaining growth potential making these patients prone to relapse. The placement of long-term, indwelling, osteodistraction springs has been tried to prevent relapse following Le Fort III osteotomy.⁷

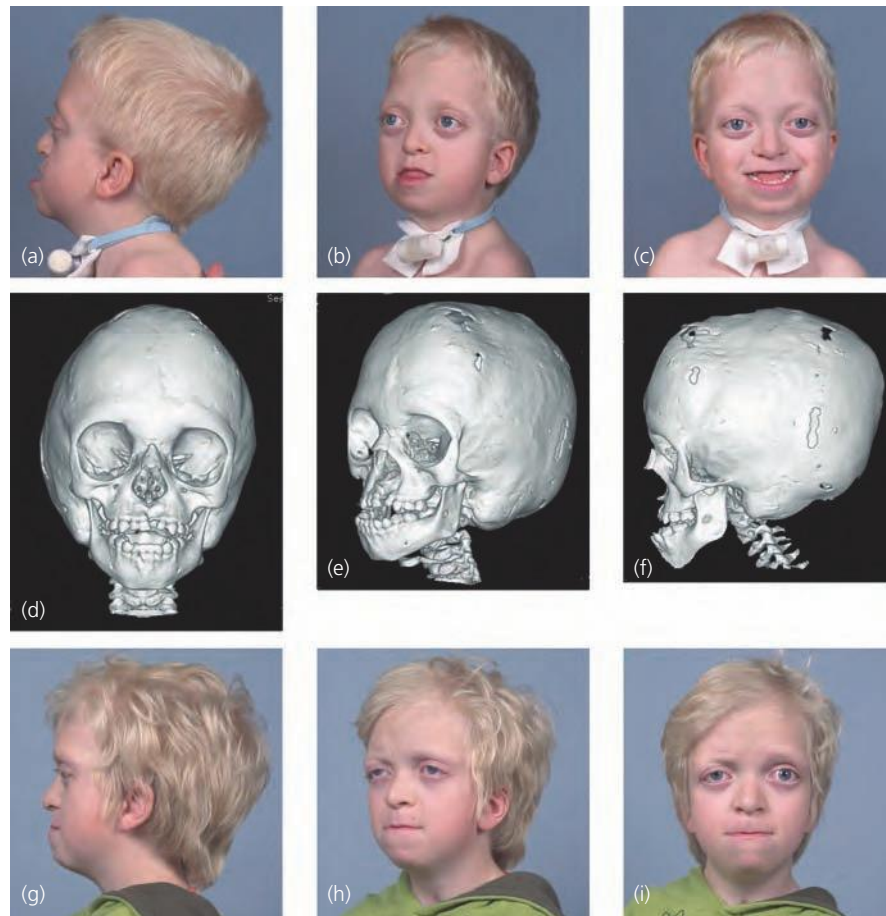


Fig. 49a.5 (a–c) Severe form of Crouzon syndrome at 5 years of age, still requiring a tracheostomy to be able to breathe. (d–f) Three-dimensional CT scan showing midface hypoplasia. (g–i) 3 years later after monobloc advancement, the child is no longer in need of a tracheostomy but there is still hypoplasia of the midface.

The final reconstruction of the Crouzon patient has to be done after skeletal maturity. The patient usually presents with shallow orbits, midface hypoplasia, and a class III malocclusion. It is not advisable to try to correct all deformities in a one-stage procedure. Orbital surgery or Le Fort III surgery is better performed as a first stage, and when the effect of that operation is stable a final orthognathic surgery is done. The author's preferred method is to address the shallow orbits by advancement and rotation of the entire orbital frames; infraorbital rims are advanced more than the supraorbital ones. This "spectacles-plasty" is an alternative to a Le Fort III or monobloc procedure.³⁶ When stable results are achieved and orthodontic treatment done the orthognathic surgery is performed.

Besides the more extensive surgery described, several minor surgical corrections may be useful for an esthetically pleasing result. Lateral canthopexies, rhinoplasty, genioplasty, contouring of bone irregularities, and fat grafting are a few examples of operations that can enhance the final result.

Pfeiffer syndrome

In Pfeiffer syndrome the presence of cranial vault malformation and midface hypoplasia is accompa-

nied by broad and deviated thumbs and great toes.³⁷ Incomplete syndactyly may be present. Three types of Pfeiffer syndrome have been described.³⁸ In type I there are brachycephaly, midface hypoplasia, and finger and toe abnormalities associated with a good prognosis and normal intelligence. Type II will have pansynostosis of the skull (cloverleaf skull), severe exorbitism, hydrocephalus, and brain dysfunction. There is a high incidence of early death. In type III the cloverleaf skull is not present, but a combination of cranial suture synostosis, exorbitism, often hydrocephalus, and brain dysfunction will also lead to a poor outcome in many cases. The cause is mutations in the FGFR 1 and 2 genes. The condition is of autosomal dominant inheritance, affecting about 1 in 100 000 births.

The craniofacial surgeon will approach the treatment of the skull deformity according to the different synostoses found, and the surgeries often have to be repeated. When hydrocephalus is present, ventriculoperitoneal shunting is carried out. Midface advancement is timed when necessary for breathing, eating, eye exposure, or psychosocial reasons. Orthognathic surgery and final cosmetic procedures are postponed until the late teens. Because the phenotype of each patient is different, an individual treatment plan is always needed.

Apert syndrome

Apert syndrome or acrocephalosyndactyly type 1 is a rare syndrome characterized by brachycephaly, hypertelorism, slanting palpebral fissures, strabismus, midface hypoplasia, and complex syndactyly of hands and feet (Fig. 49a.6). There are two main mutations of the fibroblast growth factor receptor 2 with the resulting differences in phenotypes. Both are autosomal dominant transmitted. Syndactyly is present in digits and toes 2–4 or all fingers and toes, accompanied by restricted movements of joints such as elbows and shoulders. Cleft palate is seen in less than half of the patients. Learning difficulties of varying degrees are associated with this syndrome. In adolescence, the Apert patient often suffers severe acne and hyperhidrosis.

The brachycephaly present has been seen as a result of a bicoronal synostosis. Studies have, however, revealed a much more complex dysmorphology with a combined dysplasia of the cranial base and vault.

The craniofacial surgery treatment starts during the patient's first year.³⁹ The brachycephalic head shape can be improved by cranial vault remodeling. This is done using fronto-orbital advancement techniques,⁴⁰ the back of the head may also be advanced posteriorly to further normalize the sagittal length of the skull.²⁵ Upward growth of the skull may be surgically restricted. The author's preferred method is to address all this by fronto-orbital advancement and reshaping, parasagittal strip craniectomies and suturing, and finally posterior advancement of the back of



Fig. 49a.6 (a–c) Apert syndrome with brachycephaly and midface hypoplasia in a boy at 2 months of age. (d–g) Hands and feet with multiple syndactylies. (h–j) CT scan showing wide open fontanelles and sagittal suture, closed bicoronal sutures, and occipitoparietal beaten-copper appearance of the bone caused by pressure from the brain. (k–m) Same boy at postoperative follow-up after cranioplasty at age 1 year.

the head using springs. Cleft palate is repaired at 6 months, but can be postponed if breathing difficulties are present. Hand reconstruction is carried out before the end of the first year, aiming to achieve opposition of the thumb and an effective grip by the time the child is beginning to explore their surrounds with their hands. Foot problems may often require surgical intervention, but could be postponed until before the child starts to walk.

During childhood, as in Crouzon syndrome, breathing difficulties, eating problems, eye exposure, or major psychosocial problems may occur and lead to surgical intervention such as midface advancement and/or facial bipartition. Again, the results of childhood surgery do not often last and typically surgery has to be repeated later. Visual and hearing impairment often occur and need attention.

In adolescence, orthodontic treatment will precede orthognathic surgery as described in Chapter 49b. Forehead contouring will be needed in a significant number of patients to refine the shape of the forehead. Orbital surgery for exorbitism or hypertelorism is performed in selected cases. Cheek augmentation, rhinoplasty, and eyelid surgery are adjunctive procedures for a better end result. Medical treatment for

the severe acne in these patients is also important to avoid scarring.

Saethre–Chotzen syndrome

Saethre–Chotzen syndrome is an autosomal dominant syndrome associated with the TWIST genetic mutation, characterized by brachycephaly with uni- or bicoronal synostosis, depressed nasal bridge, low hairline, low set ears with prominent crurae, and S-shaped upper eyelid ptosis (Fig. 49a.7).⁴¹ Learning difficulties may be present. Patients have a partial syndactyly between the second and third fingers and toes. Skeletal deformities are frequent. The incidence is about 1 in 25 000–50 000,⁴² but the expression of the phenotype is quite variable.

The surgical treatment of Saethre–Chotzen syndrome will require cranial remodeling according to the individual deformity, as described for other craniosynostoses. The eyelid ptosis is corrected. Sometimes a nasal correction, lateral canthopexy, or division of syndactyly will help in normalizing the appearance of this syndrome. Hearing loss, visual impairment, and seizures will also need attention when present.⁴³

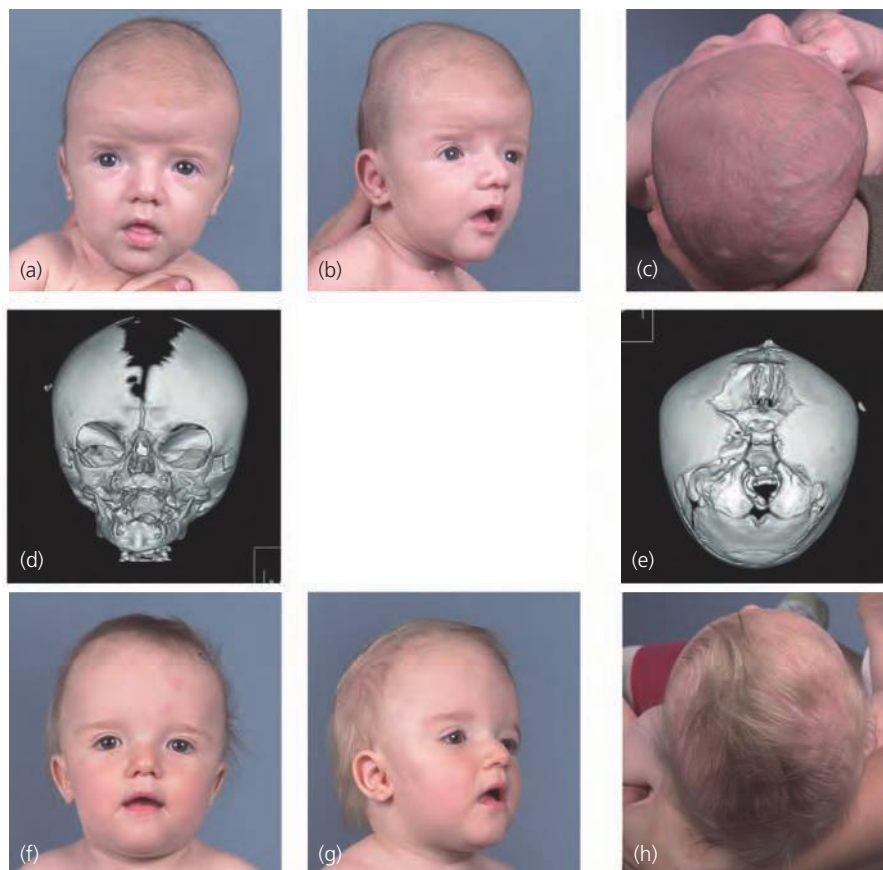


Fig 49a.7 (a–c) Saethre–Chotzen syndrome with bicoronal synostosis with the subsequent brachycephaly at 2 months. (d, e) CT scan showing bicoronal synostosis and wide open fontanelles. (f–h) More normal head shape at follow-up at 10 months postoperatively.

Carpenter syndrome

First described by Carpenter in 1901, Carpenter syndrome or acrocephalopolysyndactyly type II, is a rare autosomal recessive genetic disorder of the RAB23 gene,⁴⁴ characterized by premature closure of cranial sutures.⁴⁵ Affected individuals have a pointed head (acrocephaly) or a broad and wide one (brachycephaly). Craniosynostosis may affect the sagittal, coronal, and lambdoid sutures, and is often asymmetrical. Cerebral malformation may occur and there is a high risk for learning difficulties.⁴⁶ Facial malformations may include midface hypoplasia, malocclusion, down-slanting palpebral fissures, a flat nasal bridge, and dysplastic low-set ears. Fingers and toes are short, webbed, or fused, and there may be supernumerary ones (polydactyly). Other physical abnormalities may occur, such as congenital heart defects, umbilical hernia, cryptorchidism, or short stature.

Craniofacial surgical treatment aims at correcting the different malformations in the calvarium (craniosynostosis correction),⁴⁷ midface (hypoplasia and occlusive malformations), and eyelids.

Muenke syndrome

Muenke described 61 individuals affected by coronal craniosynostosis associated with a Pro250Arg mutation in the FGFR3 gene.⁴⁸ The phenotype shows a great variability in this population. Both uni- and bicoronal synostosis are seen (Fig. 49a.8).⁴⁹ Different skeletal abnormalities of the hands and feet have been reported, such as thimble-like middle phalanges, coned epiphyses, and carpal and tarsal fusions. These latter findings are subtle and of no functional relevance. A partial sensorineural hearing loss is common. Learning difficulties may be present in some cases.⁵⁰

Surgical results may be less rewarding in this population when compared to uni- or bicoronal craniosynostosis patients without this mutation, and repeated surgery is more often necessary.⁵¹

Treacher Collins syndrome

The above-mentioned syndromes are all characterized by craniosynostosis. This is not the case in

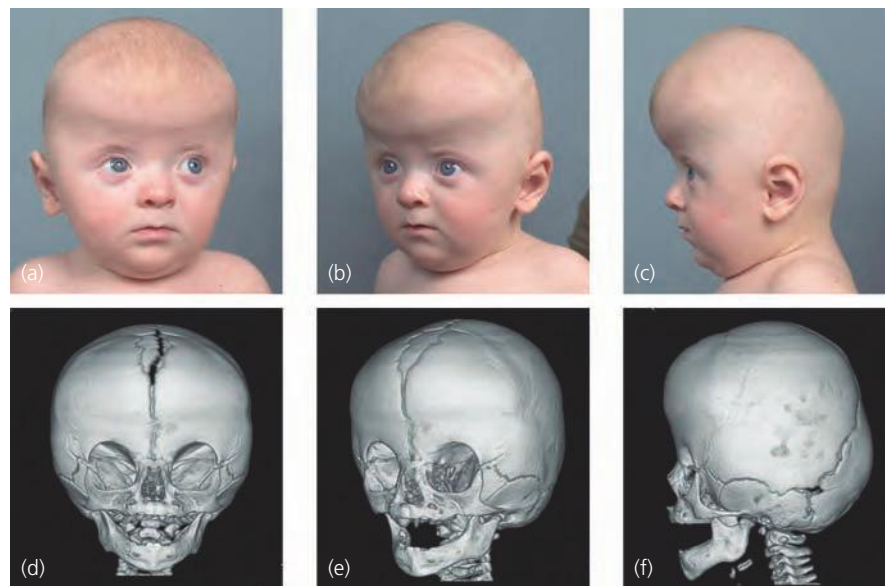


Fig. 49a.8 (a–c) A 5-year-old boy with Muenke syndrome and a deformed head shape. (d–f) Three-dimensional CT scan shows bicoronal synostosis.



Fig 49a.9 (a–c) Treacher Collins syndrome in a patient referred for additional reconstruction at 58 years of age, who has experienced return of the deformity by aging.

Treacher Collins syndrome (TCS), but it deserves mention due to the large amount of corrective craniofacial surgery that is required. The TCS patient has hypoplasia of facial structures with a convex profile of the face. The face will show downward slanting of the palpebral fissures, colobomata of the lower eyelids, hypoplasia of especially the zygoma and mandible, malformation of the external ear, middle ear malformation with hearing loss, malocclusion, abnormal hairline, and cleft palate (Fig. 49a.9).⁵²

Severely affected individuals will have a compromised airway at birth requiring a live-saving tracheostomy. The main reason for this is usually attributed to a severely retruded mandible, but the root cause may be much more complex. Closure of a cleft palate will have to be carefully planned and timed because of possible effects on the airway. Orthognathic surgery will be thoroughly addressed in Chapter 49b, so this text will focus on the other surgical areas involved.

The reconstruction of the external ear is a staged procedure using rib grafts and soft tissue coverage by different techniques as described by Brent and Nagata,^{53,54} or by a bone-anchored prosthesis. In a few cases, the middle ear deformities may be surgically correctable, but more often a bone-conducting hearing aid will be the solution for hearing improvement.

Nasal reconstruction will improve the broad nose, the dorsal hump, and loss of tip projection through a traditional rhinoplasty. Nasal surgery is done after completion of orthognathic procedures.

The lower lid coloboma and slanting palpebral fissures are difficult problems that are very hard to correct. Flaps from the upper eyelid, Z-plasties, skin transplants, and other methods have been tried with varying success.⁵⁵

Reconstruction of the zygoma deformity and lengthening of the lateral orbital wall can be achieved by a bicoronal incision using split calvarial bone grafts.⁵⁶ Lateral canthopexies are performed simultaneously.

Finally, additional soft tissue augmentation of the temporal fossa, periorbital area, or cheeks can be done using a dermal fat graft, fat transfer, or free tissue transfer.

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Chapter 49b

Craniofacial Syndrome Patients – Orthognathic Surgery

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The maxillofacial region is often affected in craniofacial syndrome patients. Treatment plans for these patients are made by the craniofacial team of specialists. Reconstruction of the maxillofacial region is most often performed after cessation of growth, although in urgent cases earlier treatment can be done. Orthognathic surgical techniques are very useful to correct malocclusion and malformations of the face. The most commonly appearing syndromes with severe effects on the maxillomandibular region are Crouzon syndrome, Apert syndrome, and Treacher Collins syndrome. Hemifacial microsomia is another developmental craniofacial disorder, which presents most frequently in a unilateral form but can also appear bilaterally. The diagnosis, treatment plan, and surgery to correct facial asymmetry in these patients are discussed in the chapter.

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Crouzon syndrome

This syndrome was first described by a French neurosurgeon in 1912. The characteristic presentation of the syndrome includes exorbitism and retrognathic max-

illary complex together with craniosynostosis. The frequency reported in the literature is about 1 case in 25000 births. The craniosynostosis usually affects both sides of the coronal sutures. This bilateral pattern gives a symmetrical appearance with a reduced anterior cranial fossa. Due to the shallow orbits, exorbitism is common and in some cases hypertelorism is also seen (Fig. 49b.1).^{1–5}

The genetic pattern is autosomal dominant inheritance. The syndrome is usually caused by mutations and newly formed gene expressions.⁶ In Crouzon syndrome the genes involved in development of osseous tissue are altered. Fifty percent of cases are spontaneous mutations and the rest are inherited. If one of the parent's genes are defective this is usually enough to transfer the disease. Although the expression of the disease may be very mild transferring the disease to a child is still a big risk.

The routine treatment in craniosynostosis patients is initial surgical treatment of the cranial bones as described in the Chapter 49a.

Maxillofacial characteristics

These patients usually have a small maxilla with a high palate and, to various extents, a constricted dental arch. Crowding of teeth is frequent but to a varying degree.⁷ Due to the maxillary hypoplasia the mandible looks prognathic, although cephalometrically it may be retrognathic. The difference in size between the maxillary and the mandibular dental arches will frequently cause a bilateral crossbite. The mental status in these patients is usually normal.

Orthodontic treatment

Orthodontic treatment usually begins 2–3 years before the orthognathic surgery. In demanding cases, however, with extreme crowding of the teeth in combination with a constricted upper dental arch, the orthodontic treatment may start earlier. A combined approach by orthodontists and maxillofacial surgeons will decide on the most suitable timing. Usually



Fig. 49b.1 (a) Patient with Crouzon syndrome after craniostylosis operation and orbital frame surgery. (b) The patient in lateral projection. (c) Lateral radiograph showing open bite, maxillary retrognathia, and tilted position of the mandible. (d) The occlusal situation. (e) Frontal view of the patient after bimaxillary surgery and genioplasty. (f) The patient in profile view. (g) Lateral radiograph of the patient 2.5 years postoperatively. (h) Occlusal situation 2.5 years after orthognathic surgery.

it is advisable to postpone the orthognathic surgery until the age of 16–18 years, with verified growth cessation demonstrated by radiographs of the hand showing growth plate closure.

The orthodontic treatment will vary in length depending on the dental occlusal situation and crowding of the teeth. The presurgical orthodontic treatment normally includes decompensation of teeth and lining of the dental arches. The treatment is always by fixed appliances and can take from 1.5–2.5 years to accomplish. Ideally the dental arches should be fitting optimally, preferably in a class I relation after surgical movements. If there are special psychological aspects involved the treatment can start earlier and surgery can also be performed earlier. The risk with an early interference is, of course, that continued growth will disturb the surgical–orthodontic result and a second operation will be needed later on.

Orthognathic surgery

The degree of maxillofacial deformity may also have an impact on the timing of surgical treatment, although it is usually advisable to postpone surgery until growth cessation. Depending on the degree of midfacial retrognathia, distraction osteogenesis can be the method of choice in demanding cases. Due to the varying expression of the syndrome it is difficult to make a routine analysis using standard cephalometric values. Every case has to be individually anal-

ysed and planned according to the clinical appearance and with regard to the orthognathic surgical methods available today.

In Crouzon syndrome there is a variation from very mild cases to very severe malformations, with the usual features being a small maxilla with a retrognathic position and a mandible of a normal size and in an orthognathic position, or sometimes also somewhat retrognathic. The chin point frequently has a flat appearance. The retrognathic position of the maxilla affects the whole midface although the base of the nose is in a normal position in relation to the infraorbital margins. If there is a pronounced exophthalmus, advancement of the infraorbital and supraorbital rims may be needed, with a so-called orbital frame operation (see Chapter 49a). When the midfacial retrognathia also affects the infraorbital margins and the base of the nose there is an indication for Le Fort III surgery with advancement of the midface, including the infraorbital margins, part of the zygoma, the base of the nose, and the maxillary complex. However, with the maxillary Le Fort III operation it is very difficult, not to say impossible, to advance the structures in a horizontal plane; it most likely becomes a rotation of the complex and it is impossible to correct the discrepancies between the upper and lower jaws. A combination of Le Fort III and Le Fort I in severe cases can be the solution of choice. Due to the relatively high risk for complications with the Le Fort III surgery an alternative is to do an orbital frame operation,

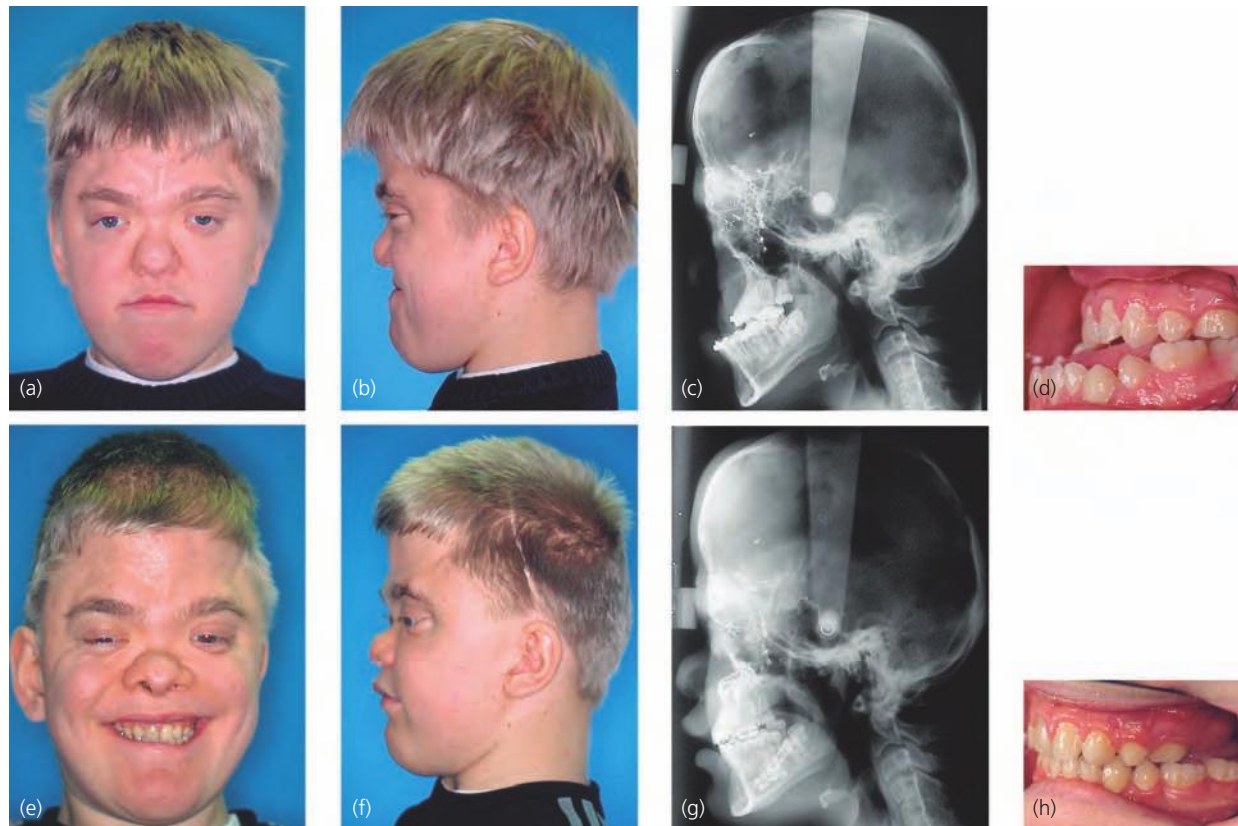


Fig. 49b.2 (a) Young male patient with Crouzon syndrome. Severe retrognathic position of the maxilla combined with a retrognathic mandible. (b) Occlusal situation after presurgical orthodontic treatment. (c) Lateral radiograph preoperatively. (d) Lateral view after surgical correction with large anterior movement of the maxilla and genioplasty. (e) Lateral radiograph postoperatively. Maxillary advancement of about 15mm. Genioplasty with chin implant. (f) Occlusal view postoperatively. (g) Lateral radiograph 5 years postoperatively.

and at a later stage continue with a Le Fort I osteotomy when the results of the orbital frame operations are clearly visible. The shallow bony orbits usually found in Crouzon syndrome patients limit the degree of orbital frame augmentation but it is still helpful in many patients.

The Le Fort I surgery, usually 6 months after the orbital frame operation, is carried out just below the infraorbital nerve and often extends into the malar process in order to increase midfacial projection. Since the maxilla mostly is smaller than the mandible, regeneration of the maxilla with widening of the dental arch is indicated (Figs 49b.2–49b.4). If the mandible has a cephalometrically and clinically acceptable sagittal position, the maxilla is advanced the whole distance to achieve a satisfying occlusal locking. If necessary chin advancement can be performed at the same time as the maxillary advancement. The desired direction of the chin advancement can easily be achieved by angling the sliding genioplasty to meet the esthetic demands. Even with extensive maxillo-facial advancements the postsurgical skeletal stability is satisfying. It is of utmost importance to achieve surgical advancement with the final position being a relaxed position without tension in the surrounding soft tissue (Figs 49b.1 and 49b.5).

Apert syndrome

Apert syndrome, also named acrocephalosyndactyly, was first described in 1906. A neurologist from France noticed these patients with certain characteristic signs including craniosynostosis, midfacial hypoplasia, and syndactylism of the hands and feet. The occurrence of the syndrome is about 1 case in 100 000 births. There is an autosomal dominant inheritance pattern in these patients. The coronal sutures are bilaterally affected with synostosis and a compressed anterior cranial fossa. The forehead is high, flat, and dominating (Fig. 49b.6).^{1–3,6} Due to disturbed development of the metopic and sagittal sutures the patient's head develops a bulging appearance. Hyperthyroidism and exorbitism are frequently involved in the syndrome.

Apert syndrome is a collection of malformations of the cranium and also the facial bones, hands, and feet. Furthermore, there are skeletal malformations, such as limited joint movements, balance difficulties, breathing problems, audiovisual problems, as well as problems with swallowing, speech, and chewing. Learning difficulties are frequently present. In 98% of patients with Apert syndrome there is a new mutation in the FFR-2 receptor gene in chromosome 10.

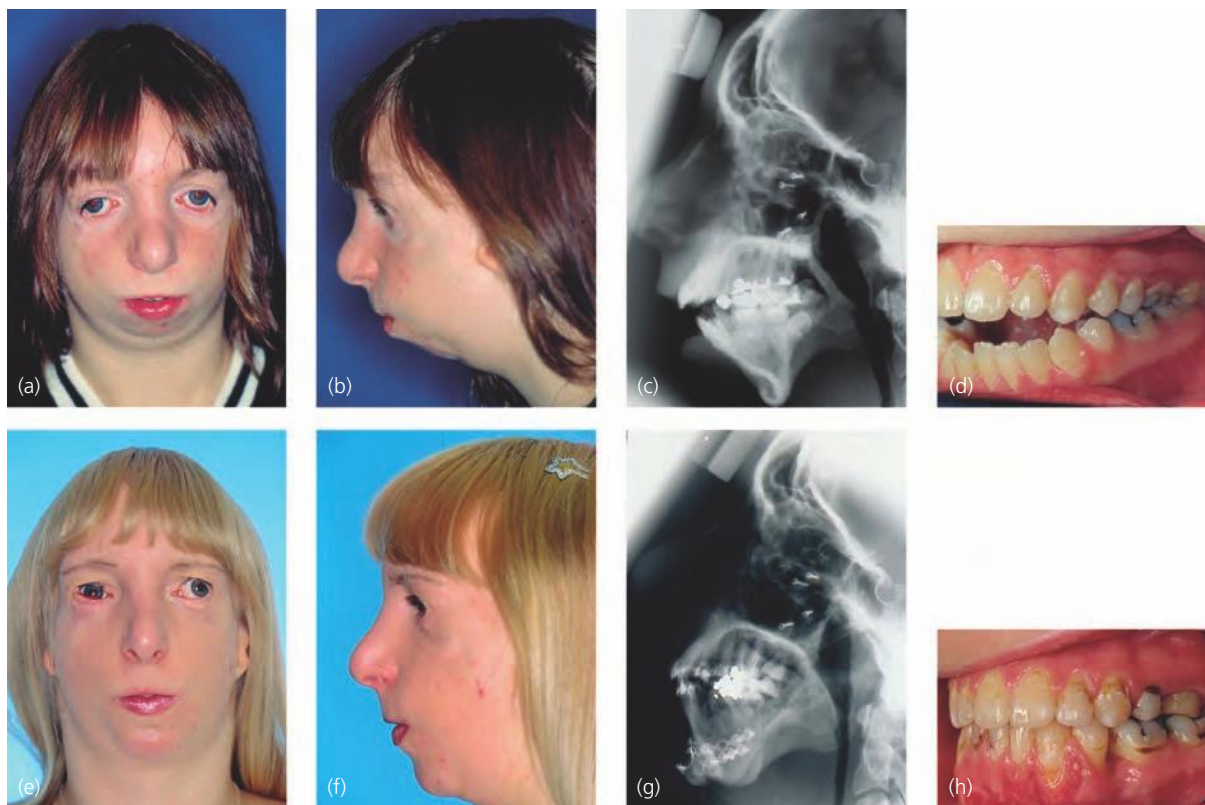


Fig. 49b.3 (a) Young female patient with Treacher Collins syndrome. (b) Clinical lateral view of the patient illustrating the small chin projection. (c) Lateral radiograph showing an open bite micrognathia without chin projection. (d) Occlusal situation preoperatively. (e) Clinical situation after orthognathic surgery. (f) Lateral view after bimaxillary surgery including genioplasty. (g) Lateral radiograph 2.5 years postoperatively. (h) Occlusal situation 2.5 years after orthognathic surgery.

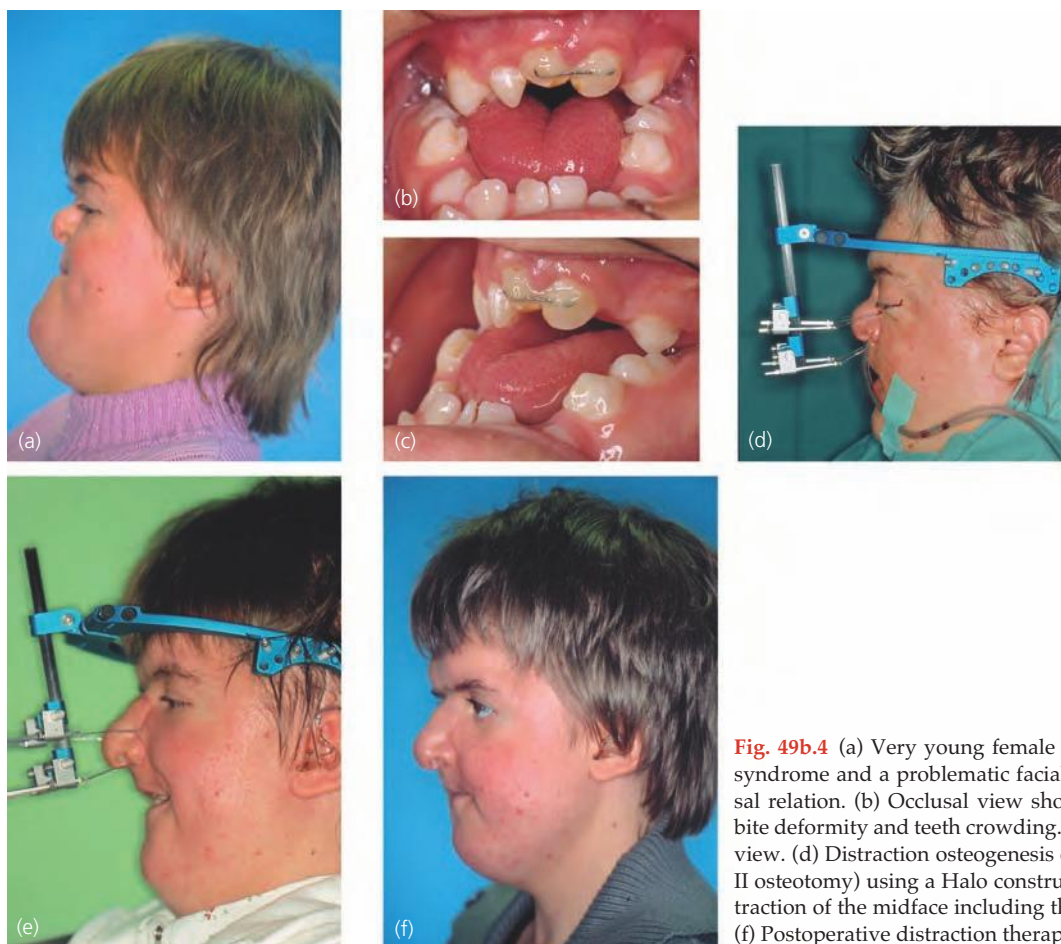


Fig. 49b.4 (a) Very young female patient with Apert syndrome and a problematic facial profile and occlusal relation. (b) Occlusal view showing a large open bite deformity and teeth crowding. (c) Lateral occlusal view. (d) Distraction osteogenesis of midface (Le Fort II osteotomy) using a Halo construction. (e) Final distraction of the midface including the base of the nose. (f) Postoperative distraction therapy.

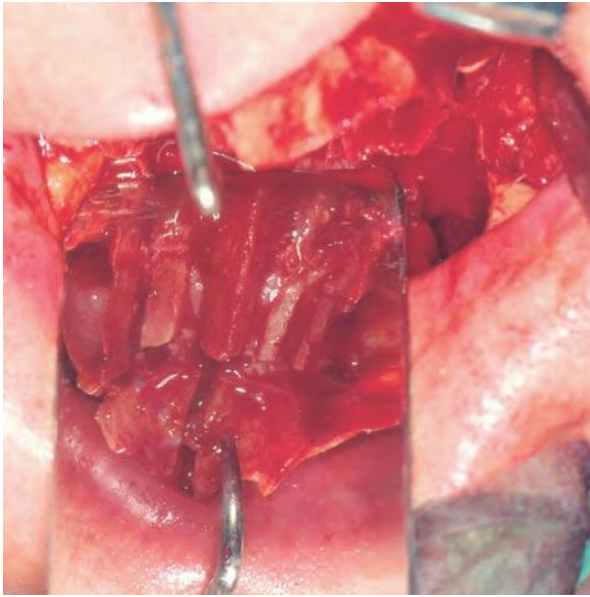


Fig. 49b.5 Clinical surgical view of six-piece segmentation of the maxilla.

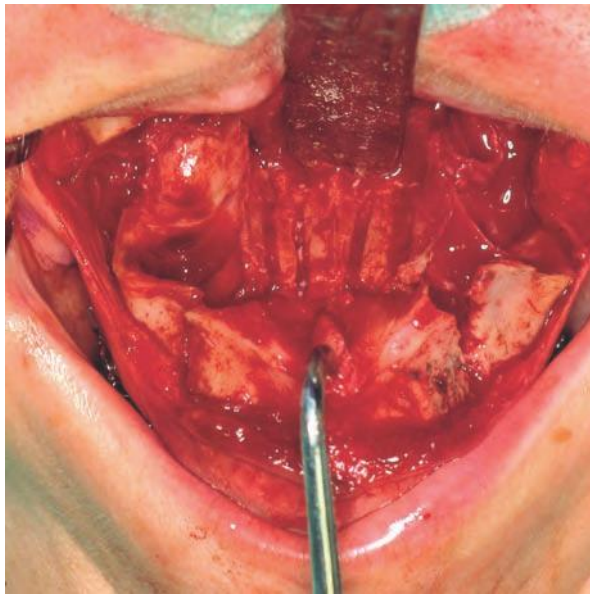


Fig. 49b.6 Surgical segmentation of the maxilla to enable transverse widening of the maxilla in seven pieces including osteotomy between central incisors.

The disease is inherited in an autosomal dominant pattern. However, there is a large variation in the degree of malformation between different patients (Fig. 49b.7).⁸

Maxillofacial characteristics

Apert syndrome patients have mostly a typical facial appearance with a rectangular-shaped head and a prominent lower face portion. The mandible is often prominent and cephalometrically prognathic while the midface is retruded and retrognathic in clinical and cephalometric aspects. The nose is frequently malformed and bulging, and in severe cases very

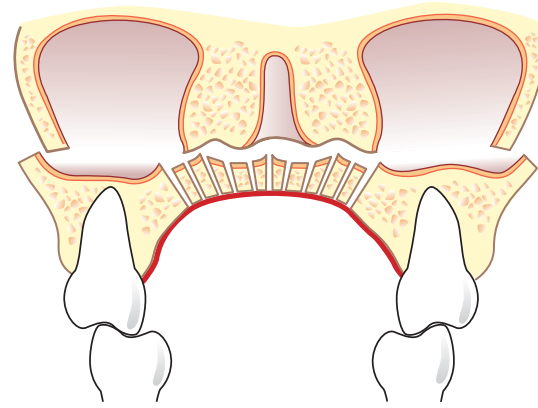


Fig. 49b.7 Schematic illustration of the segmentation procedure.

retruded with the nasal bone posterior to the glabella region.⁹

There is often difference in size between the upper and lower jaw, the maxilla being smaller and with a collapsed dental arch with a high positioned palate. The incidence of an open or submucous cleft palate is around 30%.⁷

The maxillary tuberosities are often hyperplastic and extend over the palate. Crowding of teeth is especially remarkable in the maxilla but often also occurs in the mandible. Cephalometrically and clinically there is a class III malocclusion, mostly in connection with an anterior open bite. The mandible can be truly prognathic but in many cases there is a pseudoprognathic situation due to the hypoplastic maxilla.

Orthodontic treatment

Apert syndrome patients need extensive orthodontic treatment before any orthognathic procedure can be carried out. As with all other orthognathic surgical patients, the ideal timing for surgery is after cessation of growth, normally when they are 16–18 years old. Growth indications, as shown in the hand radiographs with phalange closures, will decide the suitable time. However, as in Crouzon syndrome patients and all other severely malformed patients, earlier interference may be needed due to psychological indications.

Planning of surgery involves the surgeon and the orthodontist. Orthodontic treatment presurgically includes necessary extractions, lining of the dental arches, and decompensation if necessary. If the curve of Spee is unfavorable, segmentation of the orthodontic appliances can be chosen. The presurgical orthodontic treatment may last 1.5–2.5 years prior to the surgical treatment.

Orthognathic surgery

The most frequent orthognathic surgical approach to patients with Apert syndrome is a bimaxillary procedure with advancement of the maxilla through

a high level maxillary Le Fort I osteotomy combined with a setback of the mandible using a sagittal split operation.

In addition to the mandible being prognathic or pseudoprognathic there may be an unfavorable inclination of the anterior alveolar process with protrusion of the lower lip accentuating the prognathic appearance. In these cases it is advisable to add a segmental osteotomy procedure to the sagittal split. In many cases a chin plasty is also performed, either by sliding osteotomy or by use of chin implant material.

In the most severe cases, with retrusion of the maxilla and midface and also of the infraorbital margins and the nose, it is not possible to reconstruct the patient with a Le Fort I osteotomy; a Le Fort II surgical procedure is required, also involving part of the infraorbital margin and the base of the nose in order to achieve an adequate reconstruction. The Le Fort II procedure is difficult to advance manually to the extent that is needed; in these cases the advantage of distraction osteogenesis is clearly seen.

Distraction osteogenesis is achieved by use of a head frame and plates which are placed strategically in the maxilla, two on each side, one of them lateral to the nose and one in the infraorbital region. Wires are then attached to the plates and transferred through the soft tissue to the anterior part of the head frame allowing for a parallel distraction. The direction of distraction is of utmost importance to achieve an acceptable result. The distraction device will move the maxillary complex about 1 mm/day until a maximum of 30 mm is achieved. There is then a consolidation period of about 8 weeks. It is still difficult to obtain a good final result using only distraction in these cases.

In the end conventional orthognathic surgery has to be performed to get the best final result. The distraction procedure can begin at an early age aiming for an intermediate anterior positioning of the maxillary complex which will facilitate the final surgical procedure.

Normally, however, a bimaxillary orthognathic procedure is enough to get a satisfying result for dentofacial correction. Additional rhinoplasty and soft tissue augmentation as well as correction of irregularities in the forehead may be indicated (Figs 49b.6 and 49b.7).

Pfeiffer syndrome

This syndrome is similar to Apert syndrome. It was first described in 1964 and involves craniostosis like Apert syndrome. Characteristic features are very broad thumbs and broad great toes, and syndactyly of the hands is also sometimes seen. As in Apert syndrome there is an autosomal dominant inheritance pattern. The other craniofacial features are the same as for the Apert patients. However the degree of craniofacial disfiguration and the syndactyly in

Pfeiffer syndrome are milder and not so pronounced. These patients have normal mental development.

Saetre–Chotzen syndrome

Saetre–Chotzen syndrome was described in 1931. These patients have a similarity to Apert patients, although there is a marked facial asymmetry in Saetre–Chotzen patients.¹⁰ The symptoms include craniosynostosis, a low-set hair line, eyelid ptosis, partial syndactyly of the hands, and facial asymmetry. The syndrome has an autosomal dominant inheritance pattern like Apert syndrome. The synostosis affects the coronal suture bilaterally but in an asymmetric way which results in a plagiocephalic facial expression. The mental capacity in these patients is usually normal.

Treacher Collins syndrome

This syndrome is also called mandibulofacial dysostosis or Franceschetti–Klein syndrome and was first described in 1846.¹¹ Edward Treacher Collins reported on two cases in 1900.¹²

Treacher Collins syndrome is equally distributed among males and females. The incidence is about one in 10 000 births. Mild variations of the syndrome may not show up in statistics so the incidence rate is probably higher. The syndrome may vary quite a lot in expression.^{13–15} The cause of the disease is chromosomal changes due to mutation in chromosome 5 within q31–q33. Other genes are also affected, such as TCOF1, MSX2, FRABP, PRR1, and PRR2. The damage to the genes causes disturbances in the neural crest cells. In 50% of cases, the syndrome is a dominant inherited disease. In the remaining 50%, spontaneous mutations could be the underlying factor. The facial region and mouth develop from the first and second branchial arches. The ectomesenchyme from the neural crest is responsible for the development of cartilaginous, osseous, dental, and soft tissue structures of the facial skeleton. The disease also affects breathing due to constricted airways as well as external and internal ear, the zygoma arches, the eyes, the nose, and the jaws.¹⁶ The ear problems are both a hearing problem and an esthetic problem. These problems are handled by plastic surgeons and ENT doctors.

Maxillofacial characteristics

Treacher Collins syndrome patients have a very special appearance due to the abnormal anatomy of the mandible, the absence of zygoma, the slanting eyes, and disfiguration or loss of external ears. The mandible has a typical appearance with a short ascending ramus and a short mandibular body length. A typical finding in Treacher Collins syndrome is a concave antegonial notch of the mandibular angle. This curved

anatomy of the mandibular body together with the dental arch will, of course, give an unfavorable appearance of the curve of Spee.

The mandible is also retruded and retrognathic due to the short ramus length. Patients present with an open-bite deformity of varying magnitude. The mandible, although hypoplastic, normally contains all the essential structures. However, the condyle may be deformed or hypoplastic (Figs 49b.8 and 49b.9). The maxilla is often prominent in comparison to the lower third of the face. Although nasal breathing is compromised, the external nose is large and sometimes disfigured.

The occlusion is mostly angle class II malocclusion with anterior open bite. The palatal plane is rotated upwards in the posterior part resulting in an incorrect angle to the S-N plane and also in constriction of the choanae.

The short lower third of the face results in a steep mandibular plane which also affects the occlusal plane; together the result will be an anterior open bite with an abnormal curve of Spee.

The hypoplastic maxilla and retrognathic mandible in connection with varying degrees of coronal stenosis can complicate the upper airway space. Tracheostomy is sometimes indicated.

Orthodontic treatment

The presurgical orthodontic treatment will be started 1.5–2 years prior to the surgical date. The upper jaw teeth may be aligned in a conventional way although the curve of Spee in the mandible makes it difficult to achieve alignment of the dental arch. Segmental orthodontics may be a solution, treating the anterior part as one segment and the posterior as one right and one left segment. If the maxilla is to be surgically segmented, the orthodontic treatment is also carried out in a segmented manner.

Orthognathic surgery

The orthognathic surgical approach to these patients is difficult and it is not easy to achieve normalization of the face.

The surgical approach can be made in different ways.^{17–19} With regard to lengthening of the short ramus, osteodistraction is a technique which will increase the length of the ramus and at the same time advance the mandibular body and to some extent close the open bite deformity. The distraction can be done either extraorally or with intraoral devices. However, due to the negative curve of Spee and the curvature of the mandibular body it is difficult to achieve an optimal final result with this technique. Even after distraction osteogenesis, conventional orthognathic surgery has to be performed in order to obtain a good final occlusal result.

Another orthognathic surgical approach in the mandible is mandibular body osteotomy between the

first and second premolars or between the second premolar and the first molar depending on the curvature of the mandibular body. This osteotomy can be made in combination with myotomy of the suprahyoidal muscles, i.e. geniohyoideus and musculus digastricus, but not, of course, the genioglossus. The myotomy is made to facilitate lifting of the anterior segment of the mandibular body and straightening the curve of Spee and closing the open bite.

The mandibular procedure is often combined with a maxillary osteotomy with impaction and, if necessary, widening of the maxilla. Occasionally it is necessary to make a set-back of the maxilla by use of a segmentalized technique and removal of two premolars. Additional genioplasty is indicated in most cases.

Orthognathic surgery is preferably done after skeletal maturation at between 15 and 18 years of age.

A variety of surgical methods has been recommended in these cases, including bilateral sagittal split, osteotomy, sliding genioplasty, maxillary osteotomy, segmental osteotomy, and L-shaped osteotomy of the ramus.

Maxillary osteotomy with multisegmentation of the maxilla may correct the transverse occlusal discrepancy and may also correct the vertical height of the maxilla. However, posterior repositioning of the maxilla may be difficult, although it would help to balance the sagittal relation between the jaws. Segmental maxillary osteotomy with bilateral extraction of a premolar may be a solution in some cases.

In the author's clinic another strategy has been proposed, focusing on the hypoplastic and abnormal curvature of the mandible. Although mandibular body osteotomy is seldom done today in maxillofacial clinics, it may be a method of choice in these patients. A mandibular body osteotomy is performed using a step osteotomy in the form of a Z bone incision through the mandibular body but maintaining all the soft tissue vascular supply; it is usually made between the first molar and second premolar, depending on the case. The step osteotomy is made between the roots of the teeth and then parallel to the mandibular base above the mental foramen. If necessary, nerve transposition can be carried out. Just anterior to the mental foramen the cut goes down to and through the base of the mandible. By doing this osteotomy bilaterally the curve of Spee can be changed to a more normal appearance and the anterior open bite can also be closed. The mandibular body osteotomy may need to be combined with myotomy of the suprahyoidal muscles so that the anterior segment can be rotated to the planned position. Cortical bone grafts are positioned in the spaces left in the osteotomy areas after rotation of the anterior part of the mandible. Fixation of the mandibular body segments is achieved by use of 2.5 mm plates. Sometimes a reconstruction plate with a larger caliber may be indicated depending on the size of the anterior open bite.

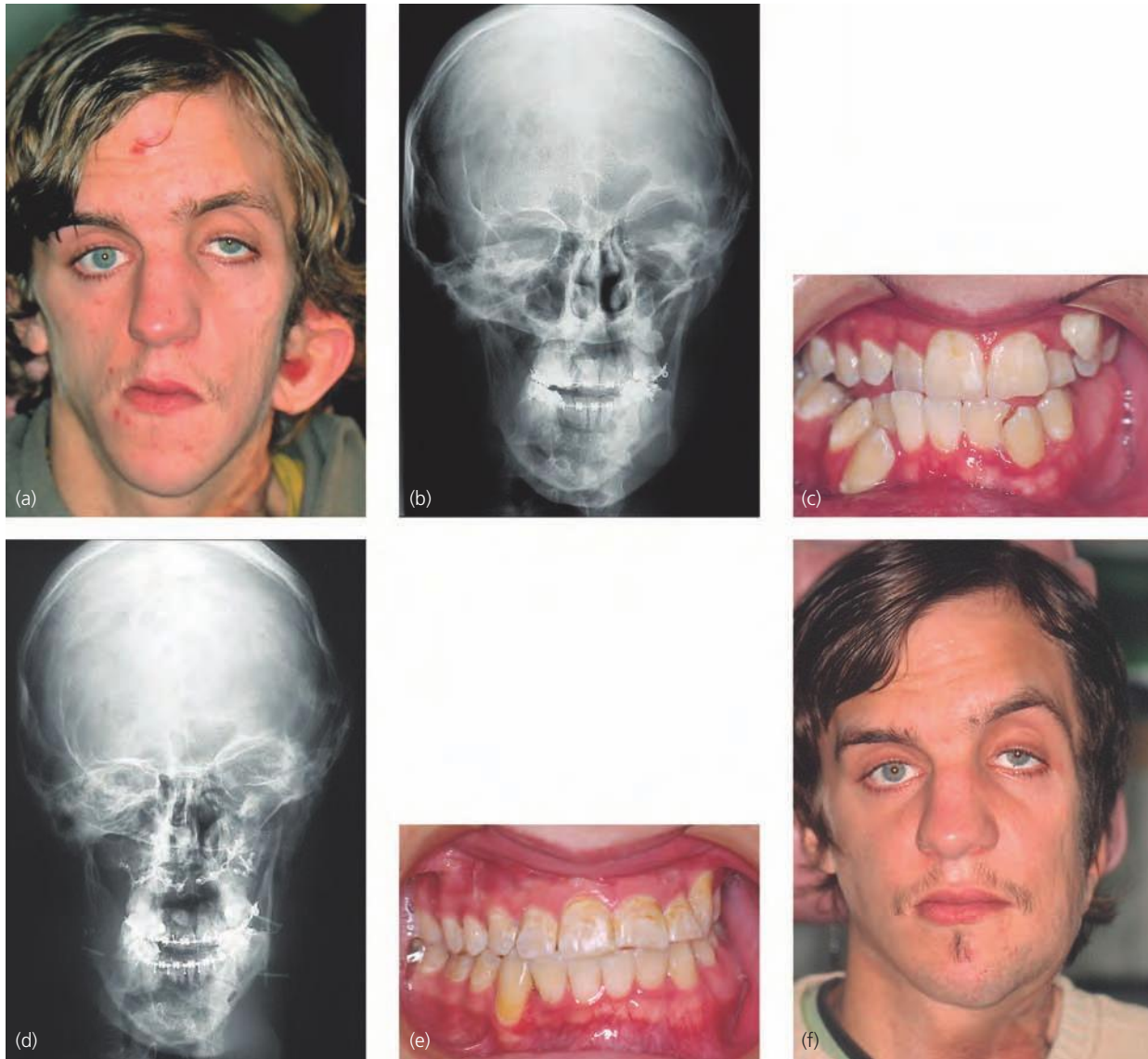


Fig. 49b.8 (a) Male patient with hemifacial microsomia preoperatively. (b) Frontal radiographic projection showing the facial asymmetry. (c) Preoperative occlusal view. (d) Frontal radiograph after surgical correction with bimaxillary osteotomy, genioplasty, and ramus augmentation. (e) Occlusal view after orthognathic surgery. (f) Clinical photograph 2.5 years after surgical orthodontic correction.

The mandibular body osteotomy can be combined with a sliding genioplasty in a direction to shorten the lower face region. The genioplasty can also be done as a second procedure after the mandible has healed for 6 months.

Maxillary osteotomy may or may not be necessary in these cases; it can correct the vertical position of the anterior teeth and correct some of the unfavorable anticlockwise rotation of the mandible.

With the above-mentioned orthognathic surgical methods it is possible to achieve an acceptable occlusion and functionally acceptable chewing. However, as has been described in the literature, Treacher Collins syndrome is one of the most challenging and difficult conditions to reconstruct. Despite numerous operations by a lot of specialists, optimal correction is impossible to achieve and patients still have the typical presentation of Treacher Collins syndrome. Although the symptoms associated with Treacher Collins syndrome are not easily removed, the patients

can still be functionally rehabilitated in a satisfactory way (Figs 49b.8 and 49b.9).

Augmentation and reconstruction of the zygoma region can be made either by use of autologous bone graft or implants of different kinds, like silicon or Teflon, or by use of lipid tissue graft. However, it is difficult to get a good long-term result in reshaping the contour of the zygomatic arches.

Attempts have been made for early surgical intervention by use of distraction osteogenesis in order to lengthen the ramus and improve the function and esthetics in childhood. However, no long-term results are yet available.

Other syndromes

Other more seldom appearing craniofacial syndromes like Moebius syndrome, Frazer syndrome, Klippel-Feil syndrome, Down syndrome, Goldenhar syn-

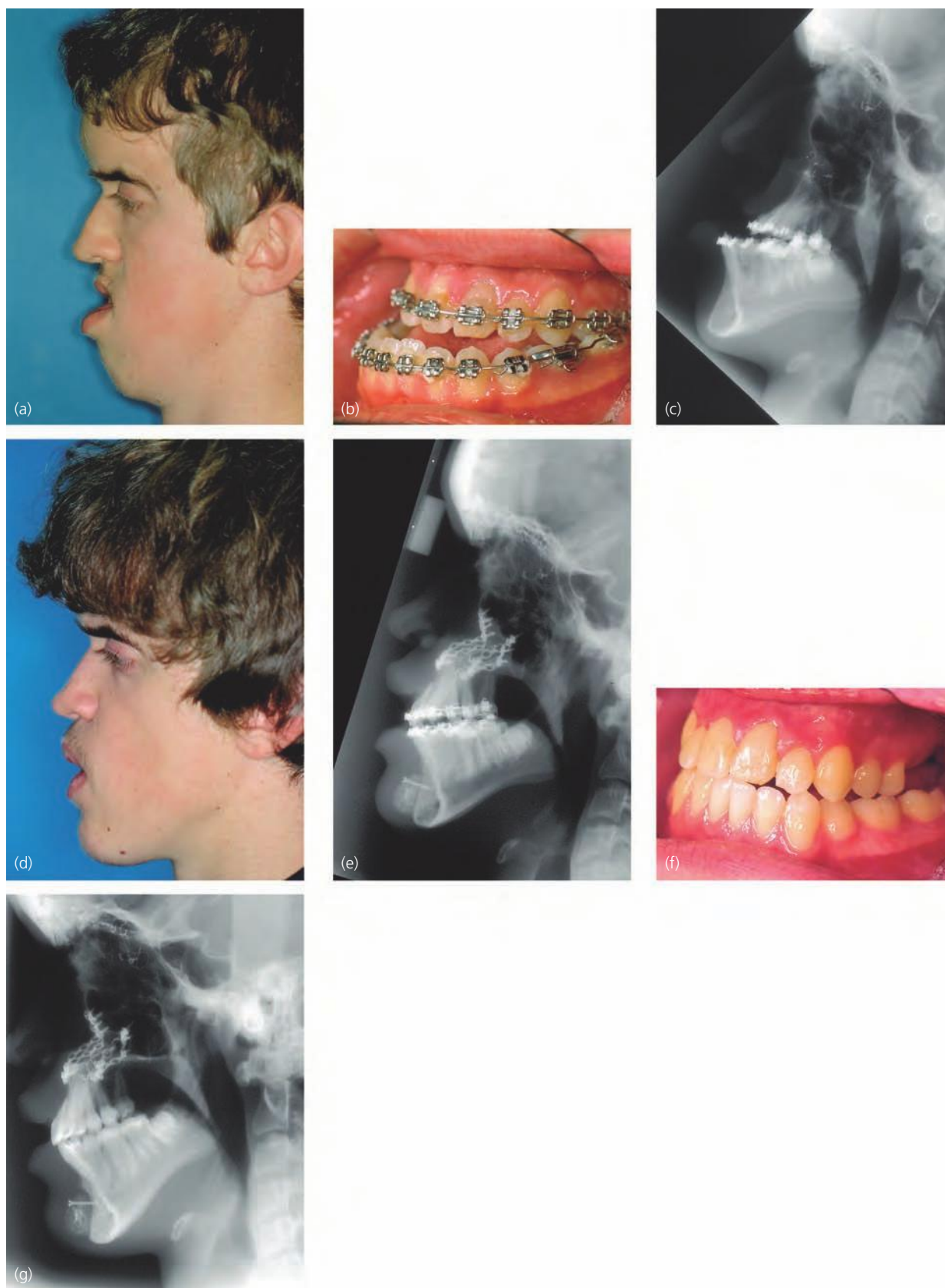


Fig. 49b.9 (a) Young male patient with Crouzon syndrome. Severe retrognathic position of the maxilla combined with a retrognathic mandible. (b) Occlusal situation after presurgical orthodontic treatment. (c) Lateral radiograph preoperatively. (d) Lateral view after surgical correction with large anterior movement of the maxilla and genioplasty. (e) Lateral radiograph postoperatively. Maxillary advancement of about 15mm. Genioplasty with chin implant. (f) Occlusal view postoperatively. (g) Lateral radiograph 5 years postoperatively.

drome with dentofacial deformities have to be individually planned and operated on with the orthognathic surgical arsenal available today.

Hemifacial microsomia

Introduction

Hemifacial microsomia presents with facial asymmetry of varying degrees depending on the severity of the disorder. This cranio-maxillofacial syndrome is an intrauterine defect affecting the branchial arch during pregnancy. The possible cause can be intrauterine bleeding or any kind of trauma and it is not an inherited disease. It most frequently affects only one side of the face and head but can occasionally present bilaterally. The expression of the syndrome is variable, depending on the severity of the deformity, from very mild with minor facial asymmetry and no effects on the ear, either external or internal, to very severe forms with loss of hearing, no external ear, no temporomandibular joint, and no mandibular ramus.^{20,21,22}

Maxillofacial characteristics

Depending on the degree of abnormality the surgical approach may vary considerably from total joint replacement and plastic surgical correction of the external ear to no surgery at all or just minor cosmetic corrections for very mild forms with all anatomical structures present.

The facial asymmetry also affects the maxilla; during the growth period it will adapt to the canted mandible and achieve a slanted position on the affected side. The occlusal plane will thus be more or less angulated horizontally towards the eye plane. In extreme cases the orbits may also be affected by the disturbed growth pattern and need surgical correction.

Orthodontic treatment

As always there is a team approach to these cases and the occlusal situation has to be evaluated before any surgical interference is made. Crowding of teeth and unfavorable inclination of the teeth are treated by lining of the dental arches and decompensation. Indicated extractions are carried out.

Orthognathic surgery

Bimaxillary surgery is indicated to level the maxilla and at the same time widen it if necessary in the transverse direction.

If the temporomandibular joint and ramus are missing, replacement will be made by costochondral rib graft and reconstruction of the fossa region with condroid tissue.^{21,23} The chondroid part of the rib

graft is trimmed and formed to act as the new joint. Part of the chondroid tissue on the rib can be used as fossa reconstruction material. The surgical procedure is preferably performed using an extraoral approach which facilitates the tissue handling.

Model surgery is necessary to get a wafer orienting the maxilla to the correct position in the vertical, horizontal, and transversal planes. Once the maxilla is positioned with miniplates and bone graft, preferably of cortical type, on the downtilted side, the mandibular osteotomy is performed and the rib graft attached.

The orthognathic surgical techniques used are the maxillary osteotomy with or without multisegmentation combined with sagittal split of the mandible, or, in special cases with a rudimentary ramus and angle of the mandible, an extraoral submandibular approach can be indicated to enable identification of the tiny anatomical structures (Fig. 49b.10).

In total joint and ramus replacement the surgical approach begins with the maxillary osteotomy to level and reposition the maxilla according to the cephalometric analysis and model surgery. Once the maxilla is positioned parallel to the eyes and in an acceptable position horizontally as well as transversely, the mandibular osteotomy can be started. It is of utmost importance that the vertical position of the maxilla achieves a normal relationship between the upper lip and frontal teeth. The upper jaw incisors should be visible 2–3 mm under a relaxed lip position. If necessary transverse widening of the maxilla can be done with the multisegmentation technique. The maxilla can also be rotated in order to get a slight augmentation on the affected side. A cortical bone graft is positioned in the gap between the maxilla and midface after repositioning of the jaw to prevent the maxilla from relapse.

It is not recommended to do both the maxilla and mandible at the same time because of the risk of getting disoriented (Fig. 49b.10).

For the costochondral rib graft reconstruction, a submandibular incision and a preauricular incision are made. After blunt dissection in the preauricular region a suitable fossa place is identified. The posterior part of the mandibular body is identified after blunt submandibular dissection.

Costochondral rib graft is obtained in a conventional manner (see Chapter 23) and the chondral part of the rib is modeled and shaped to be the new condyle. Chondral tissue from the rib can also be formed and modeled to be the fossa structure and attached to the planned fossa region to prevent ankylosis of the rib graft.

After maxillary repositioning and fixation with miniplates and the mandibular osteotomy is finalized on the non-affected side, the costochondral rib graft is placed in position after intermaxillary fixation and locking of the jaw.

The rib graft is attached to the mandibular body with plates and screws. Intermaxillary fixation is



Fig. 49b.10 (a) Male patient with Treacher Collins syndrome, frontal projection. (b) Lateral clinical view of the patient. (c) Occlusal situation with large open bite. (d) Lateral radiographic projection showing a large open bite and rotation of the mandible with the typical appearance for Treacher Collins syndrome. (e) Clinical view postoperatively after mandibular corpus osteotomy and genioplasty. (f) Clinical lateral view postoperatively. (g) Occlusal situation 2.5 years postoperatively. (h) Lateral radiograph 2.5 years after surgical correction.

maintained in these cases for 6 weeks. In cases with less severe abnormality, where there is still a functioning joint, bimaxillary procedures without joint replacement are carried out. Even if the ramus and joint are thin and tiny they may still function perfectly well.

Occlusal and functional correction can be achieved with the techniques mentioned. However, to improve the facial asymmetry both in the bony structures and the soft tissue, additional procedures like ramus implant, mandibular angle implant, and chin implants have to be carried out. The implants are available from manufacturers in different sizes. Bone graft material is not recommended due to the resorption tendency of bone graft without functional stimulation. Soft tissue augmentation using lipid tissue graft material can be done in indicated cases.

If the orbit is affected by the condition and has an abnormal position, an orbital frame operation with repositioning of the orbit may be indicated before all dentofacial reconstruction begins.

Bilateral microsomia cases, depending on the severity of the syndrome, have to be reconstructed with a bilateral costochondral rib graft if there is no functioning joint and ramus. If there are rudimentary joint structures with satisfactory function, bimaxillary procedures can be carried out with combination of maxillary osteotomy and bilateral ramus osteotomies. Additional augmentation procedures and genioplasty have to be performed to improve the facial contour.

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Chapter 50

Reconstruction of Maxillary Defects

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The chapter is an overview of treatment options for maxillary reconstruction. Classification of maxillary defects depends on the extent of the defect, regarding both the hard and soft tissue, and is the basis for the choice of which surgical technique to use. The treatment can vary from local flaps or pedicle flaps to a composite free tissue transfer with bone and a generous amount of soft tissue. Implant-supported overdentures seem to be the most predictable restoration but zygoma implants also have a place in the reconstruction of maxillary defects. Different flaps for use in surgical reconstruction are presented in detail.

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Introduction

The maxilla occupies a central location in the facial skeleton unifying the orbits, zygomaticomaxillary complex, nasal unit, and stomatognathic complex into a single functional and esthetic unit.¹ The comprehensive reconstruction of maxillary defects following trauma or tumor-ablative surgery is a complex process requiring the collaborative efforts of different medical and dental specialists, skilled in restoring missing skeletal and soft tissues, a vascularized graft bed, and the reconstruction of dental and ocular structures. Wound characteristics and patient factors help determine the most appropriate management strategy and among the more salient considerations are defect size, type of missing tissue, ability to withstand prolonged surgery, availability of donor tissue, and the expertise of the management team. Other broader issues which must also be considered include cost of rehabilitation and patient prognosis, and these require evaluation of a complex array of social, professional, economic, and emotional factors.

Historically, and still in many centers, removable dental prostheses are felt to be superior to flaps or grafts for the reconstruction of maxillary defects. Prosthetic devices are capable of restoring missing skeletal, soft tissue, and dental elements in a single procedure and do not require multiple revision surgeries to achieve function and esthetic harmony with the remaining structures. Concerns over surveillance of the tumor site allowing prompt management of recurrent disease also mitigate in favor of a removable prosthesis, but, notably, this advantage has not been shown to influence survival after resection of squamous carcinoma or high-grade salivary cancer. Furthermore, the use of advanced imaging techniques such as magnetic resonance imaging (MRI) or positron emission tomography/computed tomography (PET/CT) and the ability to visualize remote or closed sites with flexible endoscopic procedures, provide sensitive and accurate means for postablation surveillance. In addition, refinements in vascularized tissue transfer techniques and advances in dental implant technology have addressed some of the limitations to

reconstruction of the maxilla with biological tissue, and have provided patients with significant speech, masticatory, and social benefits derived from permanent obturation of their maxillary defect.

Classification and treatment approaches for maxillary defects

Classification of maxillary defects

Numerous classification systems for maxillary defects have been proposed, including those by Ohngren (1933), Aramany (1978), Spiro (1997), Davison (1998), Brown (1996), Cordeiro (2000), and Okay (2001). The more recent classifications attempt to define the anatomical extent of each defect and provide recommendations for different reconstructive approaches, based on experience. Since traumatic defects do not follow surgical principles, their characteristics and efforts to classify them are random. In this chapter, two commonly used systems will be employed, one developed by Okay, a prosthodontist, and the other by Brown, a surgeon, as foundations for the discussion.

Okay classification

The classification system proposed by Okay takes into consideration both surgical and prosthetic tech-

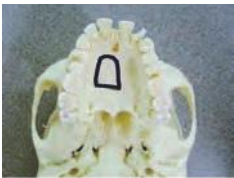
niques for maxillary reconstruction. It addresses important reconstructive goals, such as closure of oronasal communications, the provision of a stable foundation for dental restoration, reconstruction of midface symmetry, and stabilization of orbital contents. This system divides maxillary defects into three categories based on the presence or absence of dental structures that can be used for the retention of prosthetic devices (Table 50.1).

Class Ia defects involve the hard palate, but not the tooth-bearing alveolus (Fig. 50.1). Defects of the maxilla and tooth-bearing areas posterior to the



Fig. 50.1 Okay class Ia: defect resulting from previous biopsy attempt.

Table 50.1 Okay classification of maxillary defects.

Classification	Anatomy	Surgical reconstruction	Prosthetic reconstruction	
Ia	Involves hard palate but not tooth-bearing alveolus	Soft tissue graft	Maxillary obturator	
Ib	Involves any portion of the maxilla posterior to the canines or the premaxilla	Soft tissue graft	Maxillary obturator	
II	Involves any portion of the tooth-bearing maxilla but only one canine, or transverse palatotomy less than 50% of the palate	Vascularized bone-containing free flap	Maxillary obturator	
III	Involves any portion of the tooth-bearing maxillary alveolus, includes palatotomy that involves more than half of the palate surface	Vascularized bone-containing free flap	Implant-supported prosthesis	

canines, or isolated defects of the premaxilla are classified as class Ib (Fig. 50.2). A class II defect involves up to half the maxilla including the alveolus and premaxilla and includes the ipsilateral canine if present (Fig. 50.3). Anterior transverse palatotomy defects measuring less than one half of the palatal surface are included in class II. Class III defects are more extensive and involve more than half of the maxilla, the associated alveolar bone, and both canines. Total palatotomy defects and anterior transverse palatotomy defects involving more than half of the palate are classified as class III defects (Fig. 50.4). Subclass F defects involve the inferior orbital rim, and defects that involve the body of the zygoma are in the subclass Z.²

Brown classification

This system considers the involvement of structures in both vertical and horizontal planes to classify different maxillary defects.³ It begins with identification of the vertical defect (class 1–4) followed by description of the horizontal extent (A, B, C), representing absent midline structures such as the nasal septum and the contralateral maxilla (Table 50.2). This system provides a means to assess the most likely functional outcome for a patient and, like the Okay classifica-

tion, outlines the reconstructive requirements for each type of defect.

Vertical components are classified as:

- Class 1: maxillectomy with no oro-antral fistula. Class 1 defects involve the hard palate, but not the tooth-bearing alveolus (Fig. 50.1).
- Class 2: low maxillectomy.
- Class 3: high maxillectomy.
- Class 4: radical maxillectomy.

Horizontal components are classified as:

- A: unilateral alveolar maxilla and hard palate resected. Less than or equal to half the alveolar and hard palate resection not involving the nasal septum or crossing the midline.
- B: bilateral alveolar maxilla and hard palate resected. Includes a small resection that crosses the midline of the alveolar bone including the nasal septum.
- C: removal of the entire alveolar maxilla and hard palate.

Maxillary defect reconstruction

Brown class 1 defects can be treated with local flaps, or with an obturator if there is an oro-antral or oronasal communication. Similarly, class 2a defects can be obturated, or reconstructed with local and pedicle flaps, or a combination of both. As the horizontal extent increases, such as in classes 2b and 2c, prosthetic rehabilitation with an obturator becomes more difficult and implant support may become necessary to improve stability. Consequently, a composite flap may be used to assist with soft tissue closure and provide bone for implant placement. Class 3 defects, involving midline and cross-midline structures, are usually not treated with obturators, because the weight and size of a device presents challenges in achieving a functional and stable prosthesis. For Classes 3b and 3c defects, a composite vascularized flap capable of addressing both maxillary and orbital defects is the recommended treatment. Class 4 defects



Fig. 50.2 Okay class Ib: defect resulting from excision of a pleomorphic adenoma.



Fig. 50.3 Okay class II: defect resulting from resection of an adenoid cystic carcinoma.

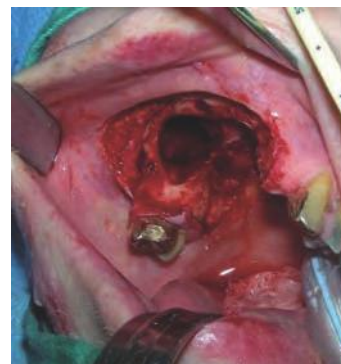


Fig. 50.4 Okay class III: defect resulting from resection of a squamous cell carcinoma.

Table 50.2 Summary of recommended treatment course for different maxillary defect classifications. (FF = vascular free flap; CFF = vascular composite free flap; OA = oro-antral fistula).

Classification of maxillary defects	Horizontal extent →	1 Palatal defect that does not involve the tooth-bearing alveolus	A Unilateral maxilla and palatal defect less than or equal to half of palate and alveolus, not involving the nasal septum	B Defect that crosses the midline, includes the nasal septum	C Defect involves the entire maxilla and alveolar bone
Vertical Extent ↓					
1 Removal of alveolar bone does not involve the nasal cavity or the maxillary sinus		Obturator, local or pedicle flap reconstruction required for palatal defect with nasal or antral involvement FF for larger defect			
2 Maxillectomy involving the nasal cavity and the maxillary sinus			Obturator Local or pedicle flap CFF	Obturator with implant support CFF	Large CFF required to close OA fistula, obturate orbit, and possibly close a cranial communication Prosthetic rehabilitation requires implant support
3 Maxillectomy involving the maxillary sinus, the nasal cavity, the orbital floor, with or without involvement of the anterior skull base			Obturator use possible but difficult with increased defect size and presence of trismus CFF	CFF Prosthetic rehabilitation requires implant support	Large CFF required to close OA fistula, obturate orbit, and possibly close a cranial communication Prosthetic rehabilitation requires implant support
4 Maxillectomy involving the maxillary sinus, the nasal cavity, exenteration of the globe with or without involvement of the anterior skull base			Large CFF required to close OA fistula, obturate orbit, and possibly close a cranial communication Prosthetic rehabilitation requires implant support	CFF Prosthetic rehabilitation requires implant support	Large CFF required to close OA fistula, obturate orbit, and possibly close a cranial communication Prosthetic rehabilitation requires implant support

are almost never amenable to obturator rehabilitation without free flap reconstruction, as they involve the entire orbit and sometimes the cranial base. A composite free tissue transfer that provides not only bone, but also a generous amount of soft tissue for closure of fistulas, obturation of the orbit, and sealing of cranial communications is needed.

Prosthetic obturation of maxillary defects

Staging of obturator fabrication

The postmaxillectomy defect undergoes significant changes as the tissues heal and remodel. Multiple

devices and modifications are required to adjust the rigid margins of the obturator to conform to the changing contours of the defect. The surgical obturator is the initial prosthesis used at surgery (Fig. 50.5a). This prosthesis addresses multiple factors to help the patient in the immediate postoperative period. The device restores palatal contours aiding speech and deglutition, provides support for surgical packing used to retain the skin or skin-substitute graft in place, and reduces wound contamination from the oral cavity. It can be wired to the remaining teeth or screwed to adjacent bone and is usually removed within 10–14 days to be replaced with an interim obturator. Generally, the surgical obturator is fabricated from acrylic, similar to a denture base plate, and does not contain teeth unless the esthetic zone is involved.

The interim obturator is inserted immediately after the surgical obturator is removed (Fig. 50.5b). This second prosthesis is used until healing is completed

and addresses both functional and esthetic needs. An acrylic base plate with teeth, clasps, and soft tissue liner is used to cover the surgical defect. Through close adaptation between the obturator and defect surfaces, retention is improved and fluid leakage into the nasal cavity is reduced, while optimizing speech and swallowing. The lining material is changed frequently to compensate for contour changes as the soft tissue margins contract and the bone margins remodel.

The definitive obturator is the final prosthesis, which is fabricated after tumor eradication has been confirmed and treatment and healing are complete, usually 6–12 months after ablative surgery (Fig. 50.6). Problems with comfort, function, retention, stability, and esthetics associated with the interim prosthesis can be addressed during design of the definitive obturator. Implant support to improve retention and stability is an option if bone is available in the appropriate sites.⁴

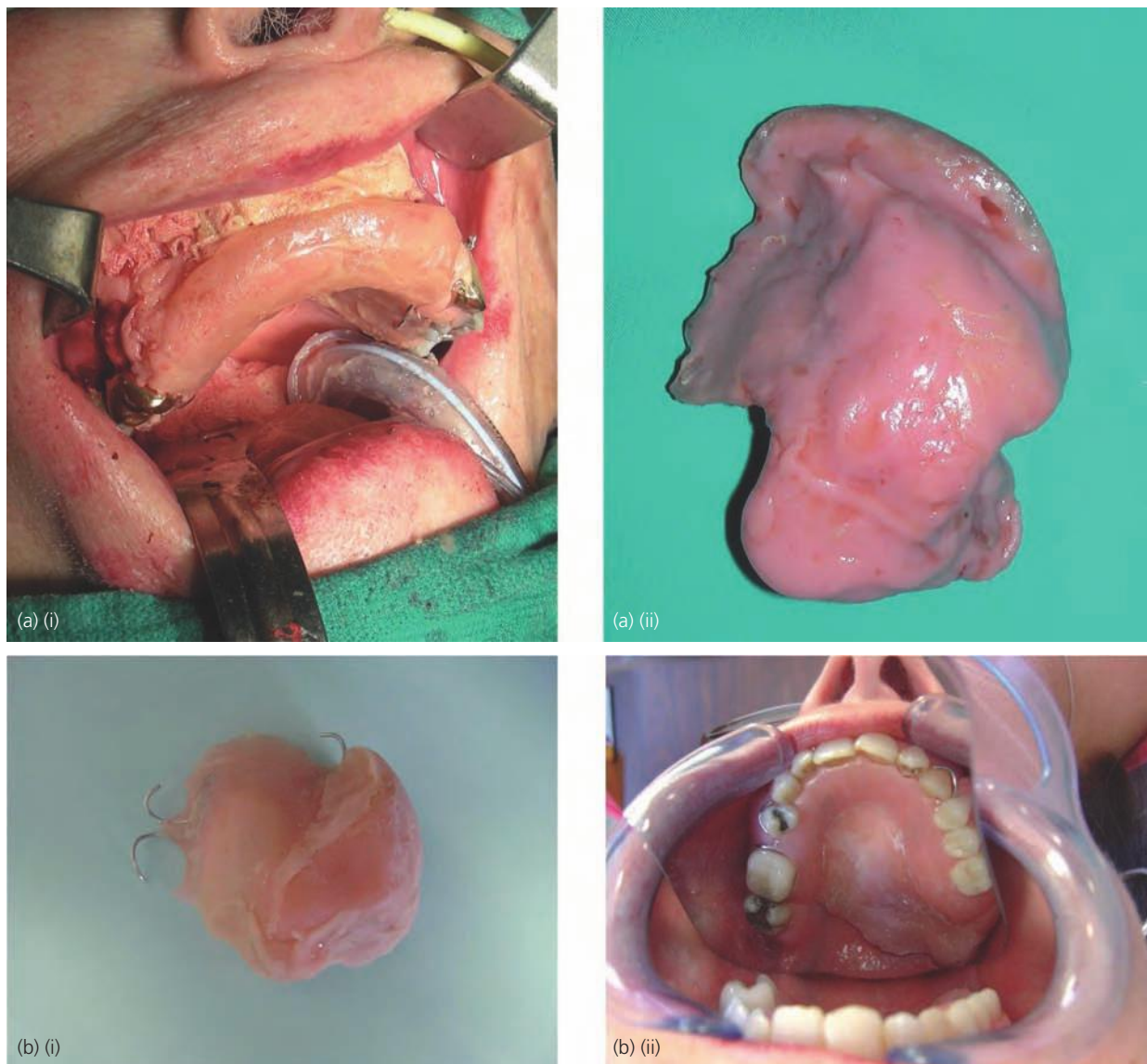


Fig. 50.5 (a) Immediate obturation of defect with surgical obturator. (b) Temporary obturation with an interim obturator.



Fig. 50.6 Definitive obturator

Facilitating obturator retention and stability

Removable obturators rely entirely on their physical characteristics for retention and stability. When reconstruction follows surgical ablation of pathology, the outcome for prosthetic rehabilitation can be improved significantly by recognizing, preserving, or enhancing those defect margins affecting retention. Mucosal coverage of the bony margins of the defect can be accomplished by placing the incision at least 0.5 cm beyond the planned osteotomy, thereby retaining a cuff of soft tissue that can be folded over the sectioned bone. Ulceration through this marginal tissue can be prevented by smoothing the bony edges before closure. Medial retention of the prosthesis is provided by engaging undercuts along the remaining maxillary bone, palatal shelf, or nasal floor. If the inferior turbinate encroaches into the defect, it should be removed since the associated undercut will most likely interfere with the paths of insertion and withdrawal. Lateral retention is provided by creating a mucosal scar band in the cheek along the margin between the cheek incision and a lining split-thickness skin graft (STSG). When possible, positioning this scar band high in the defect is preferable to placing it below the level of the medial undercut. Retention can also be aided by the addition of clasps around remaining teeth. The cuspids provide valuable anterior retention points, hence their inclusion in the Okay classification as a stratification measure. In order to preserve the periodontal health of remaining teeth, all efforts should be made to leave adequate bone support around the tooth adjacent to the planned osteotomy. This objective may require extraction of a tooth and placing the osteotomy through the dental socket.

Regardless of the attachment system used (marginal undercuts, tooth clasps, implants), reducing detrimental loading or displacement forces by enhancing obturator stability can prevent premature failure of the retention devices. Vertical stabilization and resistance to masticatory forces can be achieved

through preservation of the premaxilla when possible, and the use of overdentures with palatal coverage.⁵ Full extension of the obturator into all the recesses of the defect also provides load support and improved retention. Instability of an obturator is not only the result of masticatory loads. Displacement forces can also be created by speech and swallowing. When resection of more than one half of the soft palate is indicated, removing the entire velum should be considered, because this will augment retention, improve speech, and reduce fluid leakage. Large obturators that extend beyond the area of the maxillary tuberosity may be displaced during mandibular excursions by interference from the coronoid processes. A coronoidectomy is a simple remedy to this potential problem and may also prevent the development of trismus from postradiation fibrosis of the temporalis insertion.

Soft tissue coverage of defects

Using a split-thickness skin graft (STSG) enhances the retention and the stability of the prosthesis. A skin graft is more resistant to abrasion than respiratory mucosa and more suitable for prosthesis bearing. As the graft heals, it undergoes contraction, creating an undercut that can be engaged to improve the retention and stability of a prosthesis.⁶ Replacement of the respiratory mucosa with skin also improves hygiene by reducing secretions produced by glands normally present in respiratory tissue. STSGs are laid into defects with the dermal surface against the bone and secured in place for 2–3 weeks with compressive dressings composed of surgical packing or molded impression material (Fig. 50.7a). The packing is retained with a splint or bolster sutures placed along the margins and tied over the dressing. A successful skin graft will provide epithelial cover for all margins and surfaces of the resection (Fig. 50.7b, c).

Alternatives to autogenous skin include commercially available allogeneic, acellular dermal matrices. These have been used in a similar manner to STSG and offer several advantages, including reduced scarring and contraction and lack of donor site morbidity.⁷ However, the lack of contraction of allografts may also reduce the retention of the obturator. Additionally, the non-keratinized surface of dermal allografts is less resistant to friction created by movement of a prosthesis. The authors have observed an interesting fibroproliferative response in their use of allogeneic dermal grafts placed in maxillary defects. Over a period of several months, the grafts appear to stimulate the development of a significant amount of fibrous tissue that can bridge the entire defect (Fig. 50.8). The surface of the tissue is covered with epithelium, presumably regenerated from adjacent mucosal margins or from epithelial squames present in saliva. Obturation of a defect from an inlayed graft offers the advantage of closure of oronasal or oro-antral communications without the morbidity associ-

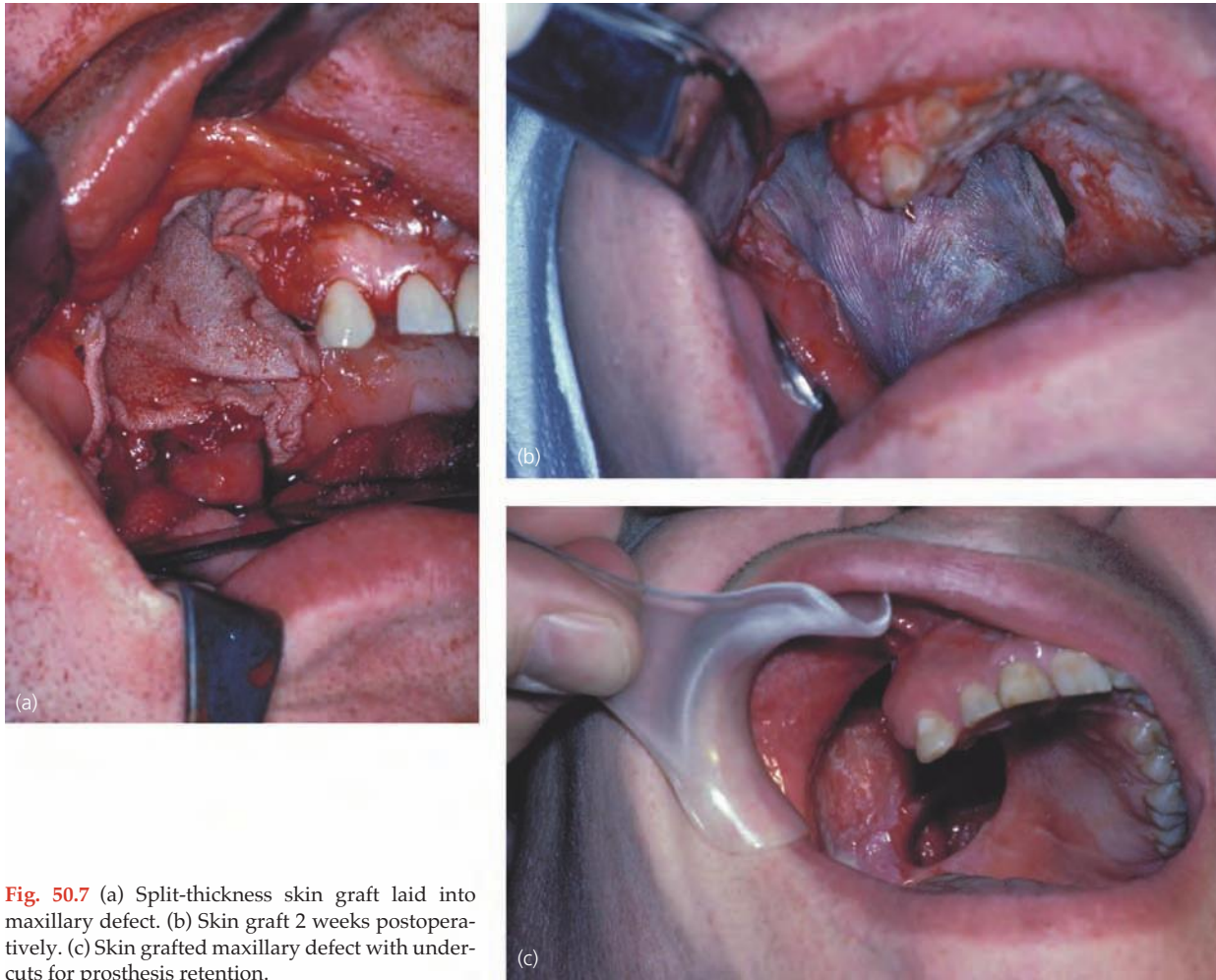


Fig. 50.7 (a) Split-thickness skin graft laid into maxillary defect. (b) Skin graft 2 weeks postoperatively. (c) Skin grafted maxillary defect with undercuts for prosthesis retention.

ated with other methods of soft tissue reconstruction, such as a rotational flap or free tissue transfer. However, prosthetic challenges are created whenever a maxillary defect is filled with soft tissue, whether from flaps or a local regenerative response; these will be discussed later in the chapter.

Implant support

Additional measures to improve the retention of an obturator include the use of dental implants. Advances in both implant and abutment technology have significantly improved outcomes. Shorter and wider implants can be accommodated within non-traditional sites in the remaining midface skeleton and have been shown to provide stability similar to longer implants through modifications to the implant surfaces (Fig. 50.9). The increase in survival rate of these shorter implants (8 mm or less) is the result of changes in surface geometry that increase surface area and porosity.⁸ Simultaneous advances in prosthetic implant attachments have also allowed less than ideally placed implants to be used as support for removable prosthetic devices. Locator attachments allow up to 40 degrees of divergence from the path of withdrawal along the implant axis. Custom abutments and cast bars with attachments can also be



Fig. 50.8 Fibroproliferative response in hemimaxillectomy defect following application of allogeneic acellular dermal graft. Proliferation of fibrous tissue has nearly filled the defect.

used to improve obturator retention. For those patients with defects amenable to placement of implants, prosthetic restoration is significantly improved from both esthetic and functional perspectives.

Adherence to sound prosthodontic principles and appreciation of an individual patient's entire condition, can lead to more successful outcome. In patients with malignant disease, the effect of adjunctive therapy, such as chemo-radiation-induced xerostomia,

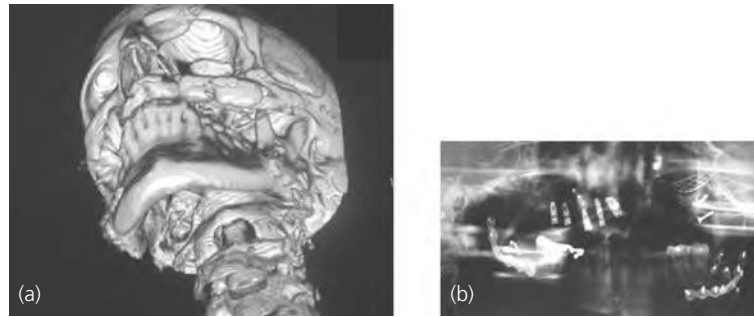


Fig. 50.9 (a) Left maxillary defect reconstructed with vascularized fibula graft. (b) Short implants used to support an overdenture. (Courtesy of George Suchko, DDS.)

mucositis, or trismus, must be taken into consideration. Difficulty in cleaning and the potential for caries mitigate against fixed restorations such as crowns and bridges. While the most predictable restoration is an implant-supported overdenture, mucosal inflammation or xerostomia may preclude its use, and trismus may affect a patient's ability to insert the prosthesis. However, when used, the palatal coverage provided by overdentures reduces detrimental forces on implants that can lead to failure.⁵

The zygoma implant

This implant was originally developed for use in a severely atrophic maxilla to provide posterior retention without additional bone graft augmentation. For this application, the zygoma (or zygomatic) implant has proven reliable even when loaded immediately.⁹ Additional data to support use of this system were provided by Kahnberg *et al.*, who identified a high survival rate and patient acceptability of restorations over a 3-year period.¹⁰

Stability of the zygoma implant system relies on three points of fixation. Bilateral posterior support is achieved with implants placed through the zygomas and anterior support is provided by two endosteal implants placed in the anterior maxilla. This tripod configuration promotes cross-arch stability by linking the separate components together and providing retention to the principal displacement forces created by mastication.

Surgical placement involves the insertion of a long implant from the palate through the maxillary sinus to engage the bone at the junction of the temporal and frontal processes of the zygoma. Proper guidance of the drill and implant is facilitated by visualizing the path of insertion through a window created in the maxillary sinus wall. When possible, the head of the implant is placed within the thicker bone found at the junction of the palate and alveolar process of the maxilla.

When used for the reconstruction of maxillary defects where the palate, anterior maxilla, and inferior portions of the zygoma have been removed, three-point stabilization for a prosthesis is no longer possible and modifications to existing techniques are required. Anterior endosteal implant support is usu-

ally missing and the only bone available to engage the zygoma implants is provided by the superior portions of the zygoma(s) above the level of the zygomatico-maxillary osteotomy. This foundation is mechanically unstable for an attached prosthesis with poor resistance to lateralizing forces. There is also poor stability of the zygoma implants due to the long and unsupported lever arms. However, for patients with extensive defects unable to undergo grafting procedures to replace bone, the zygoma implant may be the only means for providing some form of retention for a maxillary obturator. One approach to the use of zygoma implants in patients following total maxillectomy, proposed by Schmidt, uses two implants placed at different angulations through the remaining zygoma bone (Fig. 50.10). These implants are allowed to integrate for 4–6 months before loading (Fig. 50.11). Coordinated planning with a prosthodontist results in a device that incorporates cross-arch stabilization by connecting all implants with a bar and clips (Fig. 50.12). Even with the adoption of these special measures, the zygoma implants experienced a 25% failure rate and anterior endosseous implants incorporated into the design failed 30% of the time. All failures occurred before loading, which suggests that non-mechanical factors were responsible for implant loss. Radiation therapy has been implicated as the principal cause of failure in these cases. When double implants are used on each side, prosthetic retention may still be possible following loss of a single implant.

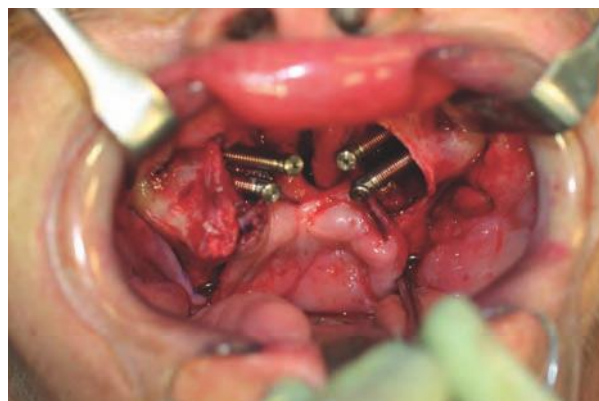


Fig. 50.10 Twin zygoma implants used for reconstruction of total maxillectomy defect. (Courtesy of Brian Schmidt, DDS, MD, PhD.)

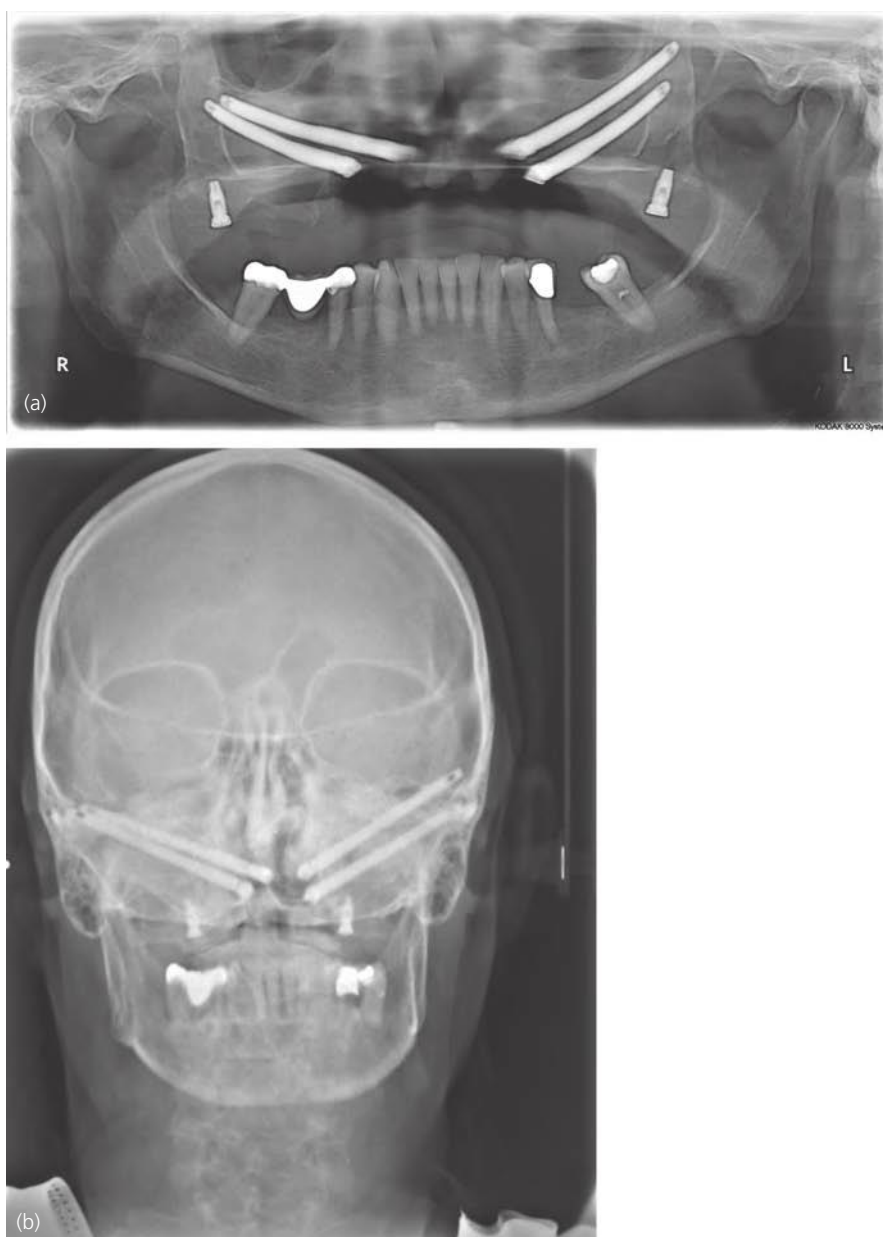


Fig. 50.11 (a, b) Zygoma implants allowed to integrate for 6 months prior to loading. (Courtesy of Brian Schmidt, DDS, MD, PhD.)

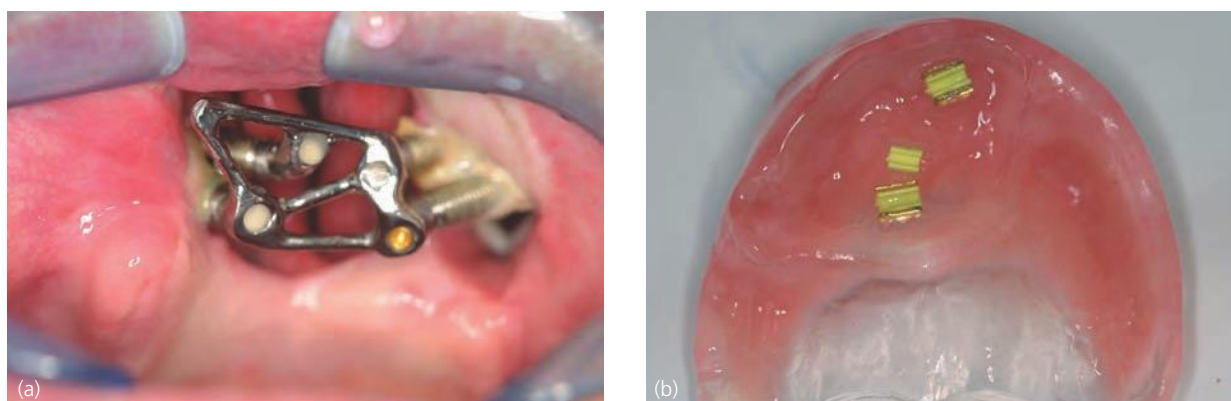


Fig. 50.12 (a) Custom fabricated framework connects all the implants providing cross-arch stabilization. (b) Overdenture attached to framework with clips. (Courtesy of Brian Schmidt, DDS, MD, PhD.)

Implants placed in irradiated bone

Radiation to bone causes a decrease in perfusion and a reduction in osteoblastic and osteoclastic activity.¹¹ As a result, an increase in implant failure and incidence of osteoradionecrosis is expected. Wagner cites a 3.4% incidence of osteoradionecrosis and close to 20% implant failure rate, after a 10-year follow-up of 60 patients who were radiated for oral cancer with over 60Gy.¹² Parel reports a similar experience describing a survival rate of 61.1% for craniofacial implants placed in irradiated bone. Other than words of caution and the usual rhetoric of “good patient selection”, consensus on methods to improve outcome in this particular patient population has not been reached. Although hyperbaric oxygen (HBO) therapy to improve implant success remains controversial, its beneficial effects on the tissue bed have been experimentally and clinically demonstrated.^{13,14} Most of the existing literature addresses implants placed after radiotherapy. It remains unclear if placing implants prior to the start of radiation improves the survival of implants.

Soft palate reconstruction

The decision to restore a soft palate defect with either a prosthetic device or surgical flap depends on size of the defect, number of teeth remaining to help support a prosthesis, and if there is a functional soft palate. Lesions of the soft palate and velopharyngeal complex are challenging defects for prosthetic rehabilitation, but tend to exhibit a better result than reconstructions of hard palate defects. Velopharyngeal closure can be achieved with an obturator and modifications can be performed as the dimensions of the defect change with healing. Soft tissue reconstruction with local or regional flaps is possible, but its effectiveness is governed by the site of the defect and the arc of rotation of the flap. If a flap is placed superiorly, it will hamper pharyngeal closure; if it is placed too inferiorly, it will interfere with both pharyngeal closure and seating of an obturator. Consequently, soft palate defects are better addressed with a prosthetic device (Fig. 50.13).

Local and regional flaps for maxillary reconstruction**Temporalis and temporoparietal-galea flaps**

The temporalis muscle flap was originally described by Golovine in 1898 for the obliteration of orbital defects following exenteration of the globe. Since then, it has been used to reconstruct different types of oral defects as an axial pattern rotational flap. Variations in technique have produced additional flaps composed of muscle and bone (temporalis-



Fig. 50.13 Obturator with soft palate extension to assist with velopharyngeal closure following excision for squamous cell carcinoma.

coronoid flap) or flaps composed entirely of the temporalis fascia with or without an attached cutaneous island (temporal fascial/fasciocutaneous flaps).

Temporalis muscle flap

The temporalis muscle arises from two regions on the skull, the temporal and infratemporal fossae, and inserts into the coronoid process. When used for oral reconstruction, this fan-shaped flap is based inferiorly on its coronoid insertion and rotated anteriorly and inferiorly into the defect site. Relative to its vascular supply, the temporalis muscle flap can be divided into three regions. The anterior portion is supplied by the anterior deep temporal artery while the middle portion is supplied by a pair of posterior deep temporal arteries. Both deep temporal branches arise from the internal maxillary artery and enter the muscle from the inferior aspect and on the deep surface. The posterior portion of the flap is supplied by the middle temporal artery, a branch of the superficial temporal artery, which also enters the muscle on its deep surface. Venous drainage and innervation from the anterior and posterior deep temporal nerves follow the same orientation as the arterial supply. Depending on the size of the defect, the temporalis muscle may be divided longitudinally into an anterior one-third flap or a posterior two-thirds flap. If necessary, the location and direction of the anterior and posterior deep temporal vessels can be confirmed with a Doppler probe following elevation of the muscle from its temporal origins.

Access to the muscle is gained with a hemicoronal or bicoronal scalp incision extending superiorly from the preauricular crease (Fig. 50.14). The coronal portion of the incision may be curved posteriorly to avoid the superficial temporal vessels. This incision is deepened through the temporoparietal fascia until the deep temporal fascia is identified. Once this structure is reached, blunt dissection in the loose areolar plane superficial to the fascia is performed in an anterior direction to expose the fascia. The preauricular inci-



Fig. 50.14 Access to temporalis muscle through hemicoronal incision. (Courtesy of Brian Schmidt, DDS, MD, PhD.)

sion is then deepened to reach the base of the zygomatic arch and the periosteum covering the arch is divided at a distance no more than 8mm from the root of the arch to avoid temporal branches of the facial nerve that cross the arch within the superficial layer of the deep temporal fascia. After the scalp and preauricular incisions have been connected, the deep temporal fascia is incised and a plane superficial to the muscle and continuous with the sub-periosteum over the arch is developed superiorly and anteriorly exposing the temporalis muscle. The fan-shaped muscle is elevated from the temporal fossa after division of the superior attachments to the cranium. From its base on the coronoid process, the muscle flap is rotated laterally over the zygomatic arch and inferiorly through the infratemporal fossa into the oral cavity. For high maxillectomies, direct access to the

mouth is achieved through the defect. With low maxillectomies, a vestibular incision in the molar area creates a communication with the infratemporal fossa for passage of the flap, taking care to avoid Stensen's duct. The zygomatic arch may be removed if it hinders rotation of the flap. The muscle flap is then sewn to the mucosal margins of the defect and, if necessary, stay sutures threaded through the bone may be used to prevent muscle retraction (Fig. 50.15). The muscle surface may be skin grafted or left bare to epithelialize secondarily (Fig. 50.16).

If a smaller amount of tissue is needed to obturate the maxillary defect, the temporalis muscle may be divided vertically at the junction of the anterior and middle one-thirds, behind the course of the anterior deep temporal neurovascular bundle. After the anterior portion of the muscle is delivered, the posterior two-thirds can be rotated anteriorly to fill the temporal defect to reduce postoperative temporal hollowing. Temporal hollowing may also be corrected with implants fabricated from alloplastic materials such as polymethylmethacrylate molded to fill the defect or prefabricated implants composed of porous polyethylene custom-prepared from stereolithographic models.

Variations of the temporalis muscle flap include a superiorly based flap, incorporating the coronoid process osteotomized at the level of the sigmoid notch, and inferiorly based flaps attached to a piece of the outer table of the temporal bone to provide both muscle and bone to reconstruct maxillary defects that include the orbital walls. The temporalis muscle flap may also be wrapped around titanium plates and mesh used for orbital and midface reconstruction.

Temporoparietal-galea flap

This is an axial pattern flap and may be used in a similar fashion as the temporalis muscle flap for maxillary reconstruction. Instead of muscle, it is composed of



Fig. 50.15 (a–c) Class 2a maxillary defect reconstructed with temporalis muscle flap. Flap is delivered intraorally through vestibule and sewn into defect. (Courtesy of Brian Schmidt, DDS, MD, PhD.)



Fig. 50.16 Temporalis muscle flap 2 weeks after insertion. Note secondary epithelialization.

the temporoparietal fascia, part of the subcutaneous musculoaponeurotic system (SMAS). It is a thinner flap and is used when a less bulky soft tissue reconstruction is desirable, such as for coverage of metal plates or bone. The vascular supply of this flap is derived from the posterior branch of the superficial temporal artery, which forms when the artery divides into anterior and posterior branches 2–4 cm above the arch. The vessels travel in the temporoparietal fascial layer and must be preserved during the superficial and deep dissections to expose the flap. Using a hemicoronal incision, a superficial dissection is made in a plane just below the subcutaneous layer of the skin. Hair follicles superficial to the level of dissection confirm that the correct plane has been achieved. The attachment between the underlying temporoparietal galea and the overlying skin can be tenacious and this dissection should be carried out carefully. After elevation of the skin, the course of the superficial temporal artery and branches should be identified visually and with the assistance of a Doppler probe (Fig. 50.17). The flap is rectangular in shape and based inferiorly at the level of the zygomatic arch. To create the posterior margin of the flap, a vertical incision is made behind the posterior branch of the superficial tempo-



Fig. 50.17 Temporoparietal–galea flap: elevation of the skin at the subcutaneous level exposes the galea. Superficial temporal vessels and branches are visualized through the galea.



Fig. 50.18 Rectangular temporoparietal–galea flap elevated.

ral artery and vein through the temporoparietal fascia to the underlying muscle. The length and width of the flap is determined by the size and location of the defect and this can be gauged with the assistance of a suture or umbilical tape rotated into the defect from a pivot point placed at the base of the flap. Before creating the anterior vertical incision of the flap, the anterior branch of the superficial temporal artery must first be ligated. Between the fascia and muscle is a layer of loose areolar tissue, which provides an easy plane for completion of the deep dissection (Fig. 50.18). The flap is rotated into the oral cavity in a similar manner as the temporalis muscle flap.

Buccal fat pad flap

Ideally suited for small retromolar and posterior maxillary defects, this axial pattern flap enjoys a robust blood supply with contributions from the buccal and deep temporal branches of the maxillary artery, the transverse facial branch of the superficial temporal artery, and buccinator branches from the facial artery. Within the fat pad, a network of small arterioles and venules is present and care must be taken to avoid disruption of these vessels through overzealous manipulation. The fat pad is multilobular with each lobe enclosed within a thin capsule and attached to adjacent structures by supporting ligaments. Variations in position of the fat pad occur throughout life accounting for the full facial contours present in infants and the hollowed appearance of the cheeks in the elderly. However, the volume of the fat pad remains fairly constant and has been estimated to be approximately 10 ml. The fat pad is organized into anterior, intermediate, and posterior lobes, with four processes (buccal, temporal, pterygoid, and pterygopalatine) extending from the posterior lobe. The different lobes lie between the buccinator medially, masseter laterally, the ascending ramus of the mandible posteriorly, and the zygoma superiorly.¹⁵ The central corpus composed of the anterior and intermediate lobes along with the buccal process of the posterior lobe represent the major components of the buccal fat pad flap (BFPF) used for reconstruction. The buccal branches of the facial nerve and the parotid duct are lateral to the fat pad and are usually not encountered during flap development.

If access to the fat pad has not already been created by the ablative procedure, an incision in the vestibular sulcus distal to the maxillary tuberosity through the periosteum will expose the buccal fat pad.¹⁶ After widening the access to prevent constriction of the BFPF pedicle, the flap is placed under tension at its leading edge while blunt dissection mobilizes the flap by dividing the supporting ligaments. Between 7 and 9 cm of length may be achieved and the flap is sewn to the defect margins either with sutures to a soft tissue margin or tied to holes drilled through the bony margins. The BFPF is a highly versatile surgical tool in maxillary reconstruction and can be used to

close class I and IIa maxillectomy defects, oro-antral communications, and lateral wall and palatal voids. However, defects larger than 4 cm in diameter, or in irradiated fields, may not be suitable for reconstruction with a BFPF.¹⁷ Although the buccal fat pad has been used as a vascularized bed to cover a bone graft during maxillary reconstruction,¹⁸ it is not sufficiently reliable for this purpose.

The BFPF epithelializes rapidly, rendering additional coverage with STSGs unnecessary. Initially, the flap surface is covered with granulation tissue and this undergoes a hyperkeratotic response before the surface fat cells are transformed into a thickened stratified squamous epithelium over a period of 5 weeks (Fig. 50.19).¹⁹ The BFPF technique is associated with low morbidity and produces minimal contour changes and facial asymmetry. The only problem is obliteration of the maxillary vestibule in the region where the flap traverses from the cheek to the maxilla, and this may compromise stability of a prosthesis (Fig. 50.20).

Preserving space after bone removal

Immediate reconstruction following tumor removal or immediately after an avulsive injury is infrequently performed, despite the benefits of ready access to the defect, lack of soft tissue contraction from scarring, and early return to function. Large, avulsive injuries following trauma typically involve the loss of hard and soft tissue, with the potential for additional tissue necrosis as the delayed effects of vascular damage become apparent. Consequently, immediate reconstruction is not undertaken in most trauma patients. For defects resulting from tumor ablation, concerns over disease eradication mitigate against immediate reconstruction. However, defects created during the treatment of localized, benign disease may be addressed at the time of removal and this approach provides the optimal functional and esthetic result (Fig. 50.21).

One of the problems associated with delayed reconstruction is scarring of the soft tissue envelope

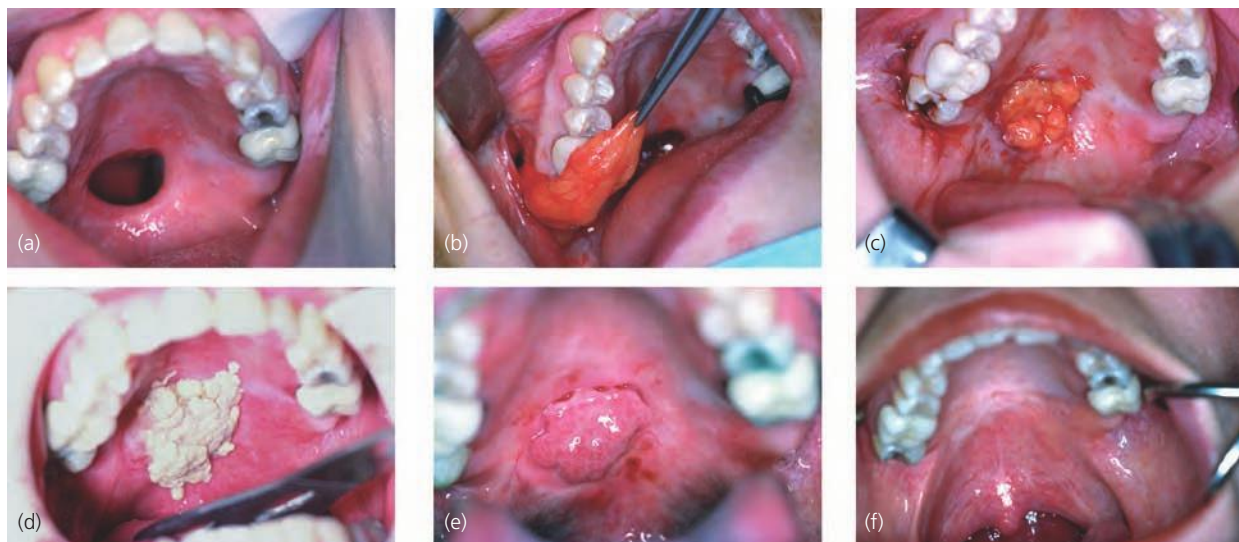


Fig. 50.19 (a) Maxillary defect following resection of low-grade mucoepidermoid carcinoma. (b) Delivery of buccal fat flap. (c) Buccal fat pad flap inset into defect. (d) Postoperative week 1; fat undergoes fibrosis. (e) Postoperative week 3; secondary epithelialization and contracture of flap. (f) 6 months postreconstruction. (Courtesy of James Wilson, DDS.)

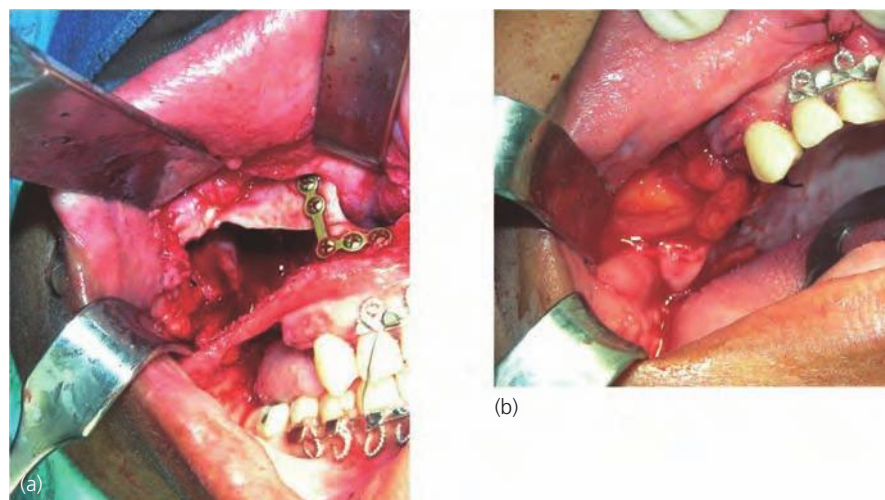


Fig. 50.20 (a, b) Lateral maxillary defect reconstructed with buccal fat flap. Note the obliteration of the maxillary vestibule.

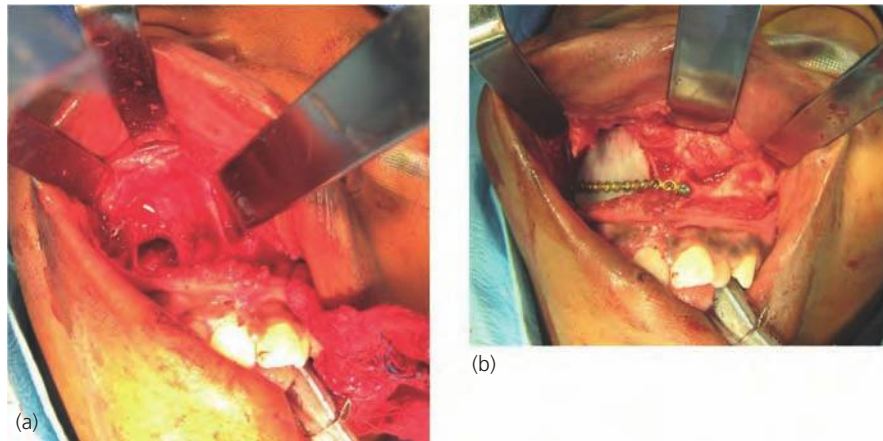


Fig. 50.21 (a, b) Immediate reconstruction of maxillary wall defect.

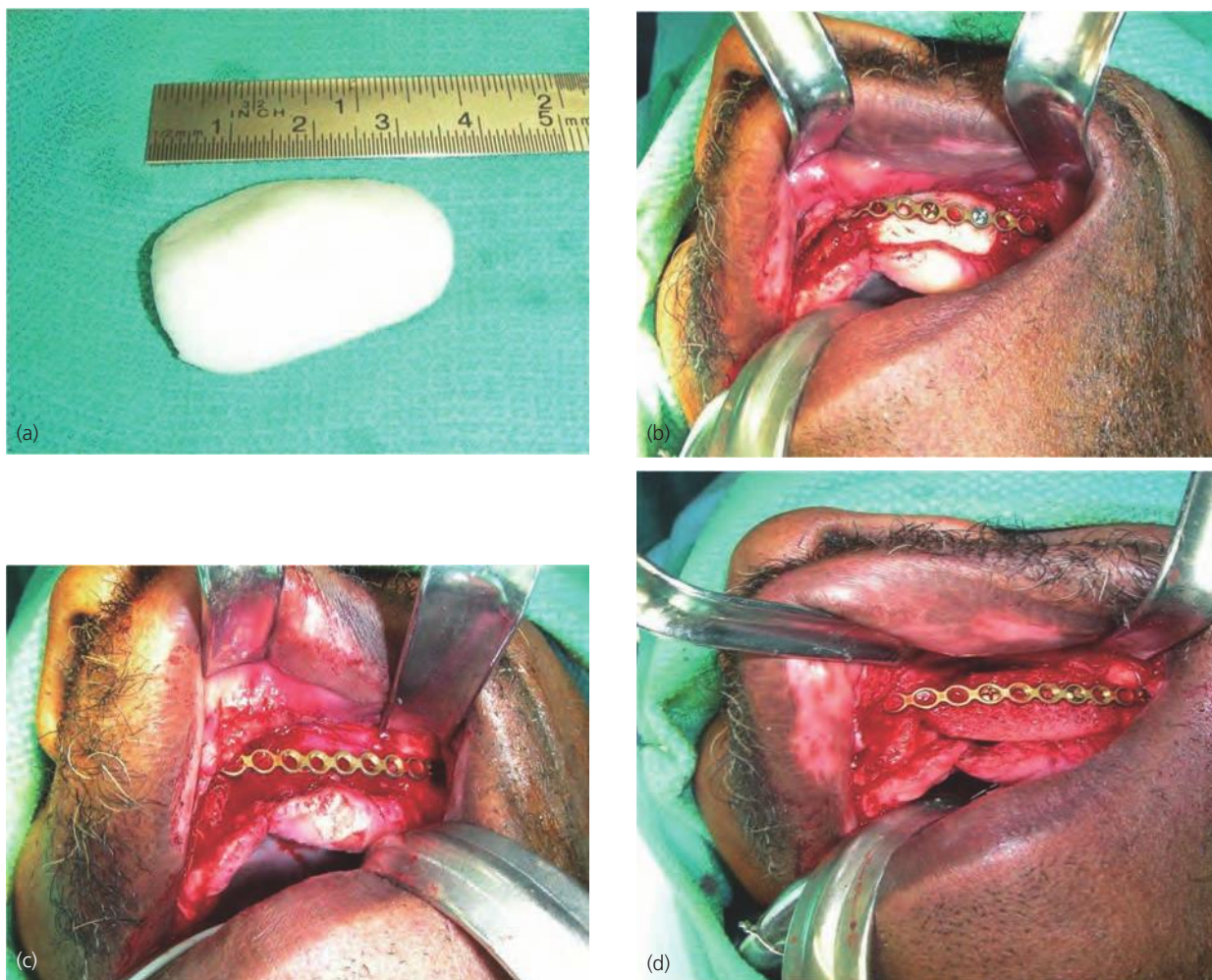


Fig. 50.22 (a, b) Custom-molded polymethylmethacrylate space maintainer formed and placed into defect. (c, d) During secondary reconstruction, spacer removed and used as template for bone graft.

surrounding the skeletal defect. This can limit the size of the graft that can be used, especially with regards to skeletal height. To preserve the dimensions of the missing skeletal bone, a methylmethacrylate (MMA) space maintainer can be used (Fig. 50.22a, b). Dehiscence of the overlying soft tissue with exposure of the device remains an issue, although there are several reports supporting its use.²⁰ In the authors' experience, problems with wound healing can be

reduced by achieving a tension-free closure around the spacer, rigidly fixating the implant in place, and limiting the interval between device implantation and secondary reconstruction of the defect. If there are concerns over bacterial wound contamination, a broad-spectrum antibiotic (a non-heat-labile antibiotic such as tobramycin or vancomycin) can be incorporated with the MMA device.²¹ Another use of the spacer is as a template to fashion a bone graft for

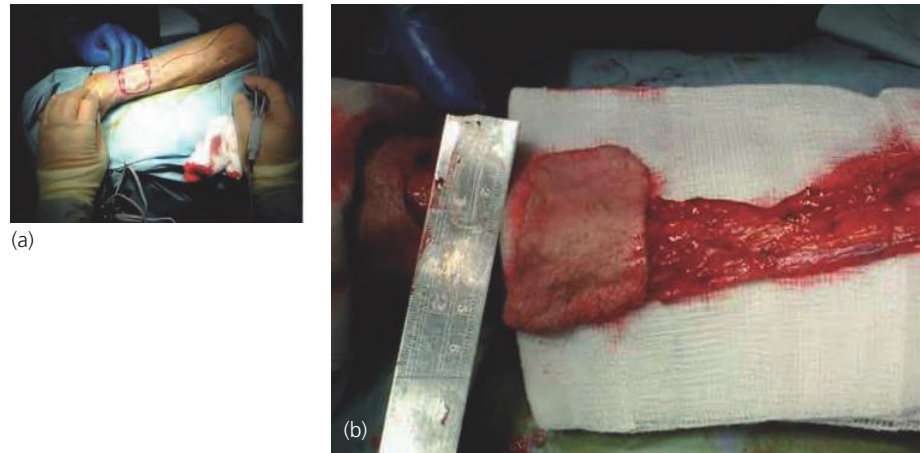


Fig. 50.23 (a) Radial forearm free flap harvest; paddle outline. (b) Radial forearm flap; vascular pedicle.

a more precise fit (Fig. 50.22c, d). MMA for these purposes is available as bone cement in sterile packaging, with and without antibiotics and in different viscosities. When used, attention should be paid at the time of secondary bone graft placement to remove any granulation tissue and the fibrous capsule which forms around the spacer to allow the early establishment of a vascular supply to the bone graft.

Free tissue transfer in maxillary reconstruction; soft tissue obturation

Where the maxillectomy defect involves the orbital adnexae or more than half of the maxillary alveolus, obturation with a prosthesis will rarely give a satisfactory outcome. In such cases surgical solutions provide superior results and patient satisfaction.²² Free tissue transfer using microsurgical anastomotic techniques has revolutionized reconstructive surgery over the last 30 years. The ability to import tissue to the head and neck from donor sites elsewhere in the body provides sources of reconstructive tissue which can be tailored to the specific requirements of the defect for an individual patient. These include both soft tissue only flaps (fasciocutaneous, e.g. radial forearm and anterolateral thigh, and musculofasciocutaneous, e.g. latissimus dorsi and rectus abdominus) and also composite soft and hard tissue flaps (osteocutaneous, e.g. fibula flap, and osteomyocutaneous, e.g. vascularized iliac crest with internal oblique with or without skin). There are challenges in reconstructing the midface in order to provide adequate restoration of form and function. A further major consideration is the distance of the recipient bed from donor vessels in the neck and a pedicle of adequate length is required for successful reconstruction.

Radial forearm free flap

First described in China by Guofan *et al.* in 1981,²³ the use of the radial forearm fasciocutaneous free flap



Fig. 50.24 Type I maxillectomy defect reconstructed with radial forearm flap contoured to recreate the buccal sulcus. Patient is able to wear a standard maxillary denture.

was described in 1982 by Muhlbauer for use in the oral cavity. It was further popularized as a reconstructive tool for the oral cavity by Soutar.²⁴ This flap is now regarded as a reconstructive workhorse in the head and neck generally, providing a reliable paddle of pliable fascia and skin to restore both function and cosmesis.

The flap is raised on the radial artery and venae comitantes vessels. The superficial venous system (cephalic vein) may also be raised and traced proximally either as a separate venous pedicle or to the venae perforantes which unite the superficial and deep venous systems (Fig. 50.23).

In the context of the limited maxillectomy defect (class 1), and especially in the edentulous patient, closure of the defect with this flap produces predictable and satisfactory outcomes. Here the flap is tailored to the size of the defect and, once inset, replaces the lost tissue as well as the upper buccal sulcus, facilitating retention of a complete denture prosthesis (Fig. 50.24). The pedicle is passed along a tunnel fashioned within the buccal tissues and the artery and vein(s) anastomosed to suitable vessels within the neck. Advantages of this technique include reliability of the flap, a long pedicle, and good-sized vessels, which facilitate anastomosis to vascular sources in the neck.

Where the defect is of greater dimensions (Brown class 2 and greater) the reconstructive aims will be tailored according to the overall oral rehabilitation plan for the patient. Ideally, adequate bone to support osseointegrated implants, together with appropriate soft tissue coverage are required to restore form and function. Previously, use of larger soft tissue free flaps such as rectus abdominus^{25–27} and latissimus dorsi^{26,28–34} myocutaneous flaps was described. While these provide adequate obturation of the maxillary defect, such bulky flaps often result in a ptotic soft tissue mass which makes prosthetic rehabilitation extremely challenging, if at all possible. Furthermore, flap ptosis can impair function, such as speech and swallowing. Facial form restoration is also suboptimal.³⁵

When skeletal and alveolar augmentation is needed to support implant placement, the free tissue transfer technique must include bone. While none of the available reconstructive options can accurately replace all the lost tissue, each of the options provides varying quality of tissue with which to obturate the defect, restore form, and provide a basis for osseointegrated implants. The principle flaps used in this regard include the radial forearm osteofasciocutaneous flap, the fibula osteofasciocutaneous flap, the scapula osteocutaneous flap, and the vascularized iliac crest with internal oblique muscle graft.

Radial forearm osteofasciocutaneous flap

The radial forearm flap, incorporating a segment of the radius, was initially described by Soutar in mandibular reconstruction³⁶ and more recently in the maxilla.^{37,38} This flap has the advantage of two-team harvest and a long and reliable pedicle. The flap is also a variant of the soft tissue only radial forearm free flap and the anatomy and surgical procedure are therefore very familiar to operators in the field of head and neck reconstruction. Bone of the distal radius can be harvested to a maximum of 40% of the diameter and up to 16 cm of the length of the radius. This provides somewhat limited bone stock and the flap is therefore best suited to smaller defects of the premaxillary region, maxillary alveolus and orbital rim^{39–41} rather than larger class 2 and greater maxillectomy defects. This flap does not provide adequate bone to support implants.

The vascular supply to the radius is developed by preserving periosteal perforators from the radial vascular pedicle, composed of the flexor pollicis longus and pronator quadratus muscles. Access to the bone is gained by incising through the muscles on the ulnar aspect and preserving the radial attachments. The harvested bone segment is freed by dividing the brachioradialis muscle laterally. This is illustrated in Fig. 50.25.

While this technique provides a reliable reconstructive flap which can be raised simultaneously by

a second surgical team, the limited bone restricts its utility to smaller defects.^{39–41} Donor site morbidity includes fracture of the remaining radius. This is reported in a small proportion of cases and can be minimized with appropriate fixation techniques. Fig. 50.26 is a radiograph demonstrating plate fixation of the radius after osteocutaneous forearm flap harvest.^{42,43}

Fibula osteocutaneous flap

This flap was originally developed to reconstruct ulna and tibial defects⁴⁴ and was later adapted for use in mandibular reconstruction.⁴⁵ After gaining popularity as a reliable method for mandibular reconstruction,⁴⁶ it was later applied to the maxillectomy defect where it has become a workhorse.^{37,47–49} The fibula flap is based on the peroneal artery and vein and provides the greatest length of available bone of any composite free flap (Fig. 50.27). From an anterior incision, perforator vessels from the peroneal artery, which supply the fasciocutaneous tissue, are identified, prior to outlining a skin paddle. The paddle is centered on these vessels, thereby increasing the survival of the skin portion of the flap (Fig. 50.28).

The bony component of the flap is triangular in cross-section and narrow and comprised mainly of compact cortical bone. The bone can be folded on itself in a “double-barrel” arrangement to increase the height of the graft, but this is more of a concern with mandibular reconstructions.⁵⁰ Pedicle length is limited by the site of bifurcation of the tibioperoneal trunk into the peroneal and posterior tibial arteries. The usual pedicle length is around 6–7 cm, but this can be increased by subperiosteal dissection of the fibula more proximally to produce a pedicle of 12 cm (Fig. 50.29).⁵¹ While revision of the graft, primarily to reduce the bulk of the soft tissue, is usually required prior to prosthetic rehabilitation of the patient, the fibula flap can achieve an excellent reconstruction of a maxillary defect (Fig. 50.30). It is capable of completely sealing the mouth from the nasal and antral cavities and provides sufficient bone to support implant placement.

Scapular angle osteomyogenous flap

A series of cases using this reconstruction method for the maxillectomy defect was described recently by Gilbert and coworkers in Toronto.³⁵ The scapula free flap has been used extensively in mandibular reconstruction since the first recognition of this donor site in 1978 by Saijo.⁵² Described specifically for reconstruction of the maxilla and mandible later by Swartz, this flap is a popular choice for head and neck reconstruction, offering versatility of orientation of skin paddles and bony paddles as well as the inclusion of muscle paddles where required.^{32,34,53–57}

The scapular angle osteomyogenous flap is particularly suited in the elderly patient population,

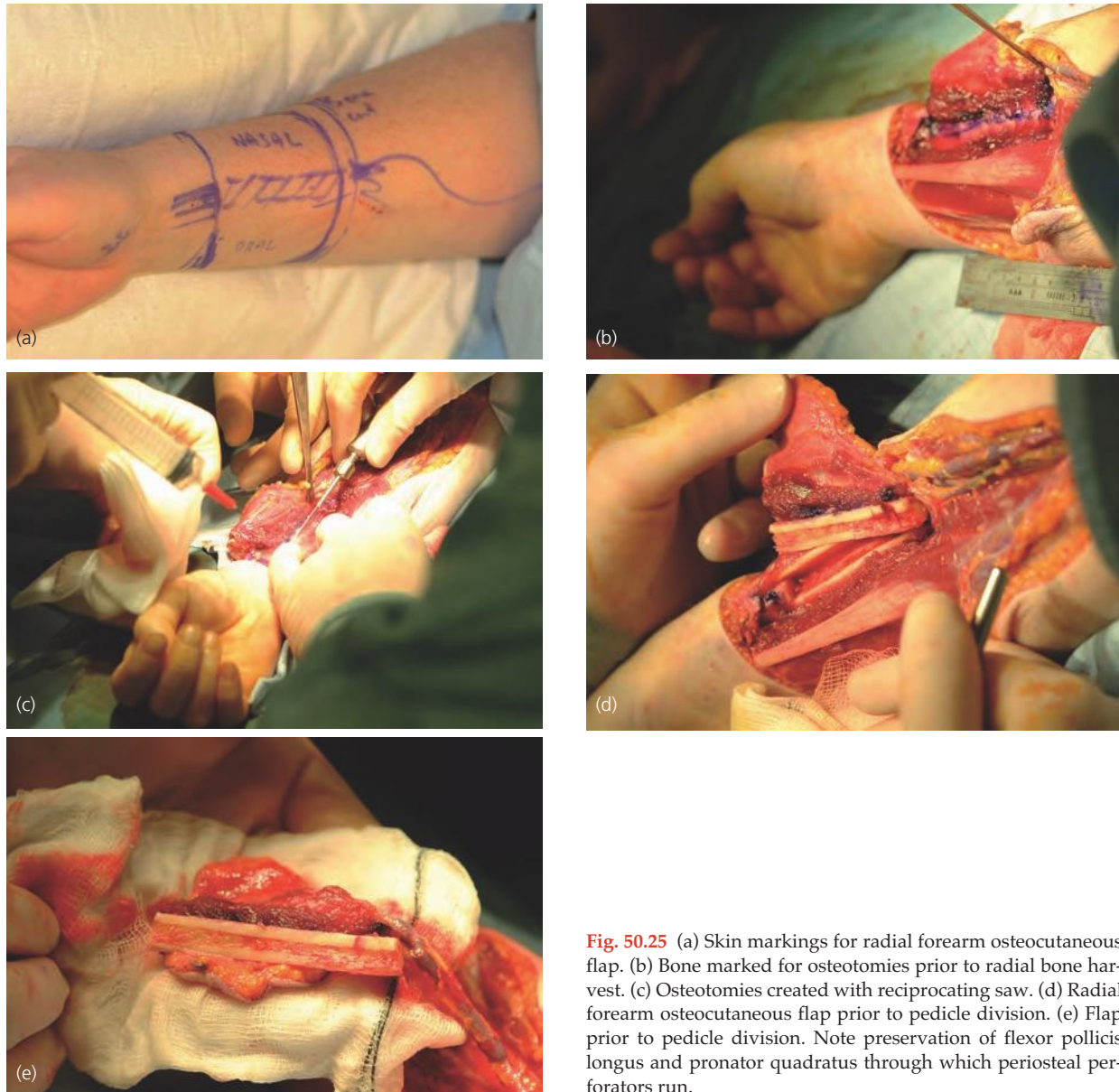


Fig. 50.25 (a) Skin markings for radial forearm osteocutaneous flap. (b) Bone marked for osteotomies prior to radial bone harvest. (c) Osteotomies created with reciprocating saw. (d) Radial forearm osteocutaneous flap prior to pedicle division. (e) Flap prior to pedicle division. Note preservation of flexor pollicis longus and pronator quadratus through which periosteal perforators run.

because of the increased incidence of peripheral vascular disease. Additionally, these patients may have difficulty ambulating after fibula flap harvest.³⁵ It is also a reliable flap with low failure rates and minimal donor site morbidity.³⁵ Advantages of this flap for use in midface reconstruction include a generous pedicle length up to 20 cm and the vessels are sufficiently large to facilitate microvascular anastomosis (Fig. 50.31). Scapular bone is similar in structure to the native maxilla, being thin medially and thicker toward the lateral border. Consequently, the bony component of this flap is harvested from the lateral margin of the scapula. Average thickness at this site is approximately 28 ± 3 mm,⁵⁸ which is adequate for implant placement, but of inferior quality when compared with the type I bone normally associated with the fibula or iliac crest.^{59,60}

The scapular bone is easy to contour using a “greenstick” method where one cortex is sectioned and the other curved by manipulation. The flap can be oriented vertically to restore the nasomaxillary



Fig. 50.26 Postoperative radiograph of donor arm after osteocutaneous radial forearm flap harvest and fixation.

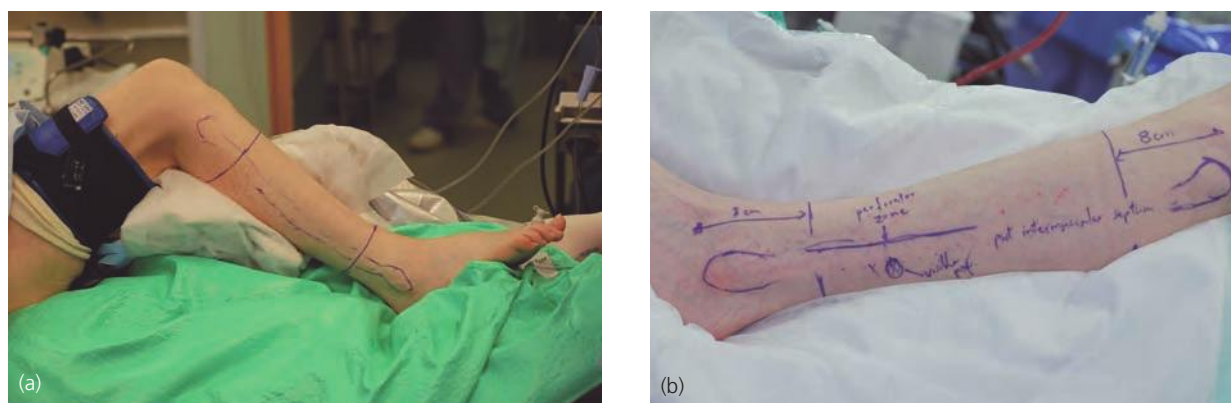


Fig. 50.27 (a, b) Fibula flap harvest skin markings. Note the 8 cm length of bone maintained proximally and distally in order to ensure stability of the knee and ankle joints respectively. The perforator zone is marked, where perforator vessels run around posterior to the fibula and then in the posterior intermuscular septum to supply the skin paddle. Making an anterior incision to allow precise identification of these perforators increases reliability of the skin paddle.

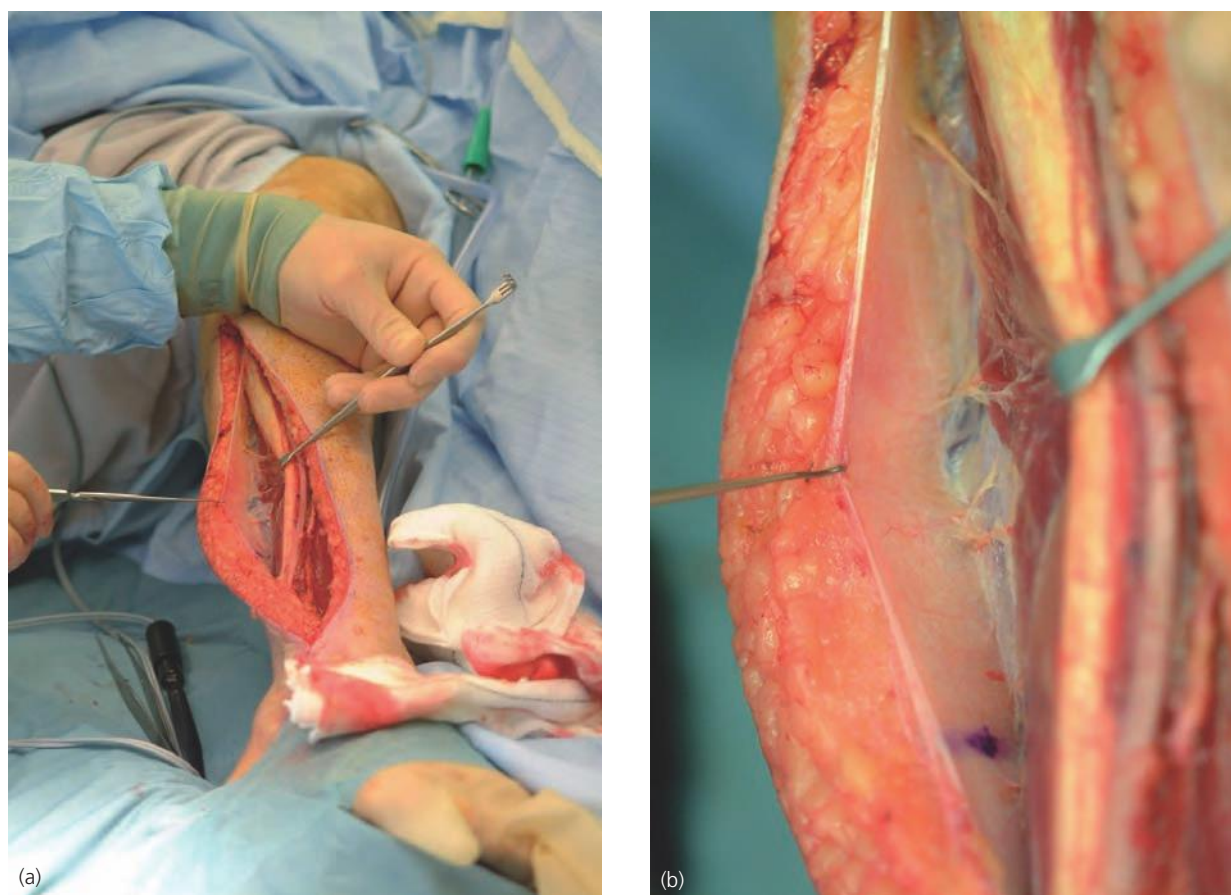


Fig. 50.28 (a, b) Development of a skin paddle. Perforator vessels from the peroneal artery passing through the posterior intermuscular septum into the fasciocutaneous paddle are demonstrated. The soft tissue component is centered on these vessels facilitating flap survival.

and zygomaticomaxillary buttresses or horizontally to replace the palatoalveolar complex in a similar fashion to that described by Brown with the vascularized iliac crest.⁶¹ In order to harvest a scapula flap, the patient must be turned to the lateral decubitus position (Fig. 50.32). A two-team approach is therefore not possible when using this technique, which adds to operative time. Clark *et al.* describe the use of a suction bean bag to achieve and maintain patient orientation during flap harvest.³⁵

Vascularized iliac crest with internal oblique muscle flap

This technique is unique in providing adequate bone to restore both facial and oral skeletal structures in both low- and high-level maxillectomy defects. Adequate bone stock is therefore available for the placement of dental implants.

Initial development of the flap, supplied by the deep circumflex iliac artery (DCIA), occurred in

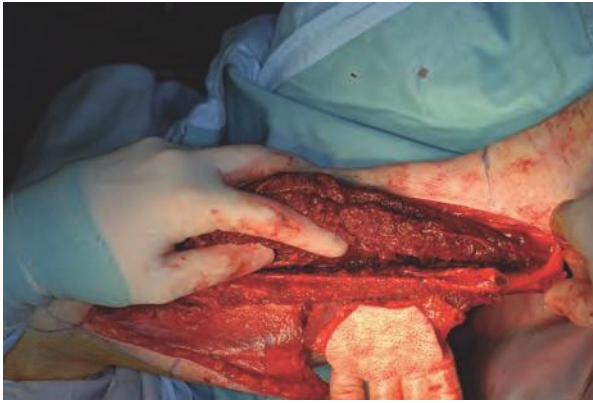


Fig. 50.29 Fibula flap harvest prior to division of the pedicle. The fasciocutaneous paddle has been developed and osteotomy of the proximal end of the fibula demonstrates the triangular cross-section of bone obtained.

England and in Australia,^{62,63} before the technique was adopted for mandibular reconstruction by Urken and colleagues.^{64,65} This group reported a large series of cases with high success rates and low donor site morbidity.⁶⁶ In addition to excellent bone volume available from this site, the accompanying internal oblique muscle, supplied by the ascending branch of the DCIA, provides excellent soft tissue for bone coverage and defect obturation. This technique has now been adopted for use in midface reconstruction^{22,37,61} and a large series using this method has been reported by Brown and colleagues.³ Low-level maxillary defects can be reconstructed with the bone flap oriented horizontally; higher-level defects position the bone flap vertically.⁶¹ In this study 87% of patients who survived the primary disease progressed to full oral rehabilitation with dental implants. Donor site problems are minimal and flap complication rates reduced with increasing experience of the operators. The vascularized iliac crest is therefore the most suc-

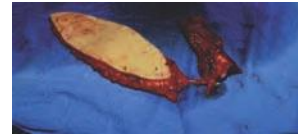


Fig. 50.31 Scapular osteomyocutaneous flap.



Fig. 50.32 Lateral decubitus positioning for scapular flap.

cessful technique in the armamentarium of microvascular surgeons for the rehabilitation of midface defects after surgery for cancer or trauma.

This technique is illustrated in Figs 50.33–50.36 and the sequence of operative events is described. An expanding lesion in the left posterior alveolus was diagnosed as a chondrosarcoma of the left maxilla. Control of regional disease was established with a selective lymphadenectomy of levels 1–3 with preservation of the facial artery and vein at the lower border of the mandible for the graft anastomosis (Fig. 50.33a). Once this had been completed, resection of the left maxilla through a modified, extended Weber-Ferguson incision was performed (Fig. 50.33b, c). Simultaneous with the ablative procedure, a second surgical team is able to raise the vascularized iliac crest graft. The incision line and femoral vessels are marked. The contour of the ipsilateral iliac crest is similar to that of the corresponding maxilla and is therefore selected. The internal oblique muscle is

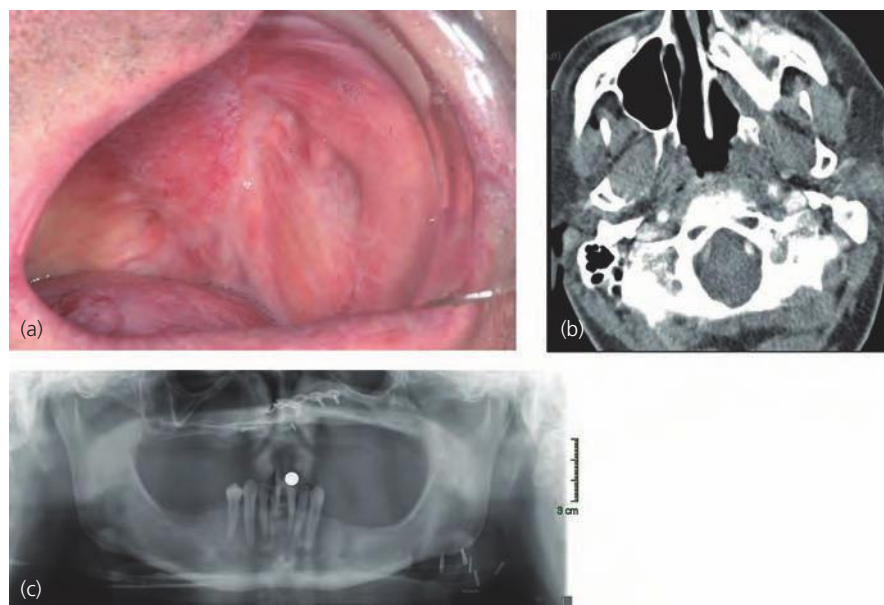


Fig. 50.30 (a–c) Fibula osteomyocutaneous reconstruction of left maxillary defect. (Courtesy of Mr Stuart Hislop.)



Fig. 50.33 (a) Completed left neck dissection with preservation of facial artery and vein. (b) Access to the left maxillary tumor gained through a modified, extended, Weber-Fergusson incision. The nasal esthetic units are also marked. (c) Maxillary defect following left maxillectomy.

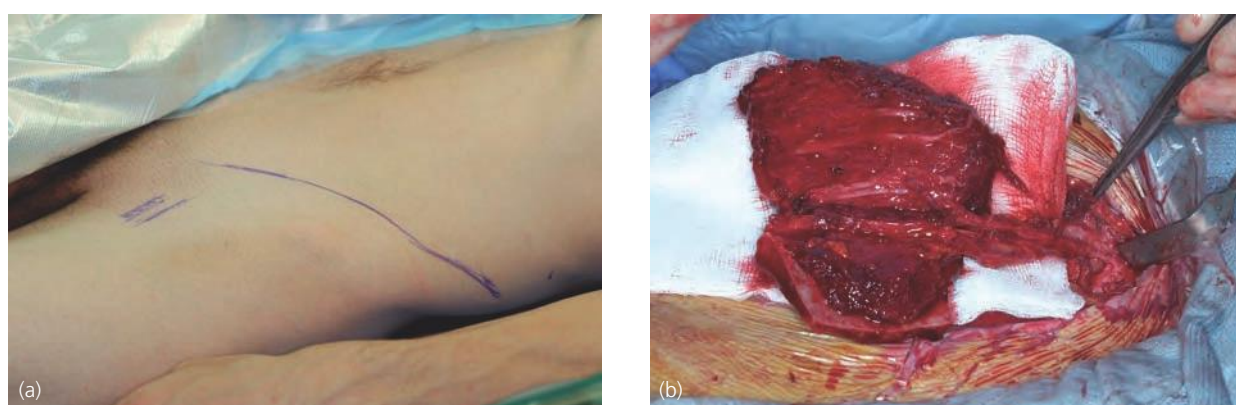


Fig. 50.34 (a) Incision for a left vascularized iliac crest harvest. Femoral vessels marked. (b) Vascularized iliac crest graft with internal oblique muscle supplied by the deep circumflex iliac vessels.

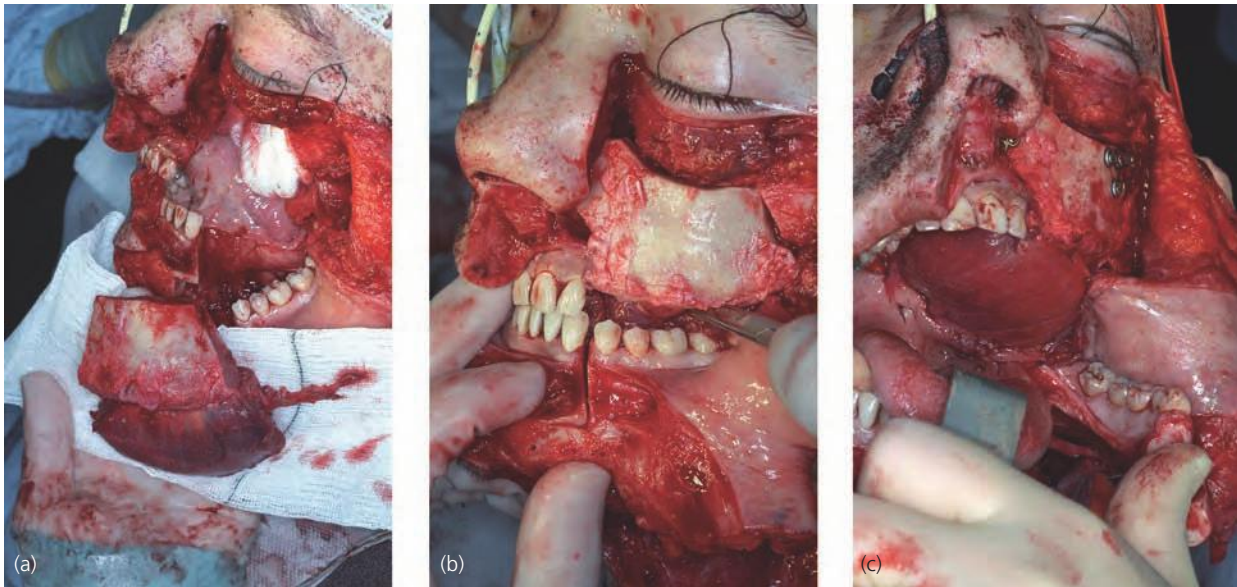


Fig. 50.35 (a) Vascularized iliac crest graft with attached internal oblique muscle and deep circumflex iliac vessels. The mandible has been osteotomized to improve access for the anastomosis to the facial vessels. (b) Bone flap inset into defect restoring the contours of the left maxilla. (c) Approximation of internal oblique muscle to buccal, palatal, and nasal lining.

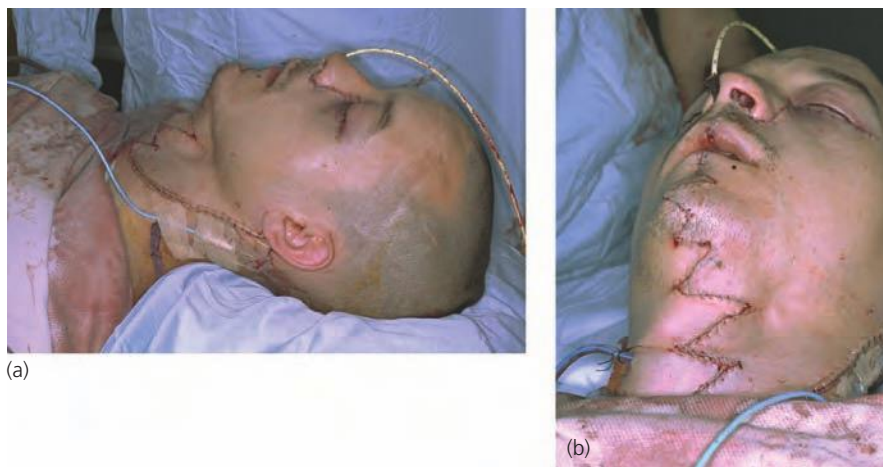


Fig. 50.36 (a) Projection of the left maxilla achieved with the iliac crest vascularized flap. (b) Incision lines are stepped to improve the esthetic qualities of the soft tissue closure.

raised on the deep circumflex iliac vessels and this forms the pedicle for the underlying crestal bone harvest (Fig. 50.34).

Once the graft has been harvested, the bone is trimmed and shaped to match the maxillary defect and the flap inset and fixated in position with bone plates (Fig. 50.35a, b). The vascular pedicle is tunneled through the buccal tissues to reach the facial vessels in the left neck and access for the anastomosis facilitated by dividing the mandible temporarily with a parasymphiseal osteotomy. The integrity of the oral lining is restored by suturing the internal oblique muscle to the buccal mucosa laterally and nasal and palatal mucosa medially and this completes obturation of the maxillary defect (Fig. 50.35c). The final views demonstrate the excellent facial contour reproduced by the iliac crest vascularized flap (Fig. 50.36).

Conclusion

Maxillary defects pose significant reconstructive challenges. This chapter presents an account of the various techniques, both biological and alloplastic, that have become the principal methods for addressing these defects. The final choice is based on several factors, principally the etiology of the defect, a patient's ability to undergo major reconstructive surgery, and, finally, the expertise available to treat the case.

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Chapter 51

Mandibular Reconstruction

M. Anthony Pogrel and Brian L. Schmidt

This chapter describes the reconstruction techniques available after marginal and segmental resection of the mandible. The non-vascularized bone grafting procedures available, including rib grafting, corticocancellous block grafting, and marrow grafting in a crib, are described. The techniques for obtaining bone grafts from the ribs, anterior iliac crest, posterior iliac crest, and tibia are described. The principles of microvascular free bone grafting to reconstruct the mandible are described. The advantages and disadvantages of staged reconstruction versus one or two procedures are described, covering the spectrum from primary bone grafting to provision of osseointegrated implant-retained prostheses. The effects of radiation therapy on mandibular reconstruction are also discussed.

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Whenever a portion of the mandible is resected, consideration must be given to subsequent reconstruction. Although most patients do receive mandibular reconstruction following resection there is still a minority for whom no reconstruction may be the most practical alternative. These cases particularly include shorter lateral defects, not involving the symphysis and parasymphysis areas, in elderly or

medically compromised patients. The subsequent deformity in the absence of reconstruction is often acceptable under these circumstances. However, at present, the “gold standard” for reconstruction is autogenous bone.

Marginal resection

Marginal resection of the mandible may be indicated for the excision of a benign tumor, a superficial malignant tumor involving periosteum only, or for infection. In this case mandibular continuity is not disrupted. However, the mandible is at risk for subsequent fracture, particularly if radiation therapy has to be given.¹ For this reason, reconstruction is often performed. Bony reconstruction will also enable subsequent insertion of osseointegrated implants, if indicated, to complete prosthodontic reconstruction. In a young patient reconstruction may not be necessary since some spontaneous regeneration of bone may occur, particularly if the periosteum is retained.² However, the mandible may need to be reinforced with a reconstruction plate (usually around 2.4 mm in thickness with appropriate screws) to minimize the risk of fracture. Following marginal resection, reconstruction can be with corticocancellous blocks from the iliac crest (full thickness or partial thickness), which can be contoured so that they will mortise into place with minimal or no fixation (Fig. 51.1). For a lesion less than about 3 cm in size this can be carried out immediately from an intraoral approach; for defects greater than this, consideration should be given to carrying this out secondarily and possibly from an extraoral approach, to avoid the risk of graft failure due to infection secondary to salivary contamination. Subsequent soft tissue grafting is often necessary to supply attached mucosa for denture or implant insertion. This is often carried out by means of a split-thickness skin graft, and normally done as a secondary procedure following incorporation of the bone graft.

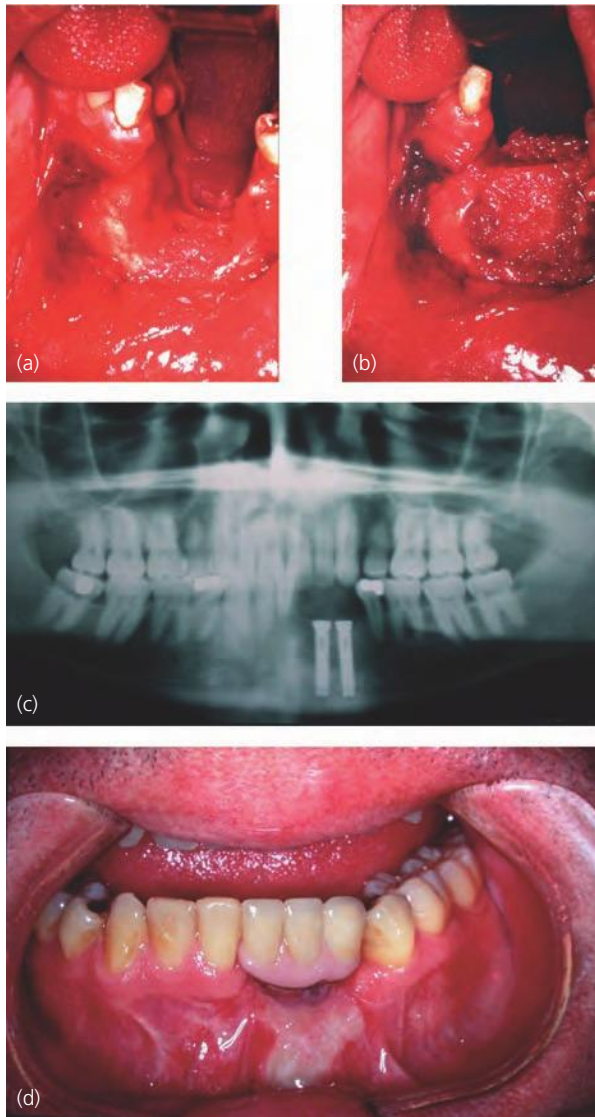


Fig. 51.1 A marginal resection of the mandible for a fibro-osseous lesion (a) reconstructed with an iliac crest bone graft morticed into place intraorally at the time of the primary resection (b) and secondarily skin grafted and implants inserted and an implant supported restoration placed (c, d).

Segmental resection

Segmental resection of the mandible with loss of mandibular continuity may be necessary for benign or malignant lesions, as well as for chronic infection.



Fig. 51.2 A fractured reconstruction plate used to span a segmental defect, which had not received a bone graft.

Temporary immediate reconstruction is often carried out by means of a reconstruction plate, but these are rarely satisfactory for long-term use. The usual history is for the plate to either exteriorize itself and become secondarily infected, or for screw loosening and mobility to occur. Depending on the forces placed on it, these problems will normally occur after some 18 months or 2 years. Plate fracture has also been reported (Fig. 51.2). In particular, reconstruction plates fail most frequently in the anterior mandible, but can survive in the long-term in the body and posterior parts of the mandible, particularly if there is adequate soft tissue coverage, which may have to be in the form of a soft tissue flap, such as a pectoralis major flap.³

Nevertheless, it is generally more appropriate to reconstruct the mandible with non-vascularized autogenous bone. Non-vascularized autogenous bone is normally used as a secondary procedure. Immediate segmental reconstruction with non-vascularized autogenous bone from an intraoral approach has been associated with a high failure rate, although it was recommended by Obwegeser amongst others.⁴⁻⁶ The reasons for failure are generally wound breakdown intraorally and contamination of the wound with saliva, or primary infection due to oral contamination. Reconstruction is therefore more normally carried out secondarily. The most appropriate time to carry this out would appear to be some 6–8 weeks following primary resection for the following reasons:^{7,8}

- this gives the intraoral wound time to heal and gain some tensile strength;
- the wound bed has time to self-decontaminate;
- scarring and fibrosis have not had an opportunity to develop.

Resection is often carried out from a combined intraoral and extraoral approach, and temporary reconstruction is carried out with a titanium reconstruction plate (usually a 2.4mm plate is adequate). Some 6–8 weeks later, definitive reconstruction is carried out from an extraoral approach using autogenous bone. Depending on the circumstances, this can be by the use of autogenous ribs (usually in a younger patient)

(Fig. 51.3), corticocancellous blocks from the iliac crest (full thickness or partial thickness) (Fig. 51.4), or cancellous bone marrow in a carrier tray.⁷ The tray can be of titanium mesh or an allogenic bone tray (Fig. 51.5), or in exceptional circumstances, it may be an autogenous bone tray, since it may be possible to carry out a segmental resection of the mandible for a benign lesion, excise the lesion from the mandible, and reinsert it as its own carrier tray.

Over the years, many attempts have been made to reconstruct the mandible using the actual autogenous mandible that was previously resected. In the case of a benign lesion, it may be possible to resect the mandible, remove the lesion, and reinsert the mandible. In most cases this does mean hollowing the mandible out to resemble a tray, perforating the tray, and

securing it with a reconstruction plate, and then completing the reconstruction with autogenous marrow within the tray. This does give the most esthetic reconstruction, since it is the patient's own mandible and therefore the contour is the most acceptable. Fig. 51.6 shows this technique used for a high-flow vascular malformation of the mandible. When this treatment is contemplated for aggressive lesions, or even malignant lesions, the resected mandible must be "sterilized" in some way to kill unwanted cells. In most cases the technique is as just described, in removing the lesion from the resected mandible, reducing the mandible to an autogenous tray and perforating it. Three different methods of sterilizing the mandible have been described. The first involves autoclaving the mandible;^{9,10} this will render the mandible non-vital, but most studies have shown that even with autogenous marrow grafting, excessive resorption normally occurs, and this technique is not widely practiced today. An alternative technique is to devitalize the mandible by means of cryotherapy,¹¹ and for this the mandibular tray is immersed in liquid nitrogen at -196°C for 10 minutes. The mandible is then allowed to thaw and is refrozen. Studies have shown that liquid nitrogen predictably causes cell death below -20°C and that the most satisfactory results are achieved with a rapid freeze and a slow thawing process. Although described in the literature, the technique does not appear to be widely practiced today. The third technique involves gamma irradiation of the mandible which will successfully devitalize the mandible,^{12,13} and does not appear to result in excessive resorption when it is reinserted and rigidly

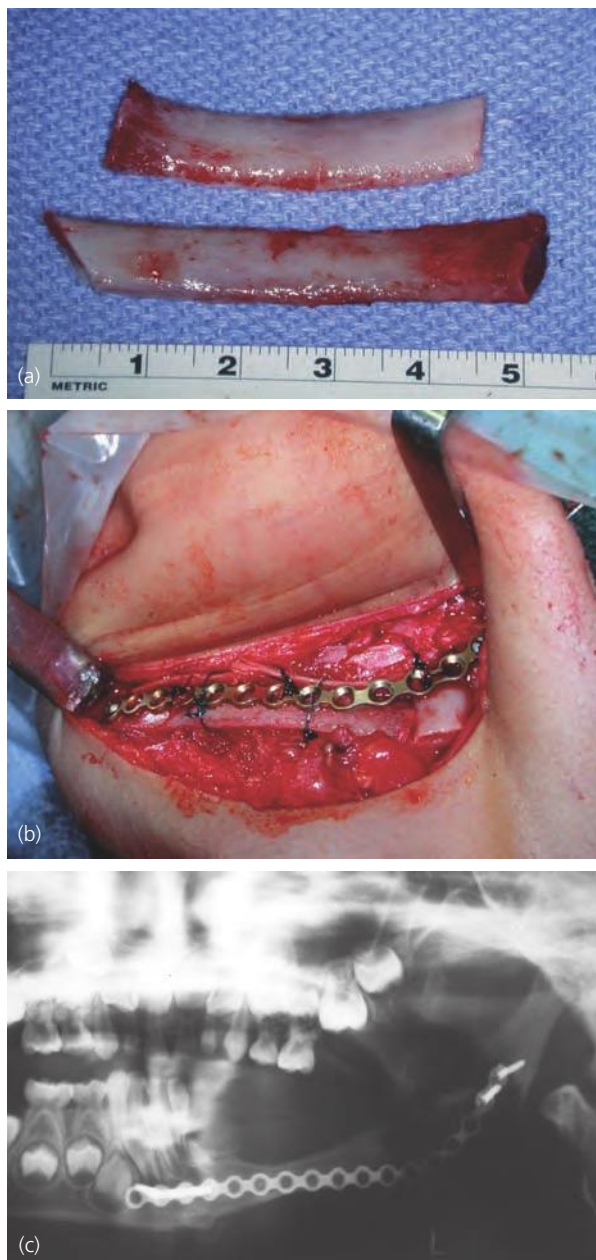


Fig. 51.3 Two ribs (a) used to reconstruct the mandible following resection of a desmoplastic fibroma in a 4-year-old. (b) Same patient at 6 years of age (c).



Fig. 51.4 Corticocancellous full thickness iliac crest bone graft used for secondary reconstruction of a segmental defect of the mandible. The graft is secured to the reconstruction plate with screws.

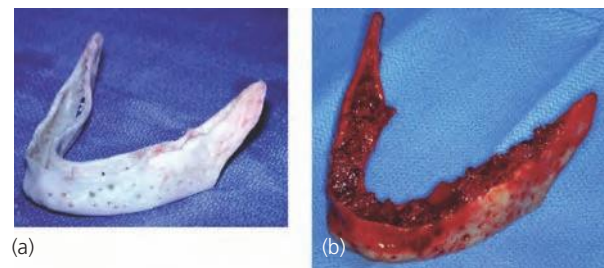


Fig. 51.5 Segmental mandibular reconstruction utilizing a freeze-dried allogenic mandibular crib (a) packed with autogenous bone marrow (b).

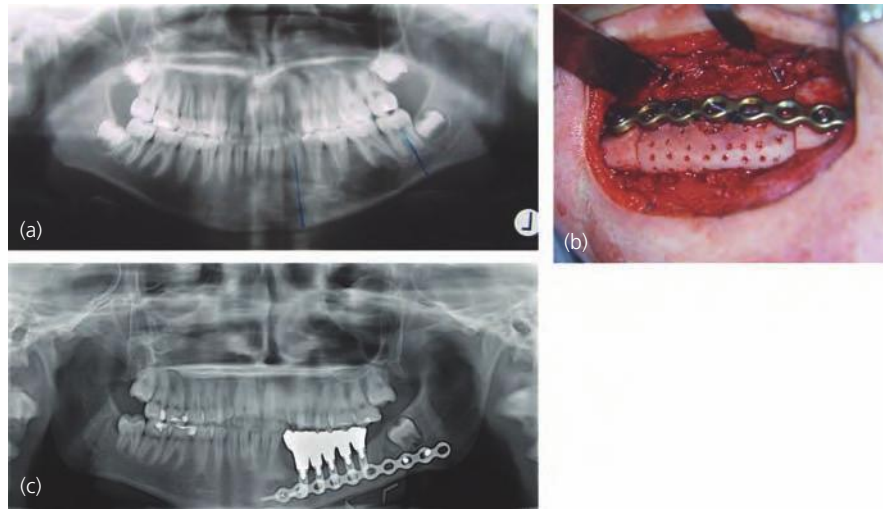


Fig. 51.6 A vascular malformation of the left posterior mandible (a) which was resected, the lesion curetted out and the mandible recontoured as an autogenous tray which was reinserted, secured, and packed with autogenous marrow (b) into which implants were later inserted (c). (Courtesy of Janice S. Lee DDS, MD, MS.)

fixed and grafted. If a source of radiation is locally available, this technique can be carried out in one session while the patient is asleep and at the same time as the initial resection. The mandible can be cleaned, placed in a sterile autoclave bag and receive around 50Gy of radiation in less than 1 hour, in time for reinsertion into the patient. Otherwise, the procedure must be staged. Any of the above techniques may result in a more acceptable contour than using an autogenous graft from another part of the body.

Studies have shown that segmental resections of up to 6cm are associated with a high success rate when reconstructed by any of these techniques. Above 6cm the failure rate starts to increase and when the resected area exceeds 9cm the failure rate from a non-vascularized graft may be as high as 75%.⁷ Fig. 51.7 shows a successful 10cm non-vascularized graft even though it has undergone considerable resorption. Failure usually occurs because of failure of the bone graft to become vascularized and reincorporate, with subsequent infection and wound breakdown.

A number of factors have been identified which may improve or compromise bone healing and the incorporation of bone grafts on reconstruction. Platelet rich plasma (PRP), and possibly other platelet-derived and other growth factors, may enhance incorporation of bone grafts and also may enhance soft tissue healing.^{14,15} Early initial results were very promising, but some subsequent papers have cast doubts on the long-term success of these factors, which have decreased their popularity.^{16,17}

Bone morphogenic proteins (BMPs) have been identified but have only recently become commercially available. They have the ability to induce bone formation by recruiting osteoblastic stem cells to produce bone, thus possibly avoiding a bone graft. In the USA only BMP-2 is currently available,¹⁸ although in Europe BMP-7 (also known as osteoprotein or OP1) is also available.¹⁹ Although these show promise, it is

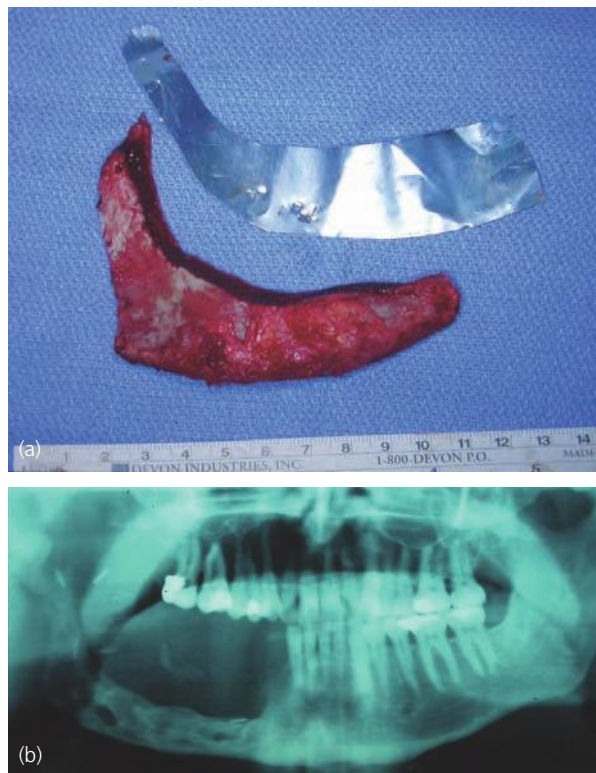


Fig. 51.7 (a) A 10cm non-vascularized full-thickness iliac crest bone graft to reconstruct a hemimandible including the condyle on removal from the hip. (b) Nine months later, after removal of the bone plate.

not known which of these factors is going to prove the most effective, and whether more than one factor is necessary to get the maximum enhancement of bone incorporation, and even whether there is a cascade process involved. At the current time, BMP-2 can only be utilized on an acellular bovine collagen sponge which is compressible and limits its usefulness to procedures such as sinus grafting for implants.¹⁸

When the bone graft has taken successfully, subsequent staged procedures include removal of the bone

plate. This may be necessary to allow subsequent vestibuloplasty and implant insertion. It may be necessary if the patient can feel the plate or screws beneath the tissue, if the plate appears to be in danger of exteriorizing itself, or if subsequent resorption of the bone graft is anticipated due to the effects of stress shielding. Stress shielding remains controversial in the maxillofacial regions. In the long bones, particularly the humerus, this has been shown to be a real issue in that when a rigid bone plate is removed after healing of a fracture of the humerus, it is not uncommon for refracture to occur since the bone plate has shielded the humerus from excessive forces, and therefore it has never fully remodeled and gained its pre-morbid strength. Although some studies have shown that stress shielding may be a factor in the healing of mandibular fractures, other studies have shown that it does not appear to be an issue.²⁰⁻²² Nevertheless, there are some patients who do seem to show excessive resorption following bone grafting, and it is felt that the presence of a rigid reconstruction plate may mean that the bone graft is not subjected to physiological forces, and therefore resorbs at an excessive rate. In these cases, removal of the bone plate may be advisable before the resorptive process becomes too pronounced. Timing of the removal of the plate is often critical, since one cannot remove it prior to the bone graft incorporating, but one wishes to remove it before excessive resorption has occurred. A time of between 4 and 9 months is normally quoted following the grafting procedure before removal of the bone plate. Occasionally bone plates also have to be removed for other reasons, including infection, screw loosening, plate fracture, and exteriorization of the plate or the patient being able to feel the plate through the soft tissues. Occasionally plates also cause discomfort with extremes of temperature. Following removal of the bone plate, it would be possible to carry out vestibuloplasty if necessary and to allow for subsequent osseointegrated implant insertion.

Non-vascularized bone grafting for mandibular reconstruction

Multiple sites have been advocated for obtaining bone that might be used for maxillofacial reconstruction. Some sites, such as the radius and ulna, are rarely used today. Cranial bone is used for post-traumatic reconstruction of the mid and upper face, but quantities are generally insufficient for mandibular reconstruction. The main sites used for non-vascularized mandibular reconstruction are ribs, the iliac bone, and the proximal tibia.

Ribs

Ribs can be used with a cartilage cap from the costochondral junction if they are required for temporo-

mandibular joint reconstruction,²³ or without cartilage for mandibular reconstruction, particularly in children.²⁴ In an adult, a considerable length of rib can be obtained (often up to 15 cm) accepting the curvature of the rib. Ribs are normally taken between the fifth and eighth ribs (avoiding the ribs with considerable attachment of the pectoralis major muscle and also avoiding the free floating ribs). Ribs can be taken from either side with equal ease.

This procedure is normally only carried out under intubated general anesthesia with the ability to administer positive pressure ventilation in the event of a pneumothorax. The surgical procedure involves identifying the rib or ribs to be taken and an incision is made directly over the rib. In females, the incision is normally placed beneath the breast or developing breast so that it will be hidden beneath the breast in the erect position. The incision is taken through the superficial musculature down to the periosteum and perichondrium. No major structures are encountered and only superficial bleeding vessels should be encountered. Once the periosteum of the rib has been identified, it is incised sharply down to the rib itself. If cartilage is to be taken, the incision should continue over the costochondral junction down to the cartilage.

At the anterior end of the incision through periosteum and perichondrium a T-junction should be made to limit the anterior extent of the perichondrial stripping, but to allow 4 or 5 mm of cartilage to be taken. A periosteal elevator is now used to strip the periosteum off the superior surface of the rib and cartilage. With great care, one then turns the corner and starts stripping periosteum from the superior and inferior surfaces of the rib. It must be remembered that the neurovascular bundle supplying the rib runs in a groove on the inner aspect of the inferior surface of the rib. It is crucial that the dissection be subperiosteal at all times. The dissection is then continued with care on the inferior surface of the rib. Although normal periosteal elevators can be used for this dissection, special instruments are available, such as the elevator (Fig. 51.8).

Once the subperiosteal dissection has been completed and the rib isolated, an instrument such as the appropriate Doyen retractor can be placed under the rib and the subperiosteal dissection confirmed and continued (Fig. 51.9). The rib is always sectioned at the anterior end first. If this is in cartilage, it can be carried out with a scalpel, but an instrument must be placed under the cartilage to prevent penetration of the pleura. If the anterior sectioning is to be in rib bone itself, then a guillotine or rib shears can be used. Once the anterior cut has been made, the rib can then be raised and, with ease, the subperiosteal dissection can continue as far posteriorly as required for the length of rib needed. Once the posterior limit has been defined, the posterior cut through the rib is normally made with a guillotine, which protects the underlying structures (Fig. 51.10). Once the rib has been taken, it is laid on one side and covered with moist gauze.

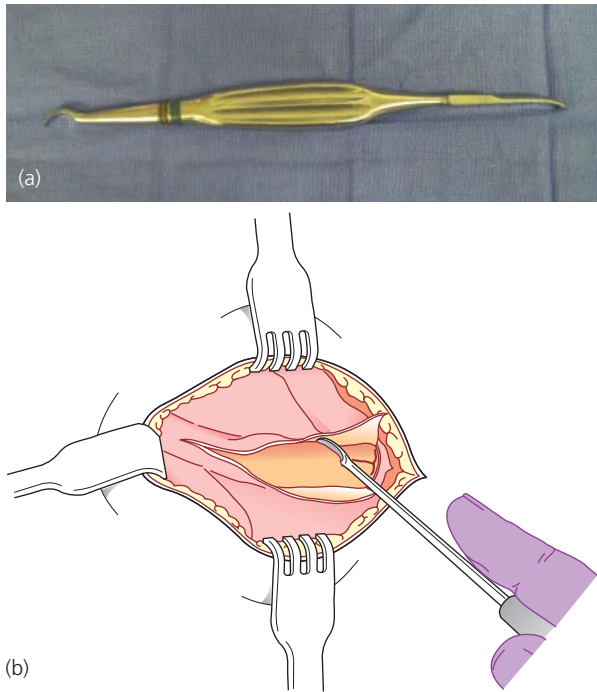


Fig. 51.8 (a) An Overholt periosteal stripper to raise periosteum around a rib. (b) Note the T-shaped incision through the periosteum.

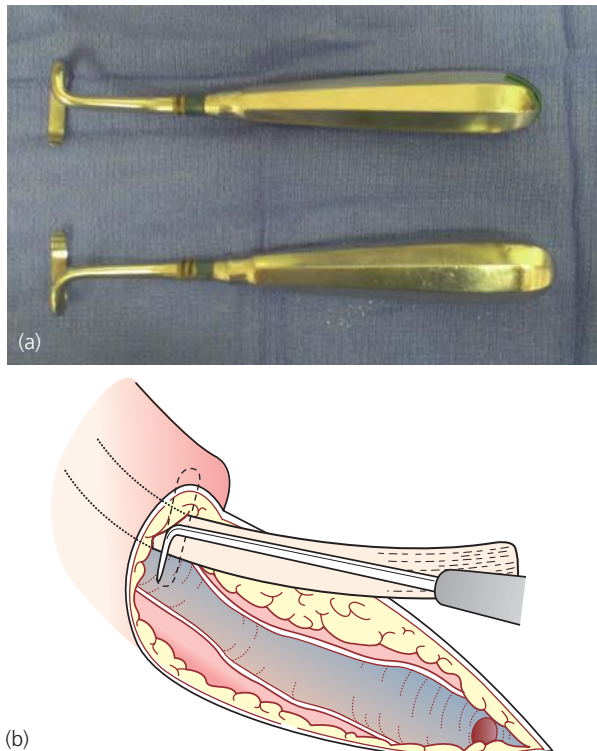


Fig. 51.9 (a) Doyen retractor (b) placed under a rib to elevate periosteum, protecting the underlying pleura.

A test is now made for the presence of a pneumothorax, and since the procedure is carried out under intubated general anesthesia, this is most easily carried out by filling the chest wall cavity with warm saline and allowing the anesthesiologist to fully inflate the chest (a Valsalva maneuver). Any bubbling will indicate a pleural leak. If the leak is very small, it may be oversewn directly, but the sutures should

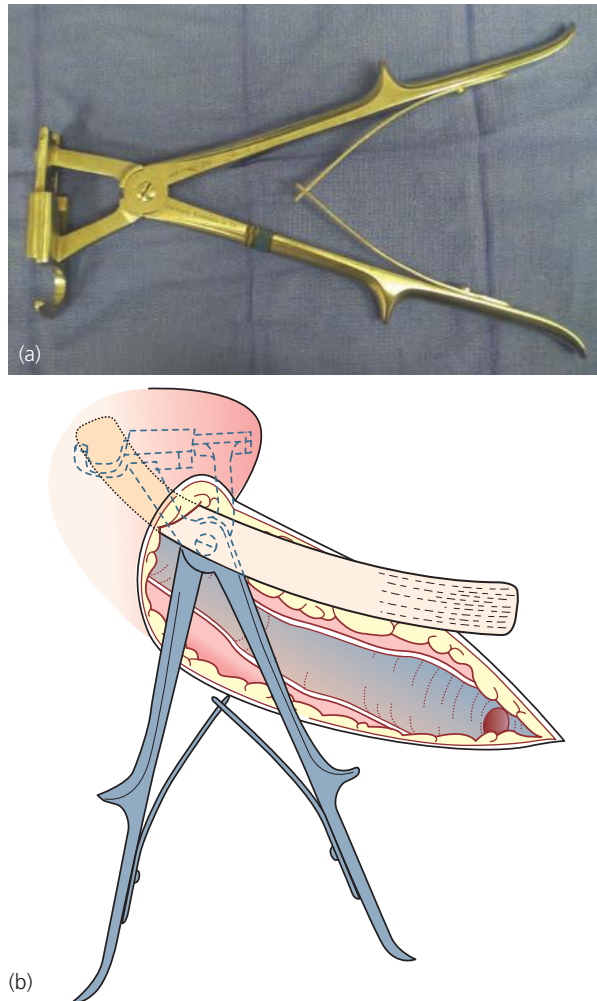


Fig. 51.10 (a) A guillotine rib (b) cutter used to cut the posterior end of the rib.

only be tightened down in conjunction with another Valsalva maneuver to drive all the air out of the pleural cavity. If the leak is a little larger, a drainage tube can be placed, and then a purse-string suture placed around it; the drainage tube is withdrawn as the purse-string suture is tightened on another Valsalva maneuver and this may give good closure of the hole (Fig. 51.11). If closure cannot be obtained, then an underwater sealed drain must be inserted, and this will normally go from the opening and emerge high in the chest in order to drain air. This drain will be placed to low-volume wall suction and left in place for 24–36 hours.

If more than one rib is required, then the adjacent rib can normally also be harvested in the same way through the same incision. If more than two ribs are required, then it is necessary to leave alternate ribs so that the patient does not develop a flail chest. Sometimes this requires a second chest wall incision, but sometimes it can be obtained through the one incision if it is correctly placed. Closure is carried out in layers and postoperatively an upright chest radiograph is obtained as soon as possible to check for the presence of a pneumothorax. With any pneumothorax occupying more than about 15% of the lung

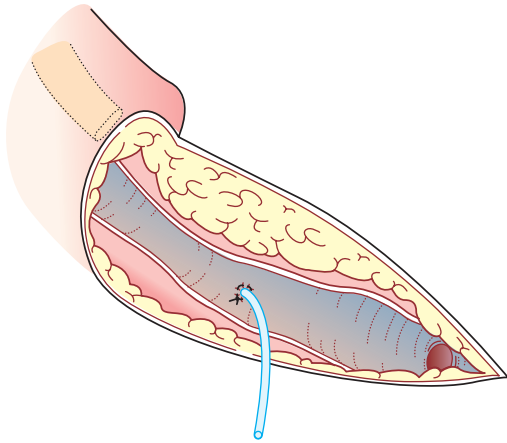


Fig. 51.11 A pursestring suture round a drainage tube to close a small pneumothorax.

volume, an opinion should be obtained with regard to placement of an underwater sealed drain. At the completion of the surgery, a long-acting local anesthetic, either given as a single injection or via a subcutaneous infusion device, can make postoperative physical therapy and chest movement more acceptable to the patient.²⁵ Chest wall discomfort is to be expected on deep inspiration for some time after this procedure. Postoperative physical therapy with inspiratory spirometry is often indicated.

When the wound is closed, attention can now be turned to the harvested ribs. If it is required to include costochondral cartilage, this can now be shaped with a scalpel to leave a cap of about 2 mm of cartilage on the end of the rib (Fig. 51.12). This approximates well to a temporomandibular joint. If more cartilage is left, there is a risk of it separating from the rib on pressure, and also more risk of overgrowth occurring from the cartilage. The rib can be effectively straightened with a rib morcellizer, but care must be taken not to splinter the rib. Ribs from younger people will straighten more easily than ribs from older patients. When securing ribs in place with screws, they generally require a washer of some kind to prevent the screw from splintering the rib. In some cases it is preferable to use a bone plate and put the plates and screws through the plate, or even titanium mesh and place the screws through the mesh, so the pressure is spread more evenly.

Iliac crest

Anterior iliac crest

This donor site is the workhorse of the reconstructive oral and maxillofacial surgeon.²⁶ The landmark of the anterior iliac spine can normally be identified by palpation on all but the most obese patients and the outline of the crest of the ilium can also be mapped out on the skin. The initial incision should not be placed right over the crest since this can be uncomfortable postoperatively with some clothing. It is also advisable to hide the incision below the crest of the ridge in



Fig. 51.12 Two ribs with cartilage caps to reconstruct the temporomandibular joints.

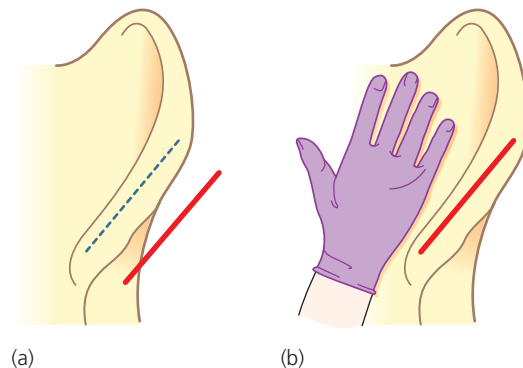


Fig. 51.13 Site of the incision to approach the anterior iliac crest. Note the palm of the hand to provide tension so the incision is over the iliac crest (a) so that the final scar is below the iliac crest (b, c).

connection with various items of clothing. The normal technique is to pull the skin upwards by depressing the soft tissues superior to the crest with the palm of the hand. This will allow an incision to be made directly over the crest of the ilium, but when the tension is released from the incision, it will lie 3–4 cm below the iliac crest (Fig. 51.13). The incision can be made directly through skin, subcutaneous tissue, Scarpa's fascia, any superficial rectus muscle, and down to the iliac crest. If one approaches the iliac crest directly, virtually no major muscle attachments are encountered. Incisions from the lateral side will

entail sectioning of the gluteal muscles. There is a number of superficial sensory nerves passing over the crest of the ridge, and in particular, the iliohypogastric nerve is normally at risk and often leaves patients with a palm-sized area of paresthesia on the lateral side of the iliac crest. The lateral cutaneous nerve of the thigh is normally placed more anterior.

Once the crest has been identified, the incision can be taken down to the bone itself and subperiosteal elevation can be commenced. In most cases, it is advisable to formally identify the anterior limit of the iliac crest and the origin of the inguinal ligament. As one goes more posteriorly, the tubercle is identified some 5–6 cm posterior to the anterior spine, and this is the most productive site for harvesting bone marrow (Fig. 51.14). If medial cortex of the ilium is required, then the subperiosteal dissection only needs to be continued on the medial side, and this strips away easily, since the iliacus muscle is not attached to the ilium superiorly, and there are no other muscle attachments or vital structures in this area. On the lateral side, however, the gluteal muscles are attached, and if full-thickness grafts are required, these will need to be stripped away resulting in short-term discomfort and stiffness.

To obtain marrow

This is normally obtained directly over the iliac tubercle, since this is the widest point of the ilium (some 5–6 cm posterior to the anterior iliac spine), although it can be obtained elsewhere in the ilium. A lid from the superior surface of the iliac crest is normally raised, centered over the tubercle. This can take the form of bone cuts anteriorly and posteriorly joined by a cut directly along the middle of the iliac crest to

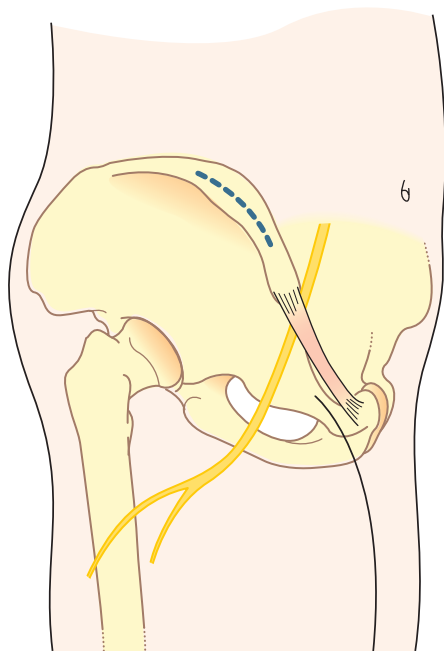


Fig. 51.14 The tubercle (with dotted line over it) has the largest source of cancellous bone in the anterior ilium. It is 5–6 cm posterior to the anterior superior iliac spine.

raise small trap doors medially and laterally to expose the crest. If this is to be carried out, periosteum should remain attached laterally and medially so that these trap doors do not become totally free grafts. On completion of the procedure, these trap doors can be replaced. Cuts can be made with chisels, drills, or reciprocating saw. Alternatively, a single coffin lid type bone flap can be raised based either laterally or medially; again, periosteum should be retained at the site of the hinge so that it does not become a totally free flap (Fig. 51.15). Marrow is now exposed and can be curetted with suitable size curettes. One should start with the largest curette possible to fit between the lateral and medial plates of bone, and curettage may need to be quite aggressive. As one descends inferiorly, the size of curette may need to be decreased to fit between the lateral and medial plates. Care should be taken to attempt to avoid perforation, which normally occurs on the medial side. Small perforations can be accepted, the main risk is to underlying structures. Amounts vary, but 20–30 ml of compressed bone can normally be obtained from the average ilium. If necessary, both left and right sides can be used at a single operation. On completion of taking the marrow, the bone flap can be replaced and is normally self-retaining, though if required a suture can be placed through holes drilled in the bone. The marrow that is taken is normally placed in a container and covered with a moist gauze. Closure is in layers, starting with the periosteum and including Scarpa's fascia, and the wound does not normally need to be drained.

Corticocancellous blocks

The anterior iliac crest lends itself to taking corticocancellous blocks for more extensive reconstruction. If bone is to be taken from the medial surface of the iliac crest, then the periosteum and iliacus muscle are retracted with an abdominal retractor and the area of bone to be taken is outlined with a drill or reciprocating saw. The inferior margin of the bone to be taken only needs to be outlined through the cortex. The bone to be taken will generally include a portion of the iliac crest, and so the anterior and posterior cuts are made through approximately 50% of the iliac crest, and are then joined along the center of the iliac crest. The block of bone is then removed utilizing appropriate sized straight and curved chisels. In this way, copious amounts of bone can be removed in strips approximately 2–3 cm in width (Fig. 51.16). Strips wider than this tend to fracture. On completion, a suction drain is often placed over the harvesting site and left in place for 24–36 hours, or until drainage is less than 20 ml in 8 hours.

For major mandibular reconstruction, through and through corticocancellous blocks can be taken to include both lateral and medial cortices of the ilium, leaving a hole in the ilium. For this, extensive periosteal stripping needs to take place on both the medial and lateral surfaces to identify the iliac ligament,

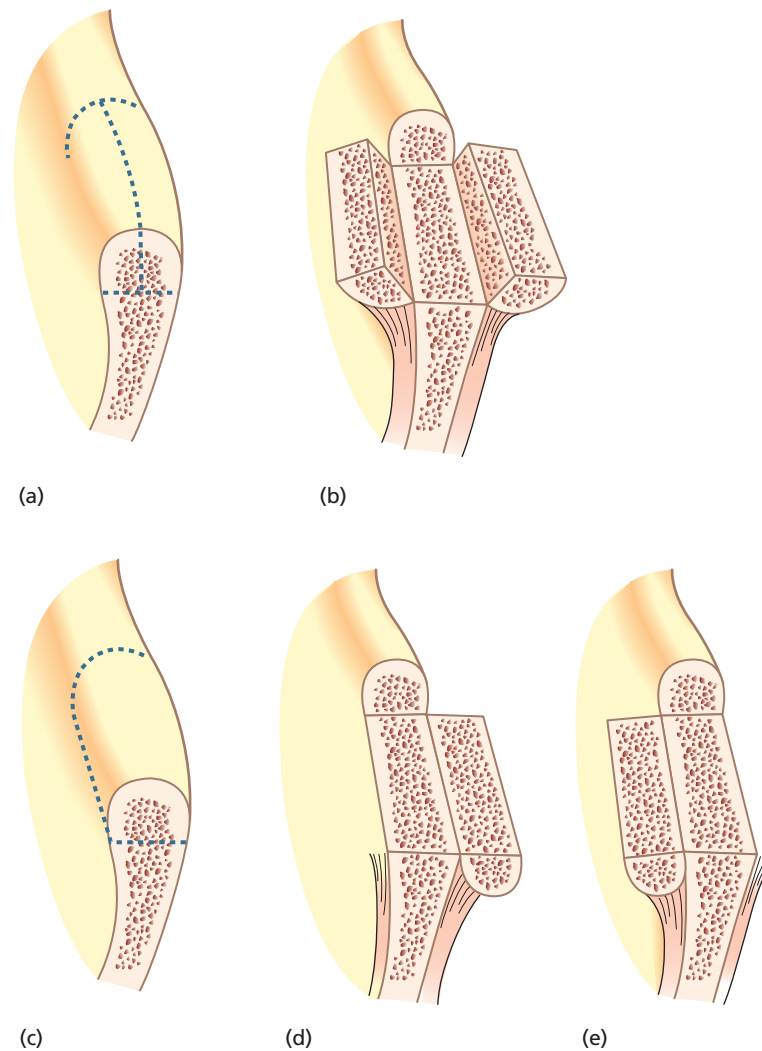


Fig. 51.15 The variety of “trapdoor” and “coffin lid” type incisions, based laterally and medially, that are used to access the cancellous bone in the iliac tubercle.

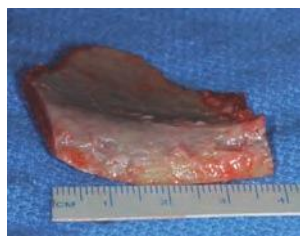


Fig. 51.16 Corticocancellous strips of bone taken from the medial surface of the ilium.

anterior notch, and posteriorly towards the sacroiliac joint, which should not be entered. This defines the area of bone that can be taken. A template is often helpful depicting the shape of bone required, and this can be made from sterilized aluminum from a soft drink can. A flexible plastic surgical ruler can also be used. The easiest way to take the size of block required is to remove the associated iliac crest. If this is carried out, the iliac crest is removed as a single block, starting at least 1 cm posterior to the anterior superior iliac spine, to prevent its fracture, and proceeding posteriorly, 1 cm posterior to the posterior limit of the block. In this way, the crest can be removed and then wired or plated back in place once the

block has taken, and it will mortise into place nicely (Fig. 51.17). The corticocancellous full-thickness block can now be taken from above, with appropriate drills and saws. However, this does weaken the crest considerably, and may cause more postoperative morbidity. A preferable technique is to leave the iliac crest intact and to take the full-thickness portion of bone from below it (Fig. 51.18). These blocks are normally taken from the ipsilateral side of the patient, since the curvature is more appropriate to the mandible on the same side. If the crest is to be left intact, the approach is normally from the medial side where access is better, and is with a combination of reciprocal saw, oscillating saw, and drills. Once the block has been taken, some marrow can usually be obtained from around the edges, and is often helpful in the reconstruction to augment any defects.

The wound is normally drained with a suction drain and closure is then carried out in the normal way in layers. Long-term postoperative radiographs show that a corticocancellous block less than 2–3 cm will normally form new bone, whereas a larger one will leave a permanent defect in the ilium, although herniation does not occur (Fig. 51.19). However, if it

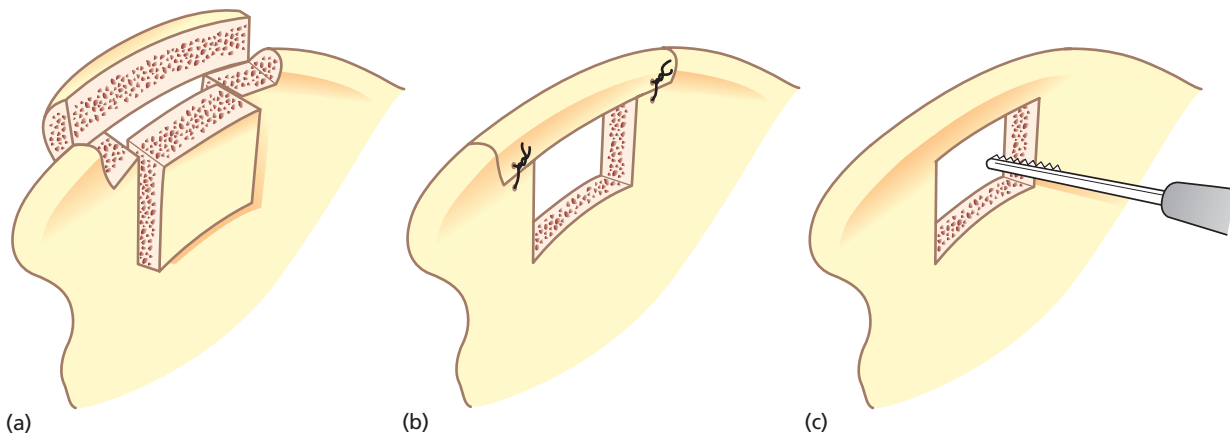


Fig. 51.17 (a, b) The technique of removing and rewiring the iliac crest to remove corticocancellous blocks of bone from below the crest, similar to the one seen in Fig. 51.4. (c) By using oscillating and reciprocating saws the crest can often be left intact for greater strength and decreased morbidity.

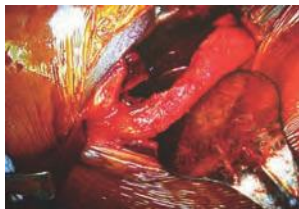


Fig. 51.18 The residual defect seen clinically.

is felt that it may be necessary to go back again to obtain more bone for subsequent reconstructions, it is not necessary to drain the site and in this case blood will accumulate in the defect and, if periosteum is intact, may form new bone that can be reharvested. This does, however, run the risk of extensive hematoma formation. A drain placed in a full-thickness donor site will normally drain a total of 100–200 ml of blood over 24–36 hours, and should be removed when drainage is below 20 ml in 8 hours. It is often helpful not to remove the drain until the patient has been mobilized, since assuming the upright posture can result in drainage of additional pockets of fluid.

Large corticocancellous grafts can be harvested in this way and a single block can include the body, ascending ramus, and even the condyle (see Fig. 51.7). The block obtained is normally shaped to fit the defect, and the cortex is thinned with a large bur and perforated to aid revascularization.

Posterior iliac crest

The posterior iliac crest has been advocated for obtaining bone for orthopedic procedures on the spine, but has also been suggested as a donor site for oral and maxillofacial surgical procedures.²⁷ Large amounts of marrow can be obtained (up to 40–50 ml of compressed marrow from each side), and small corticocancellous blocks can be obtained. The incision runs over the posterior iliac crest which can normally be palpated and starts approximately 3 cm lateral to the midline of the center of the spine, and as it goes superiorly, it moves laterally at about 15° to stay over the posterior iliac crest. The incision is deepened



Fig. 51.19 Radiograph of ilium 6 months post iliac crest graft showing residual asymptomatic defect.

down to the posterior crest, and no major structures are identified en route, except for the cluneal nerves, which do supply the ipsilateral skin around the anus itself and can give an area of paresthesia in this region. They can normally be retracted with the soft tissues.

The main complications of this technique are possible involvement of the sacroiliac joint, and postoperative back discomfort, which is often difficult to separate from any other cause of back pain. The main disadvantage of this technique, however, is that it is necessary to turn the patient over to obtain the bone, and this is technically difficult to perform (particularly on an obese patient), and may provide some difficulties in staging the procedure and keeping the recipient site sterile if it has been opened first. This wound does not normally need to be drained.

Tibia

The proximal end of the tibia has been advocated as a donor site for bone marrow, and small pieces of cortex only. Both lateral²⁸ and medial²⁹ approaches have been suggested. Both approaches are fairly straightforward. In the lateral approach, Gerdy's tubercle is identified and the incision carried down at 45° from it inferiorly for 2–3 cm and by blunt dissection can be carried straight down to the tibia. There are normally no major vessels or nerves encountered in this approach. Similarly, the medial approach entails a

vertical incision 1 cm medial to the anterior prominence of the tibia, and blunt dissection down to the tibia itself. The major complication from tibial bone harvest is tibial plateau fracture, which has been noted in connection with maxillofacial procedures, and can be a major long-term complication. For this reason, no bone should be taken (either cortical or cancellous) within 1 cm of the tibial plateau.

An oval window is made through the cortical bone of the tibia. This is normally approximately 2 cm by 1 cm, and is performed with a drill (Fig. 51.20). It should be oval without sharp edges to prevent any stress fractures developing from the corners of the window. The window can either be removed completely (and used as a cortical graft), or replaced at the end of the procedure, or swung out on attached periosteum, either laterally or medially. Once through the cortical bone, marrow can be curetted with ease,



Fig. 51.20 Oval window in the lateral tibia and bone removed.

although one should stay at least 1 cm below the plateau. Twenty milliliters of compressed marrow can often be obtained from the tibia, although it is always of a more fatty appearance than marrow from the hip. Nevertheless, it does perform well clinically when used in a carrier tray for mandibular reconstruction.

The wound is normally repaired in layers. Post-operative weightbearing precautions vary widely. In practice, the medial epicondyle of the tibia takes approximately 55% of the body weight, whilst the lateral epicondyle takes approximately 45% of the weight. Therefore, one would assume that tibial plateau fractures were more likely to happen from the medial approach, and more weightbearing precautions may need to be taken. Both in orthopedic and oral and maxillofacial surgery, however, weightbearing precautions following tibial grafting vary widely, from some centers advocating no precautions at all and letting the patient walk immediately, to those that provide a cane, or walker, or even instructing the patient not to be weightbearing on the knee for 3–4 weeks. In practice, the complication rate does not seem to vary, so it would appear that in most cases, minimal weightbearing precautions are necessary.

Bone-containing microvascular free flaps

An alternative technique for reconstruction following segmental resection is with a microvascular free flap. Possible donor sites include the fibula (Fig. 51.21),^{30–32} iliac crest³³ or scapula.³⁴ The radius was previously



Fig. 51.21 (a) Mandibular reconstruction including the condyle with a segmental free fibula bone graft. (b) It was subsequently secondarily restored with osseointegrated implants following thinning of the intraoral soft tissue component of the graft.

used for this purpose, but it is associated with a high incidence of subsequent fracture of the radius, and this technique is rarely employed today.³⁵ To be successful, there need to be suitable feeding vessels; these would normally be the facial vessels, but, if necessary, other vessels can be used. If the length of the vascular pedicle is in doubt, intervening vein grafts can be used to enable successful anastomosis. Conceptually, the advantages of a microvascular reconstruction are:⁷

- soft tissue can be transferred at the same time as the bone;
- all procedures can be performed primarily;
- this technique can be used in areas of poor vascularity, including previous radiation therapy;
- success rate remains high for longer-span reconstructions.

The disadvantages of reconstruction with a vascularized graft may include:

- The bulk of soft tissue transferred with the bone is often too thick for subsequent prosthodontic reconstruction and subsequent procedures must be carried out to thin the soft tissues.
- The bulk of bone is often suboptimal for later implant insertion, though the initial graft can be augmented or modified in some way to make implant insertion more feasible. For example, a



Fig. 51.22 Cross-section of a fibula showing the thick cortical bone with a smaller area of predominately yellow, fatty, marrow with an overall diameter of 10–12 mm.

fibular bone graft can be doubled over on itself to create a double thickness (a single fibula is approximately 10–12 mm in thickness) (Fig. 51.22) to ensure that more bone is present for implant insertion.

Reconstruction involving the mandibular condyle

When the condyle is resected as part of a surgical procedure, another layer of complexity is added to the reconstructive process. The mandibular condyle is a complex articulation involving both hinge and sliding mechanisms with an intervening meniscus and muscle attachment to the condyle and meniscus themselves. It is not possible to reconstruct all elements of the joint, and therefore any reconstruction must be suboptimal as far as function is concerned. Nevertheless, a reasonable joint reconstruction is possible.

Rib grafting

An autogenous rib, including a 1–2 mm of costal cartilage, has been used for many years to reconstruct the condyle, and long-term follow-up is available showing very satisfactory long-term remodeling of the rib into a very acceptable condyle (Fig. 51.23).^{36–38} In younger patients the rib appears to grow in harmony with the rest of the face, although cases of overgrowth of the reconstructed condyle have been reported, and a growth-arresting procedure is occasionally necessary. This usually consists of a minimal arthroplasty to remove the active condylar and subchondral region.

Alloplastic condyle

A number of alloplastic condyles have come and gone over the years, and a small number are still available.^{39,40} In general, the smaller the joint to be replaced, the more difficult it is to make a successful alloplastic joint and the more risks and complications appear to

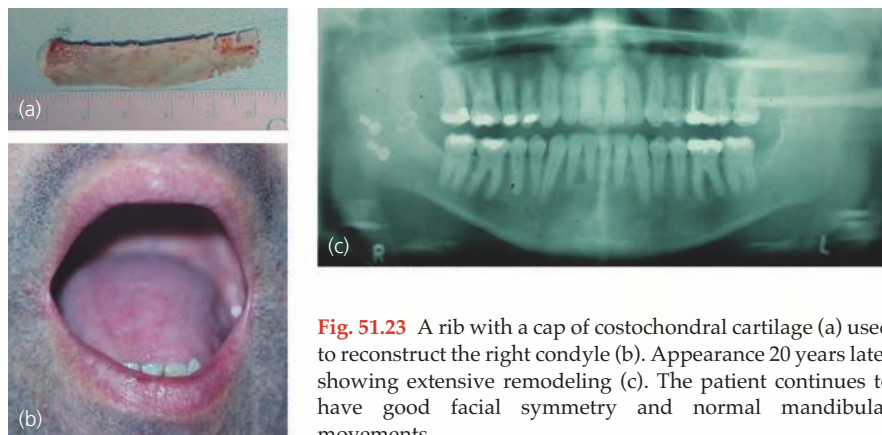


Fig. 51.23 A rib with a cap of costochondral cartilage (a) used to reconstruct the right condyle (b). Appearance 20 years later showing extensive remodeling (c). The patient continues to have good facial symmetry and normal mandibular movements.

be associated with the process. Therefore, for the hip joint, good long-term success is reported, but as the joints get smaller, the reconstruction becomes more difficult. Additionally, the mandibular condyle, with its combination of hinge and sliding motion, has proved extremely difficult to reproduce in an alloplastic replacement. The indication for alloplastic replacement in most cases is for function rather than for pain. Most replacements include both the glenoid fossa and the condyle, and it is generally recommended that both are placed at the same time. In some cases, the alloplastic joint is a stock replacement, whilst in other cases it is custom made from a computed tomography (CT)-generated three-dimensional model. Most implants are constructed of titanium with or without an element of high impact polymer (Fig. 51.24). Long-term results from these implants are generally not currently available.

Replacement with a metatarsal

Condylar reconstruction with the second or third metatarsal bone has been reported with some success, but long-term results are not available, and in some cases the deformity to the foot has been felt to be unacceptable, with collapse of the remaining tarsal bones and a subsequent discrepancy in foot and shoe size.⁴¹ Nevertheless, the size of a metatarsal is very compatible with that of a condyle, and surgically it makes an excellent replacement.

Sternoclavicular joint

A full-thickness, or split-thickness, sternoclavicular joint has been used as a condylar replacement, with or without an attached portion of the sternum to replace the glenoid fossa.^{42,43} The advantages are said to be that the joint is of the appropriate size and it is a joint with a meniscus between the joint surfaces. Disadvantages include the risk of pneumothorax as well as damage to the nerves and vessels at the root of the neck, particularly the brachial plexus.

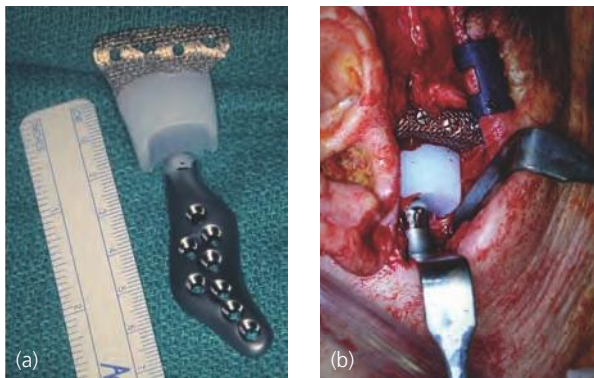


Fig. 51.24 (a) A custom-made TMJ prosthesis constructed on a three-dimensional model. (b) On surgical placement.

Microvascular reconstruction of the condyle

Microvascular reconstruction of the condyle is possible using a fibula.⁴⁴ The fibular bone is normally rounded off to fit in the glenoid fossa, and a temporalis fascia flap can be placed to separate the fibula from the glenoid fossa, since there is no cartilage on the surface of the fibula. This method of reconstruction is obviously more complex, more time consuming, and associated with a longer hospital stay, but can be used, particularly for patients who have received preoperative radiation therapy.

Staged techniques

Reconstruction of the mandibular condyle, ramus of mandible, and body of mandible is associated with particular problems. Primary reconstruction with a non-vascularized bone graft is associated with a high failure rate, normally from wound breakdown in the mandibular body section associated with thin intraoral mucosa and wound contamination. Breakdown rarely occurs over the ascending ramus of the mandible or condyle area since these are not normally contaminated by saliva and have covering from the medial pterygoid muscle medially and the masseter muscle laterally, providing a bulk of vascularized soft tissue. However, secondary reconstruction, even carried out after 6 or 8 weeks, is problematic, since it is difficult to gain secondary access to the glenoid fossa to insert a non-vascularized graft, such as a rib graft secondarily. Although temporary alloplastic condyles are available, they are sometimes difficult to insert, and are very often difficult to remove, since fibrosis often forms around the condyle head making subsequent removal and replacement with a rib graft difficult (Fig. 51.25). For this reason, a staged reconstruction has been advocated.⁸ In this technique, following resection, immediate reconstruction of the condyle and ascending ramus is carried out with a rib graft (Fig. 51.26). This maintains the articulation of the glenoid fossa, and generally survives because of the vascularity of the surrounding musculature and the adequacy of the intraoral closure. Temporary reconstruction of the body of the mandible is with a titanium reconstruction plate. Some 6–8 weeks later, the body of the mandible is then reconstructed from an extraoral approach, as detailed previously, using corticocancellous blocks from the ilium. In this way, the reconstruction of both the condyle and the body of the mandible is optimized, and high success rates have been reported.

Radiation therapy

Radiation therapy to the mandibular area complicates reconstruction. Radiation therapy causes an endarteritis, which decreases the vascularity, cellularity, and

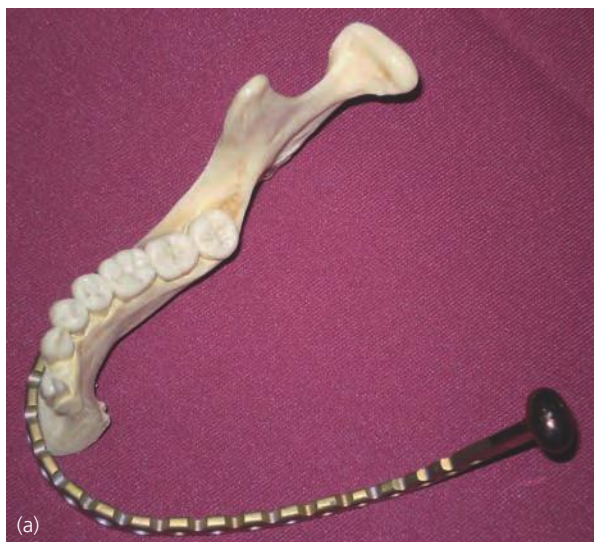


Fig. 51.25 A temporary alloplastic titanium condyle attached to a reconstruction plate. Removal of the condyle for secondary autogenous reconstruction can be difficult due to fibrosis and scarring.

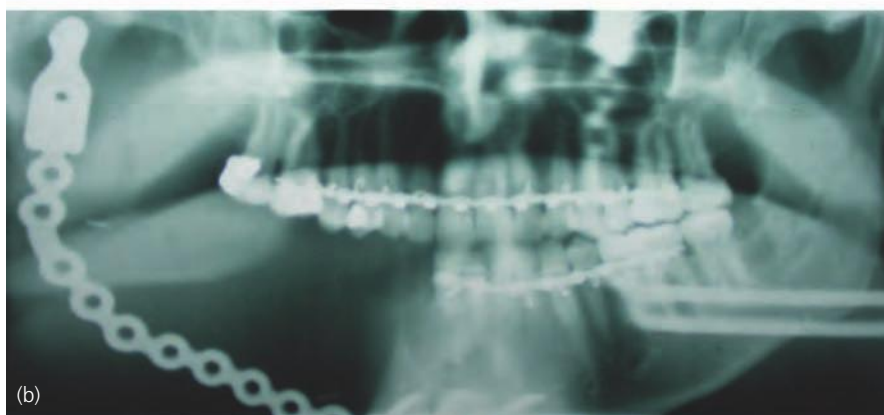


Fig. 51.26 A rib graft attached to a reconstruction plate for primary reconstruction of the condyle. The mandibular body will be reconstructed secondarily with a corticocancellous block graft from the iliac crest.

tissue oxygenation of the associated tissues. The endarteritis and hypocellularity usually commence some 3–4 months following the radiation therapy and are continuous in nature; they are well established by 6 months. The tendency is for the endarteritis and hypocellularity to become more pronounced with time. There is no evidence that it is a reversible process. There is, however, a so-called “golden window” up to 3–4 months following radiation therapy during

which bone grafting can be carried out in a normal way. From a practical point of view, the immediate local complications of radiation therapy will often preclude the bone graft during the first 6 weeks following radiation therapy, while the mucositis, etc. resolve, and therefore the “golden window” is really from 6 weeks following radiation therapy to 3–4 months following radiation therapy. After this time, the failure rate from non-vascularized bone grafts is generally felt to be unacceptably high due to the avascular tissue bed. If it is intended to carry out mandibular reconstruction in an area that has received more than 55 Gy of radiation, it should either be with a vascularized graft⁴⁵ or possibly following a course of hyperbaric oxygen therapy to increase the vascularity and cellularity of the tissue bed. There is good evidence that hyperbaric oxygen (30–40 dives at 2.4 atmospheres pressure for approximately 90 minutes) can produce a tissue bed in which a non-vascularized bone graft can survive.^{46,47} There is no evidence that hyperbaric oxygen can reactivate a dormant malignant tumor or cause recurrence of a dormant malignant tumor. If the bone graft takes satisfactorily, implants can subsequently be inserted.⁴⁸

In the case of a vascularized graft, if previous radiation therapy has been given, although the nature of the recipient vessels may be changed and they may have a greater element of fibrosis, successful

anastomosis, and re-establishment of vascularity are possible.

If a microvascular graft is inserted immediately and then radiation therapy given subsequently, then the microvascular graft will be affected by radiation therapy just like all the other tissues, and subsequently implant insertion may be compromised. If, however, the microvascular graft is inserted after radiation therapy, then it will not have been subjected to irradiation, and, providing the vascularity is adequate, implants can be inserted with safety even though the surrounding tissues may have been irradiated.

Bisphosphonate therapy

The use of medically administered bisphosphonates, either intravenously, in the management of bony metastases in malignancies such as multiple myeloma, breast cancer, and prostate cancer, or oral forms of bisphosphonates, used mainly for the management of postmenopausal osteoporosis, have been shown to decrease bone turnover, initially by inhibiting osteoclast function and later also inhibiting osteoblast function. Although of potential value in some conditions where bone turnover may be excessive, their use has compromised some bone reconstruction and grafting procedures; if a patient is taking one of these medications, its possible effect on any bone graft or mandibular reconstruction should be evaluated.⁴⁹ With the intravenous forms, discontinuing the medication normally has no effect since the bisphosphonate continues to be excreted by the bone on to its surface for up to 10 years. However, with the oral forms, it is possible that discontinuing the medication for 6–9 months will allow bone turnover to recover, and if the bisphosphonates are not recommenced for 6 months after any bone grafting, this may allow the graft to incorporate and remodel to a certain extent. Bisphosphonate administration will slow down, or prevent, bone remodeling.

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Chapter 52

Tissue Engineering and Reconstruction

Henning Schliephake

The aim of tissue engineering is the enhancement of tissue repair by regeneration of local tissues adjacent to a defect and/or *ex vivo* generation of artificial tissue hybrids that can act like grafted tissue in the defect area after implantation. In general there are two major avenues through which this aim is approached: growth factors that mediate tissue proliferation and differentiation are administered to accomplish defect repair, or cells – in particular adult stem cells generated from the tissue to be replaced and expanded *in vitro* – are implanted into the defect to form new tissue. Both approaches have been extensively explored in experimental and preclinical settings. Clinical applications are, however, rather sparse due to a number of biological and technical issues that are associated with human use. This chapter outlines present applications of tissue engineering techniques in maxillofacial reconstruction.

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Introduction

Tissue engineering in reconstructive surgery encompasses all measures that are suitable to enhance tissue regeneration *in vivo* in order to obviate the need for autogenous grafts. The term tissue engineering has been defined as “the application of the principles and

methods of engineering and the life sciences towards fundamental understanding of structure/functional relationships in tissues...and the development of biological substitutes to restore, maintain, or improve tissue functions”.¹ Initially, activities in this area were mainly focused on cell-based approaches aiming at the generation of tissue-like constructs by combining *ex vivo* expanded cell populations with various types of scaffolds. Today, the field of tissue engineering has expanded tremendously, in that not only cells and scaffolds but also growth factors, controlled release carriers, engineering of biomaterials and many other areas of basic and applied research are considered to be part of the field of tissue engineering.

The interest and attention that this rapidly developing area has received are based on the vision that the growing understanding of tissue healing and the achievements of biotechnology will be of profound therapeutic relevance. In clinical reality, reconstructive surgery has arrived at a standard of care that allows for repair and restoration of the vast majority of patients with established techniques; the challenge of tissue engineering in clinical treatment is the reduction of surgical morbidity by the application of biological signals or bio-artificial components cultivated from the patient's own cells, that can replace the lost body part or accomplish its repair without the need for autogenous tissue transfer.

During the past decade, the principles of tissue engineering have also been applied to oral and maxillofacial surgery. Bone tissue is a major focus, but oral mucosa, cartilage, and salivary glands have also received some attention. All approaches in tissue engineering have to consider basic principles of tissue biology and the specific requirements of those tissues that should be replaced or repaired. These are the conditions for cell differentiation and morphological organization as well as the interactions between different types of cells necessary for repair. They form the basis for successful tissue engineering in reconstructive surgery.

Basic principles of tissue engineering

Regeneration of tissues is a complex and highly orchestrated process that, nevertheless, proceeds along a rather uniform pathway including the three well known steps of inflammation, proliferation, and remodeling. During this process biological signals accomplish the increase in cell numbers that fill the defect or cover the wound. At the same time, specialization of the newly formed tissue occurs through morphogenic signals which induce the tissue specific differentiation. Most of the biological signals that increase both proliferation and induce differentiation of cells are conveyed by polypeptide growth factors. These growth factors are supplied by either local cells or by circulating cells and blood components such as macrophages and platelets. Moreover, growth factors originate from the extracellular matrix (ECM) where they are stored and released during tissue remodeling and repair. During this process, the extracellular matrix serves an additional purpose by providing a three-dimensional scaffold for the migration of cells and their arrangement in a tissue-specific manner. In this way, cells, signaling molecules, and the ECM are closely linked and form the basis of tissue homeostasis and regeneration *in vivo*.

When these three components are transferred to the *in vitro* environment of tissue-engineered constructs, the extracellular matrix is replaced by synthetic or natural scaffolds which are used to accommodate and arrange the cells in a three-dimensional fashion. The triad of cells, signals, and scaffolds thus makes up the "classic" tissue engineering triad. However, beyond these three components, angiogenesis and vascularization play an important role in cellular behavior and tissue repair, not only because blood supply is necessary for cell survival and development, but also because vessels provide a reservoir of undifferentiated perivascular cells that are recruited during tissue repair. Therefore, it is entirely the combination of at least four major factors that contribute to tissue regeneration and, thus, to the success of tissue engineering approaches: cells, growth factors, scaffolds, and vascularization.

Although the role of individual factors during tissue generation and repair is quite well researched, many details of this process are still unclear. In particular, the key features for the determination of tissue and organ morphology that result in the creation of the tissue-specific complex micro-architecture of vessels, stroma, and specialized cells, as well as a specific organ shape, are not completely understood. Additionally, the role of biological or mechanical interactions between the newly formed cells and their micro-environment has not yet been analyzed to an extent that would allow for the simulation of *in vivo* conditions of tissue regeneration in an *ex vivo* environment. As neither the technology for the cultivation

of vascularized cell-seeded scaffolds nor the possibility to provide *in vivo* conditions for *ex vivo* tissue formation are available yet, all tissue engineering endeavors have to rely on the contribution of the *in vivo* milieu of the local tissue at the site of implantation.

Most of the tissue engineering strategies have used only two of the four basic components yet. In general, the approaches can be grossly divided into cell-based approaches and those using growth factors. Cell-based devices employ specialized cells and scaffolds that are implanted into the recipient site. Ideally, these cells are autogenous cells that have been obtained from the tissue to be replaced by a minimally invasive surgical procedure with negligible morbidity. The initially small number of cells is expanded *ex vivo* and seeded into scaffolds to obtain a biohybrid construct that provides specialized cells in a three-dimensional arrangement. After implantation of these constructs into the *in vivo* environment, the seeded cells are supposed to continue with proliferation and differentiation, thus forming the desired tissue within the implanted scaffold. This approach relies on the host tissue to contribute the necessary cytokines that provide appropriate signals for proliferation, final differentiation, and tissue formation through the cells within the scaffolds as well as vascularization of the scaffolds to keep the implanted cells alive.

Approaches that are based on growth factors apply them in scaffold materials which serve two purposes. On the one hand, scaffolds act as carriers for the signaling molecules, providing a three-dimensional structure from which the growth factors are released to recruit specialized cells required for tissue repair. On the other hand, they function as space-making devices which prevent soft tissue collapse and provide room for the newly formed tissue to penetrate and unfold. These approaches rely on the host tissue to supply both vascularization and the necessary population of either specialized cells or undifferentiated cells that can be induced into the required type of tissue by the applied growth factor.

Both strategies are still struggling with a number of fundamental issues. Cell-based approaches deal with questions of *in vitro* conditions for tissue-specific expansion, seeding, and cultivation, as well as cell survival and function *in vivo*, while strategies for the application of growth factors focus on dosage and controlled release as well as possible interactions with the carrier material.

In order to overcome some of the problems of cell-based devices, combinations of scaffolds, cells, and growth factors have been explored. In the majority of cases recombinant cells have been employed which were designed to produce the desired growth factor after implantation *in vivo* to enhance vascularization and to avoid dedifferentiation of the implanted cells under adverse conditions of initial wound healing with decreased oxygen supply, decreased pH, and

impaired metabolic supply. Some approaches have used scaffolds directly loaded with growth factors in combination with cells. The results of all these experiments have shown that the combined use of cells, growth factors, and scaffolds may hold some promise for the improvement of the functionality of tissue-engineered biohybrid scaffolds.

Biomaterials

Biomaterial tissue interaction

Biomaterials are implanted to improve tissue function or to enhance tissue repair. In reconstructive surgery they are used to fill defects, to bridge gaps, or to cover wounds. They may function as scaffolds for tissue ingrowth and thereby enhance tissue regeneration or act as stabilizing devices to fix or join loose tissue parts. There is a large variety of different materials that are used and their characteristics differ according to the mechanical and biological requirements of their application. The same holds true for interactions between biomaterials and recipient tissues. They vary across the implantation period and their nature depends very much on whether the material is loaded or unloaded and whether it is intended to stay permanently in the body or is meant to degrade and disappear. Early events which occur immediately after implantation or during early stages of healing differ from those that can be seen after tissue integration. While the latter depend very much on the chemical nature of the material and its behavior in terms of degradation and material release, the former are considered to be affected mostly by physical surface characteristics.

When a biomaterial is inserted into living tissues, a cascade of events is initiated that starts with the adsorption of biomolecules to the material's surface. Important characteristics for the immediate interaction with the *in vivo* environment are surface charge and surface energy of the material. An increase in surface energy improves the wettability of the material surface facilitating the adsorption of serum proteins and other biomolecules such as fibronectin.^{2,3} This initial wetting, also referred to as flash spread, that is accompanied by the adsorption of biomolecules, is one of the most important preconditions for cells to become attached to the material surface and to establish tissue contact with the biomaterial. Surface energy and surface charge themselves depend on the material composition and on the surface structure. Metals and mineralized materials commonly have a negative surface charge under physiological conditions due to the presence of oxide molecules on their surface. This results in the adsorption of positively charged molecules to the biomaterial surface (Figs 52.1 and 51.2).

Surface energy varies considerably between different material classes. While polymers such as poly-

lactic acid (PLA) have a rather low surface energy and hence a rather hydrophobic surface characteristic, the higher surface energy of many metals and ceramics in particular renders these surfaces hydrophilic which can enhance tissue integration from the material side. Hydrophobic interactions between a material surface and adsorbed proteins can go along with stronger interacting forces between the surface and the protein, facilitating critical conformational changes of the adsorbed molecule, which may result in decreased functional activity of the respective protein. As hydrophilic surfaces are associated with less conformational changes of the adsorbed proteins, increased hydrophilicity can support the cascade of cellular events involved in the attachment, migration, and tissue-specific differentiation of cells.

Another feature that affects the interaction between biomaterials and living tissues is the surface texture. Microrough surfaces have been shown to affect cellular attachment and differentiation. Several factors are considered to contribute to the improved performance of these surface modifications when compared to non-structured surfaces. One positive effect of microstructured surfaces comes through the increase in surface energy that is associated with increased surface roughness and hence improved wettability.⁴ Another positive effect on cellular behavior is mediated through the microtexture itself. This has been

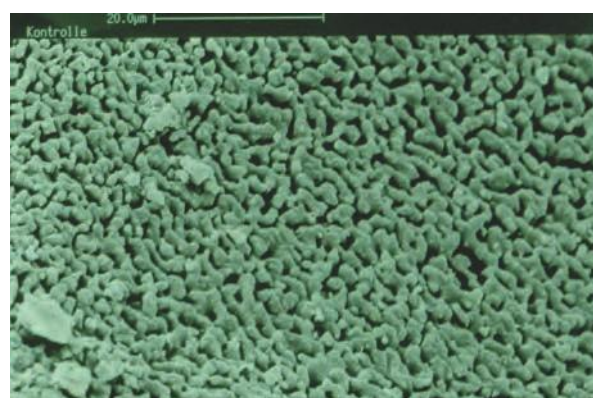


Fig. 52.1 Scanning electron microscope (SEM) image of the crystalline structure of the surface of a hydroxylapatite scaffold.

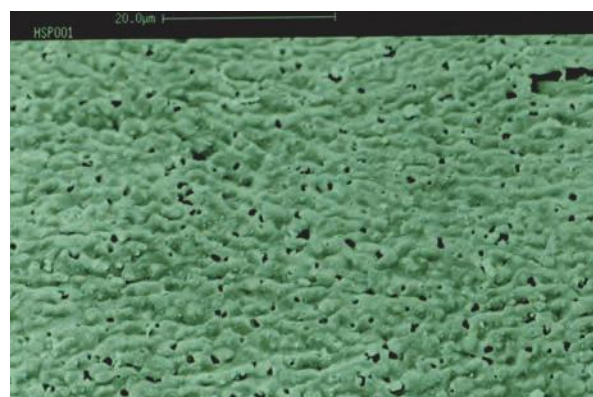


Fig. 52.2 Surface covered by proteins adsorbed from cell culture medium.

shown in surface modifications of titanium implants. Different degrees of surface roughness have been shown to modify cellular production of receptors that mediate adhesion to titanium surfaces.⁵ Moreover, increased secretion of molecules indicating osteogenic differentiation of cells in contact with the microtextured surface as well as enhanced production of cytokines involved in bone formation has been reported *in vitro*.^{6,7}

Surface characteristics play an important role, not only *in vivo* for the biomaterial tissue interaction, but also in the *in vitro* environment for tissue engineering purposes, as cellular attachment, migration, and differentiation are key issues in the construction of biohybrid scaffolds. In this way material science in biotechnology can contribute substantially to the success of tissue engineering by optimizing immediate and late interactions both between seeded cells *in vitro* and living tissues after implantation *in vivo*.

Scaffolds

Scaffolds are used as carriers for cells and/or growth factors. Ideally, they provide a three-dimensional structure of interconnecting porosity that supports the ingrowth of vessels to supply the implanted cells and/or allows for the release and diffusion of biological signals which then enhance ingrowth of undifferentiated cells through a chemotactic gradient and accomplish final tissue-specific differentiation.

In general, the plethora of materials that are used as scaffolds can be divided into organic and inorganic materials. Organic materials may originate from natural precursors such as collagen, chitosan, or silk (Figs 52.3 and 52.4), or can be produced from synthetic

polymers such as PLA (Fig. 52.5) and polyglycolic acid (PGA). Inorganic materials commonly consist of metals, alloys, or mineral compositions. Mineral compositions may vary considerably containing mixtures of calcium, sodium, potassium, silicates, magnesium, aluminum, zirconia, carbonates, or phosphates. Mineralized scaffolds may be purely synthetic or derived from natural precursors such as corals, algae, or bovine bone (Figs 52.6 and 52.7).

A critical feature for the performance of scaffolds *in vivo* is the degree of porosity. Only scaffolds with interconnecting pores will become vascularized and completely penetrated by ingrowing tissue. Pore sizes are divided into micropores (<10 μ m) and macropores (>50–60 μ m). The former can enhance the retention of growth factors soaked into the material or have an effect on cell attachment, migration, and differentiation, whereas the latter play an important role for the nature of the ingrowing tissue. While pores of 50 μ m allow for the ingrowth of individual

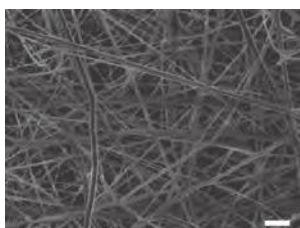


Fig. 52.3 SEM image of electro spun chitosan. (Reproduced with permission from Schiffman JD, Schauer CL. One-step electrospinning of cross-linked chitosan fibers. *Biomacromolecules* 2007; 8: 2665–7. Copyright © 2007 American Chemical Society.)

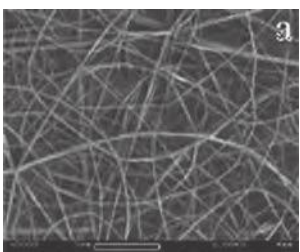


Fig. 52.4 SEM image of a fibrous silk scaffold. (Reproduced with permission from Bini E, Foo CW, Huang J, Karageorgiou V, Kitchel B, Kaplan DL. RGD-functionalized bioengineered spider dragline silk biomaterial. *Biomacromolecules* 2006; 7: 3139–45. Copyright © 2006 American Chemical Society.)



Fig. 52.5 SEM image of a non-woven fibrous poly(lactic acid) scaffold.

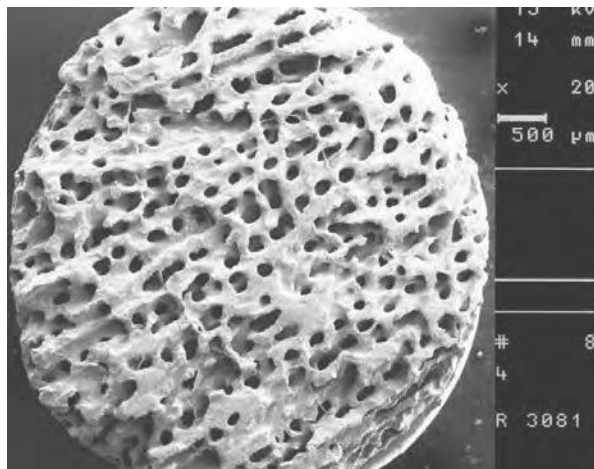


Fig. 52.6 SEM image of a coralline CaCO₃ scaffold.

cells, vascular ingrowth can be seen at pore sizes $>150\mu\text{m}$ and ingrowth of mineralized bone requires a pore size of $250\mu\text{m}$.

There is always a link between the scaffold material and the scaffold structure in that the strength and the elasticity of the various materials allow only for limited variations in scaffold geometry. Organic scaffolds of natural origin, such as chitosan and silk that are produced by electrospinning, commonly form a porous textile structure.^{8–10} The same holds true for polymer fibers that may be also produced as non-woven fleeces. They all have in common a thin and elastic fibrous scaffold structure that allows for cell attachment but may impair spreading of cells that require a flat surface structure (Fig. 52.8). The advantage of this type of scaffold, however, is the wide open pore space which enhances metabolic exchange and tissue penetration. Conversely, organic scaffolds become very brittle if their structural elements fall short of a minimum thickness. Metallic scaffolds can be produced as open porous three-dimensional structures by selective laser melting. However, the pore size of these types of scaffolds is far above the micrometer range. Mineralized scaffolds from synthetic materials are commonly produced from slurry or powders that are sintered in the presence of a foaming agent such as H_2O_2 or with the addition of NaCl or sugar as porogens that are subsequently removed during sintering or leaching. An alternative

route is the imbibition of organic scaffolds of defined geometry with calcium phosphate slurry and subsequent removal of the organic components during sintering.¹¹ The disadvantage of this technique is the fact that the resulting bubbles and void spaces that make up the pore spaces are not necessarily interconnected, which impairs tissue penetration and complete integration (Fig. 52.9). Mineralized scaffolds from natural precursors so far are the only ones that can provide a truly interconnecting open porous structure at the micrometer range.

For *in vitro* fabrication of biohybrid constructs, the structure and the nature of the scaffold material also have an impact on the morphology and the behavior of the seeded cells, in that the shape of the pore walls and the geometry of the scaffold components affect the attachment, proliferation, and migration of the cells. However, as the scaffold structure is closely linked to the scaffold material, the effect on cellular behavior, such as proliferation and differentiation, is always a combined effect of both surface morphology and material composition.

Biofunctionalization

The fact that the initial interactions of biomaterials with their host tissues are based on the adsorption of biomolecules from the *in vivo* milieu on the material surface has promoted the idea of enhancing the integration of these materials by modifying the surface conditions either by enhancing adsorption of these molecules *in vivo* or by activating biomaterial surfaces with biologically active molecules (BAMs). The strategies vary from altering the physical characteristics

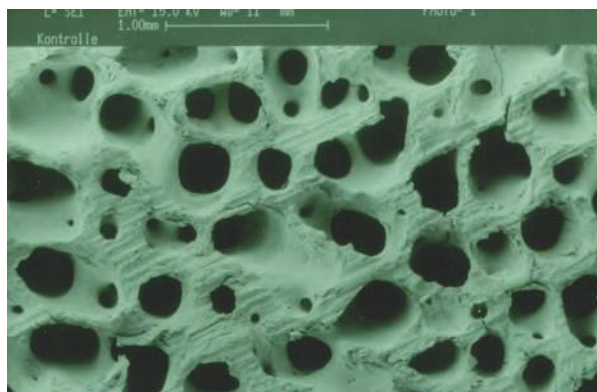


Fig. 52.7 SEM image of an anorganic bovine bone scaffold.

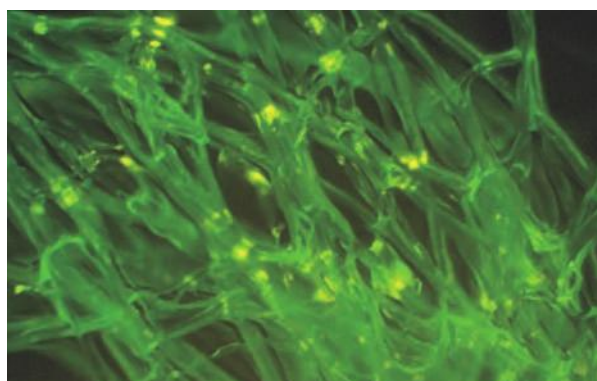


Fig. 52.8 Light microscopic picture of a textile polylactic acid scaffold with cells located at cross points of the fibers. NCell nuclei are colored green (Pico Green).

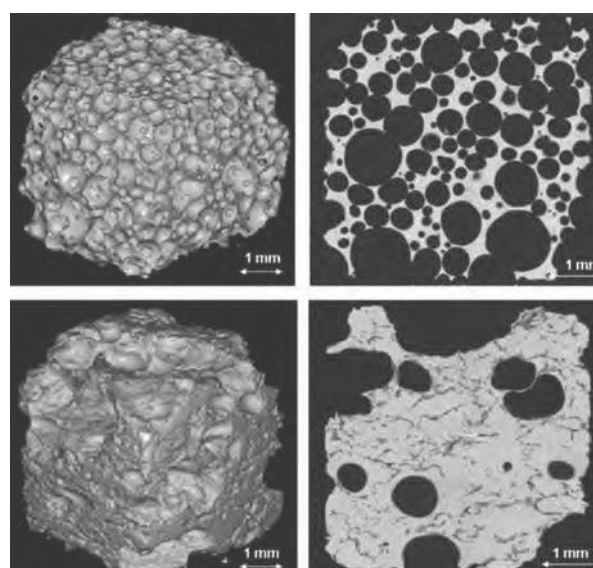


Fig. 52.9 Three-dimensional reconstructions from microtomographic images of porous hydroxylapatite scaffolds produced from calcium phosphate slurries. (Reproduced with permission from Peyrin F, Mastrogiacomo M, Cancedda R, Martinetti R. SEM and 3D synchrotron radiation microtomography in the study of bioceramic scaffolds for tissue engineering applications. *Biotechnol Bioeng* 2007; 97: 638–48. Copyright © 2007 John Wiley & Sons, Inc.)

through increased surface roughness to physico-chemical and/or chemical modification.¹² Many of these approaches have been used with metal surfaces, namely endosseous implants made of titanium or titanium alloys.^{13–16} Although metal implants do not yet play a substantial role in tissue engineering, measures to enhance peri-implant bone healing next to these implants fall into the realm of tissue engineering and the technology developed is also applicable to many other biomaterials used in this field.

Chemical modifications are probably the most promising approaches to enhance tissue integration and to modify peri-implant tissue reactions. Among these, strategies using organic chemistry to anchor biologically active molecules on the scaffold surface are particularly attractive as the critical process of cellular attachment could be short cut or even tailored to specific tissue requirements. In this way, the process of tissue integration could be considerably accelerated by placing a selected pattern of signals on the biomaterial surface. For these applications proteins or peptides involved in cell attachment and cell communication are of particular interest.

Key molecules for cell attachment and migration of cells are RGD peptides (a sequence of three amino acids Arg-Lys-Asp). Their biological effect is mediated through binding to integrin receptors on the cell surface.¹⁷ This activates a cytoplasmic signaling pathway that can alter cellular architecture or initiate cell migration and proliferation. A number of molecules from the ECM present different kinds of RGD motifs and bind to different integrin subunits. Collagen, for example, binds to $\alpha_2\beta_1$ units, while RGD motifs of bone-specific proteins such as vitronectin, osteopontin, and osteoprotegerin bind to $\alpha_v\beta_3$ and $\alpha_v\beta_5$ receptors.^{18,19} The specificity required to address different integrin receptor subunits is based on more complex peptide sequences (RGDXYZ motifs) than the short core sequence of three amino acids.^{20–24} Specifically designed RGDXYZ motifs, thus, could mimic certain components of the native

ECM and could selectively direct a specific population of cells to the material surface. In this way, bio-functionalization of material surfaces using RGD sequences has been successfully accomplished with various scaffold designs of a large number of materials such as chitosan, silk, polylactic and polyglycolic acid, polyethylene glycol (PEG), and titanium.^{25–30} All reports had proven increased cell attachment, cell proliferation or enhanced *in vivo* tissue integration (Figs 52.10 and 52.11).

Besides providing a matrix for cell attachment and migration, many components of the ECM have a reservoir function for growth factors that are released during tissue repair. In bone tissue, for example, glycosaminoglycans play an important role in the storage and release of growth factors from the bone matrix during remodeling and regeneration of bone tissue. In order to mimic natural events such as ECM degradation and release of BAMs, the use of specific organic coatings that mimic the function of native ECM could elicit a targeted cellular reaction and enhance the integration of the biomaterial into a specific tissue. Strategies using this approach have been developed by employing biomimetic ECM-like synthetic polymers that contain biologically active molecules that are released through the activity of matrix metalloproteinases (MMPs)³¹ and by using components of the native ECM that can accumulate BAMs (Figs 52.12 and 52.13).^{32–34} The latter approach followed a self-organizing principle, according to which ECM components such as chondroitin sulfate assemble spontaneously with collagen in a near natural fashion and could accumulate BAMs. This would allow for a more specific or enhanced biomaterial tissue interaction.

Methods to immobilize BAMs on to biomaterial surfaces depend on the stability of the BAMs themselves, as well as their molecular weight and structure. In general, BAMs can be attached to the surface of biomaterials through adsorption, covalent binding, nanomechanical incorporation, and self-organizing

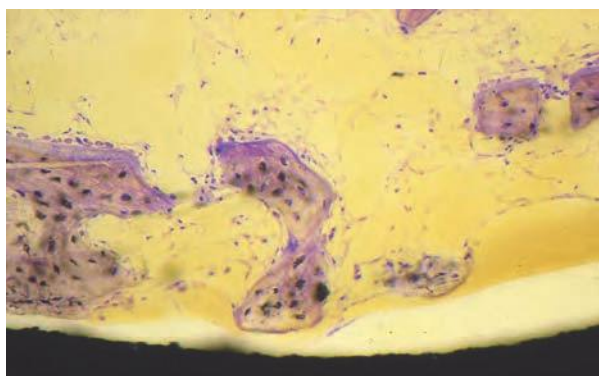


Fig. 52.10 Reduced bone implant contact on a machined titanium surface. (Reproduced with permission from Schliephake H, Scharnweber D, Dard M, Sewing A, Aref A, Roessler S. Functionalization of dental implant surfaces using adhesion molecules. *J Biomed Mater Res B; Appl Biomater* 2005; 73: 88–96. Copyright © 2005 John Wiley & Sons, Inc.)

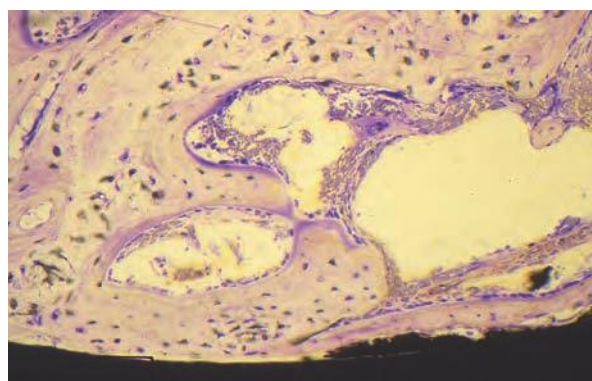


Fig. 52.11 Significantly enhanced bone implant contact on a collagen/RGD peptide coated titanium surface. (Reproduced with permission from Schliephake H, Scharnweber D, Dard M, Sewing A, Aref A, Roessler S. Functionalization of dental implant surfaces using adhesion molecules. *J Biomed Mater Res B; Appl Biomater* 2005; 73: 88–96. Copyright © 2005 John Wiley & Sons, Inc.)

organic layers.¹² As many biomaterial surfaces carry a negative charge under *in vivo* conditions, macromolecules positively charged under these conditions could adsorb with great stability due to electrostatic interactions. This has been used by Ruiz-Taylor *et al.* for immobilization of poly(L-lysine) on titanium surfaces.³⁵ In this approach, poly(L-lysine) was not the BAM itself but was used as an anchor molecule for covalently coupled PEG chains and RGD groups. This coating has been reported to be highly promotive for cell adhesion due to the incorporated RGD motifs.³⁶ The self-organization principle of organic layers has been also applied for the immobilization of collagen together with proteoglycans and glycosaminoglycans, which are the most interesting candidates due to their interactive potential with respect to bone matrix assembly and binding of growth factors. Designed components derived thereof through methods of functional glycomics have been used in more advanced approaches of matrix engineering.^{37–39} In this way, biofunctionalization of material surfaces

can significantly enhance their function *in vitro* and *in vivo*, particularly when growth factors are anchored to the surface.

Application of growth factors

Growth factors

Growth factors are polypeptides of approximately 6–45 kD that act as signaling molecules for cellular communication. They play an important role in morphogenesis and repair of tissues and organs. Their range of activity varies considerably during embryogenesis, postnatal growth, and adulthood. During embryonic development, growth factors are involved in limb patterning and morphogenesis of many organs, while in postnatal life their role is restricted mainly to tissue regeneration and repair. The biological effect of an individual growth factor *in vivo* is modulated by a complex system of feedback loops, which involve other growth factors, enzymes, and binding proteins.

Growth factors have a complex three-dimensional structure that is made up of either a single polypeptide chain or two chains that are identical in homodimeric molecules and different in heterodimeric molecules. This complex structure allows for selective binding to specific receptors on the cell surface. Receptors are transmembranous proteins which undergo conformational changes upon binding of the growth factor. This activates intracellular phosphorylating enzymes, which induce an intracellular signaling pathway by aggregation of cofactors and other proteins that migrate to the nucleus. Together with other transcription factors they activate a set of genes, which then bring about the specific changes in cellular activity or phenotype.

The effect of growth factors on their target cells can be different. They can act as mitogens in that they enhance proliferation of certain cell types. Some growth factors are also morphogenic in that they change the phenotype of their target cells. Growth factors acting in an autocrine manner affect the secreting cell itself, whereas paracrine growth factors affect their neighboring cells. For some growth factors, an endocrine effect is assumed or established from elevated serum levels. Many growth factors are deposited in the ECM where they are released during matrix degradation and act as part of a complex network of signals with mutual effects during tissue remodeling and regeneration.

The therapeutic application of growth factors for the repair of damaged tissue is used to enhance the level of regeneration beyond the limit to which spontaneous tissue repair has been downregulated in postnatal life. Thus, defects that would otherwise not heal in life time (“critical size defects”) are intended to be repaired without the need for additional tissue transfer.

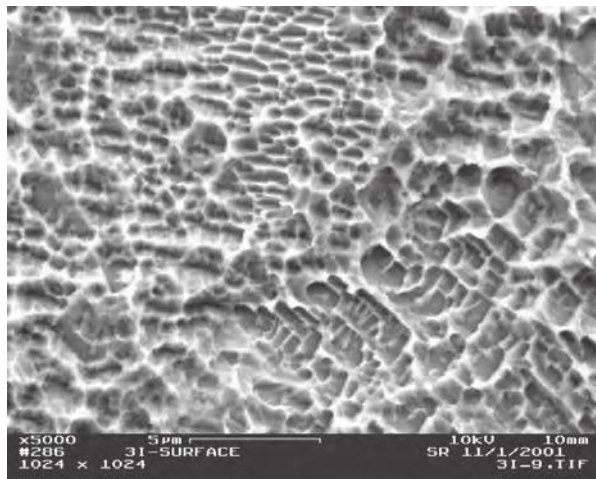


Fig. 52.12 SEM image of a titanium surface microstructured by sandblasting and etching.



Fig. 52.13 SEM image of a sandblasted/acid etched titanium surface coated with collagen and chondroitin sulfate (CS). The typical structure is visible in the collagen fibers.

Many growth and differentiation factors of different origins have been employed both in preclinical and clinical tissue repair. When single growth factors are intended for therapeutic application, recombinant growth factors are commonly used. Recombinant growth factors are identical to human growth factors with respect to amino acid sequences and three-dimensional structure. They are produced by either eukaryotic cells or bacteria (*Escherichia coli*) that have been transfected/transduced with the respective gene.

The majority of growth factors used in maxillofacial reconstruction are directed towards the repair of mesenchymal tissues. Some of them are also involved in the interaction between mesenchymal tissue and epithelium during mucosa repair. In particular, bone regeneration is a clinical focus of tissue engineering using growth factors. There are at least six growth factors involved in bone regeneration that have been used in maxillofacial reconstruction in a large number of animal models:

- platelet-derived growth factor (PDGF);^{40–42}
- basic fibroblast growth factor (bFGF);^{43–46}
- insulin-like growth factor (IGF);^{47–50}
- transforming growth factor beta (TGFβ);^{51–53}
- vascular endothelial growth factor (VEGF);^{54,55}
- bone morphogenetic proteins (BMPs).^{56–59}

PDGF is a dimeric protein of 30 kD with two different peptide chains (A and B) which combine to three isoforms of PDGF: two homodimeric forms (PDGF-AA and PDGF-BB) and one heterodimeric molecule (PDGF-AB). PDGF-AB and PDGF-BB are available systemically in the α-granules of platelets, whereas PDGF-AA is secreted locally by osteoblasts. *In vivo*, PDGF plays an important role in the recruitment of cells for the repair of mesenchymal tissues such as bone, cartilage, and connective tissue. The effect is predominantly mitogenic and chemotactic for precursor cells in that proliferation is stimulated, whereas differentiation into the specialized cells or tissue maturation is suppressed. Thus, PDGF acts as a key factor during early stages of regeneration and remodeling of these tissues by activating undifferentiated precursor cells, thereby increasing the number of inducible cells. However, it does not exert morphogenic effects by inducing differentiation into bone or cartilage tissue.

Basic fibroblast growth factor (bFGF, FGF2) is one of the 23 members of the fibroblast growth factor family. Native bFGF is bound to heparin and stored in the ECM. In bone tissue, biologically active bFGF is produced by osteoblasts and deposited in the bone matrix from where it is released during repair and remodeling. bFGF increases proliferation of mesenchymal cells and supports the differentiation of cartilage and bone cells. Moreover, it enhances the proliferation of endothelial cells and accelerates endochondral ossification presumably through

increased vascularization of the cartilage. Thus, bFGF acts as a mediator of bone formation at a rather early stage by increasing cell numbers and vascularization. It has no direct morphogenic effect on bone or cartilage tissue but has to be considered as one of the important adjuvant factors in this complex process.

IGFs are single-chain molecules that exist in two isoforms (IGF I and IGF II). In contrast to many other factors that act merely at a local level, IGFs also play a systemic role in postnatal growth. At a local level, IGFs act as survival factors that suppress cellular apoptosis and increase proliferation in a number of mesenchymal cell types such as osteoblasts, chondroblasts, and fibroblasts. Thereby, they support tissue regeneration and repair, albeit on a rather generic level.

TGFβ belongs to the TGF superfamily, a large family of more than 30 proteins that also includes the BMPs. TGFβ molecules are secreted as inactive molecules and activated by cleaving off N-terminal portions of the molecule. TGFβ molecules are multifunctional in that they affect adhesion, proliferation, and apoptosis of numerous cell types. They are involved in remodeling and repair of cartilage tissue and there is a dose-dependent effect on proliferation and differentiation of bone cells, in that high concentrations of TGFβ have a negative effect, whereas low concentrations increase cell numbers and maturation. TGFβ has a prominent role during soft tissue regeneration. It enhances matrix production and supports contraction of fibroblasts. In this way it can contribute to excessive scarring and the occurrence of radiogenic fibrosis.

VEGF is a homodimeric protein of which a number of modifications are known (VEGF A through F). The modifications differ in the length of the molecule. VEGFs are ubiquitous and play a central role in angiogenesis during tissue repair and inflammation. They are also important contributors to the occurrence of malignancies through the induction of neo-vascularization. Tissue hypoxia induces the production and secretion of VEGFs that interact with other growth factors to induce the development of a stable and defined system of mature vessels. VEGFs are involved in tissue regeneration and repair in more than one respect, as the newly formed vessels not only provide oxygen supply but also bring a perivascular population of undifferentiated precursor cells into the area of regeneration.

BMPs are homodimeric proteins of approximately 30 kD. Today, almost 30 proteins have been identified that belong to this family. During embryogenesis, BMPs play an almost universal role in the development of the body as they are involved in the organogenesis of kidney, lung, heart, central nervous system, eye, skin, and teeth. The most prominent effect of BMPs in postnatal life is the ability to induce the formation of bone tissue in ectopic sites, where otherwise no bone formation would occur. This ability is unique as it is able to recruit undifferentiated mesenchymal

cells through chemotaxis and induce the complete cascade of molecular events by the expression of bone-specific enzymes, proteins, and transcription factors such as alkaline phosphatase, osteocalcin, and Runx2 that are necessary to transform precursor cells into active osteoblasts that start to form bone.

The limited availability and the high costs of recombinant growth factors have prompted the search for alternatives, such as autogenous growth factors. One source of autogenous growth factors has been identified in concentrated platelet preparations (platelet-rich plasma, PRP). Platelets are among the first elements of the wound healing cascade to appear at a site of injury. They carry a variety of growth factors that are supposed to enhance wound closure and tissue repair. PRP, therefore, represents a mixture of autogenous growth factors with predominantly mitogenic activity suitable to initiate and accelerate tissue repair through an increase in cell proliferation. PRP is produced through a two-step centrifugation procedure that is subject to some variation with respect to the effect of platelet concentration and growth factor content. There are different centrifugation systems on the market and their ability to concentrate platelets varies between the factor 3 and 10. Additionally, there is considerable variation within the individual systems so that the reproducibility of the procedure has to be considered as low and requires individual quality control by platelet counts after centrifugation. Unfortunately, the amount of growth factors contained within each individual preparation of PRP is also not correlated with the number of platelets,⁶⁰ probably because the activation of platelets *ex vivo* and the release of growth factors from the alpha granules are not always reliable.

PRP contains all four growth factors: PDGF, TGF β , VEGF, and epithelial growth factor (EGF). The growth factors are deposited in the alpha granules from where they are released after the platelets have become attached to collagen fibers of the vessel wall or after exposure to thrombin. During therapeutic application of PRP, thrombin and calcium are added to release the growth factors from the platelets. *In vitro*, PRP enhances the proliferation of fibroblasts from the gingiva and the periodontal ligament. Proliferation of osteoprogenitor cells and osteoblasts is enhanced in a concentration-dependent manner; however, terminal osteogenic differentiation of progenitor cells is suppressed.⁶¹ Positive effects on the proliferation of epithelial cells have not been clearly shown. *In vivo*, PRP has been extensively tested in preclinical studies, mainly to explore the ability to enhance bone regeneration. PRP has been used in conjunction with both autogenous bone grafts and synthetic or natural carriers such as tricalcium phosphate (TCP), bovine bone mineral, collagen, and demineralized freeze-dried bone^{62–66} in peri-implant bone defects, sinus floor augmentations, onlay grafts, and segmental mandibular defects.^{67–71} The picture that arises from these studies is unclear because the number of studies

which failed to prove significant enhancement of bone regeneration is only slightly larger than the number of reports that could show a positive effect. The failure of PRP to prove a positive effect in preclinical studies has been explained by the inability to prepare appropriate amounts of PRP from animal blood with adequate quality.⁷² Unfortunately, the results derived from clinical studies are even less encouraging. A large number of clinical case reports or case series describe the use of PRP; however, there are only a few controlled trials.^{73–76} Only one of these studies has proven a significantly positive effect on bone regeneration in sinus lifts⁷⁶ while the remaining reports could not show a significant enhancement of bone formation in the augmented areas. One of the reasons for the failure of autogenous growth factors in PRP to show consistently positive clinical results may be the poor reproducibility with respect to platelet count and growth factor content of the individual preparations.

Recombinant growth factors have been tested as single growth factors and in combination with others merely with respect to their ability to enhance bone regeneration. In preclinical settings most of the human recombinant bone growth factors have not shown truly osteoinductive properties when they were tested as single factors. Only when used in conjunction with other growth factors did PDGF, bFGF, and IGF show enhancement of bone regeneration in peri-implant or periodontal regeneration models,^{41,43,48–50,77,78} whereas isolated administration failed to significantly increase bone formation (Fig. 52.14).⁴⁴ In this way, it is comparable to TGF β that has shown enhancement of bone formation of varying degrees depending on the carrier used,^{80,81} but when combined with PDGF or BMP has resulted in significantly increased bone formation.⁵⁹ VEGF has come into focus as an important cofactor for bone formation for induction of bone tissue in conjunction with BMPs,⁵⁴ due to the abilities

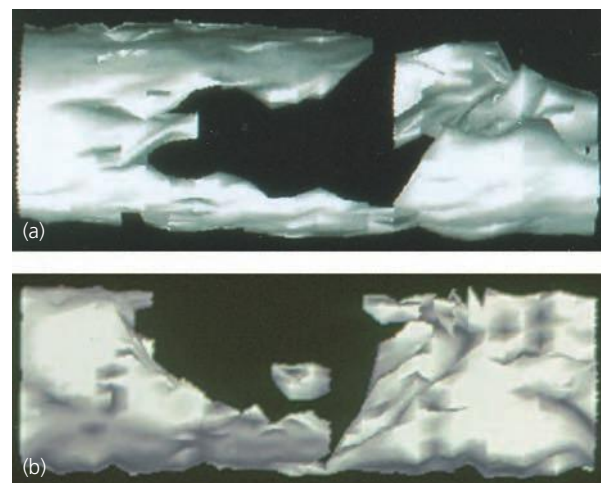


Fig. 52.14 Reconstruction of bone regeneration inside anorganic bovine bone cylinders forming bridging of mandibular defects in minipigs. Incomplete bone fill is visible in scaffolds both with 230 μ g bFGF (a) or without bFGF (b).

both to enhance bone regeneration and increase vascularization.^{55,82}

The only group of growth factors that has consistently promoted bone formation in almost all preclinical settings is the family of BMPs. There are three isoforms of BMPs, BMP-2, BMP-4, and BMP-7 (also called osteogenic protein 1 (OP-1)), that have been explored in the vast majority of studies. Experimental repair of alveolar bone defects, sinus floor augmentations, and mandibular reconstructions has been successfully performed using these molecules.^{83–86} In particular, critical size defect models of the mandible and segmental defects have been evaluated in a number of experimental models with a large variety of carriers. Collagen, collagen/HA/TCP, anorganic bovine bone, hyaluronic acid, PLA/PGA coated gelatin sponges, and PGLA beads have been employed with various doses of either rhBMP-2, rhBMP4, or rhBMP7 in rodents, minipigs, and non-human primates.^{87–93} Another growth factor from the BMP fam-

ily that has been recently introduced into preclinical and clinical testing is growth and differentiation factor 5 (GDF-5). GDF-5, in conjunction with particulate TCP, has been shown to increase bone formation in sinus floor augmentations in minipigs (Fig. 52.15) where it has also proven to be superior to a mixture of the carrier material and autogenous minced bone.^{94,95}

Considering the entire data derived from preclinical testing of growth factors in oral maxillofacial surgery it is very appreciable that the most promising field for the use of growth factors appears to be bone regeneration, in particular the use of BMPs. Nevertheless, data about clinical application of BMPs is still sparse. Recently, BMP2 has received FDA approval for application in maxillofacial reconstruction and a small number of studies in recent years has shown that BMP can be also effective in generating bone in humans.^{96,97} However, not all reports have been clearly convincing for various reasons.^{98,99} One of these reasons is the lack of appropriate delivery

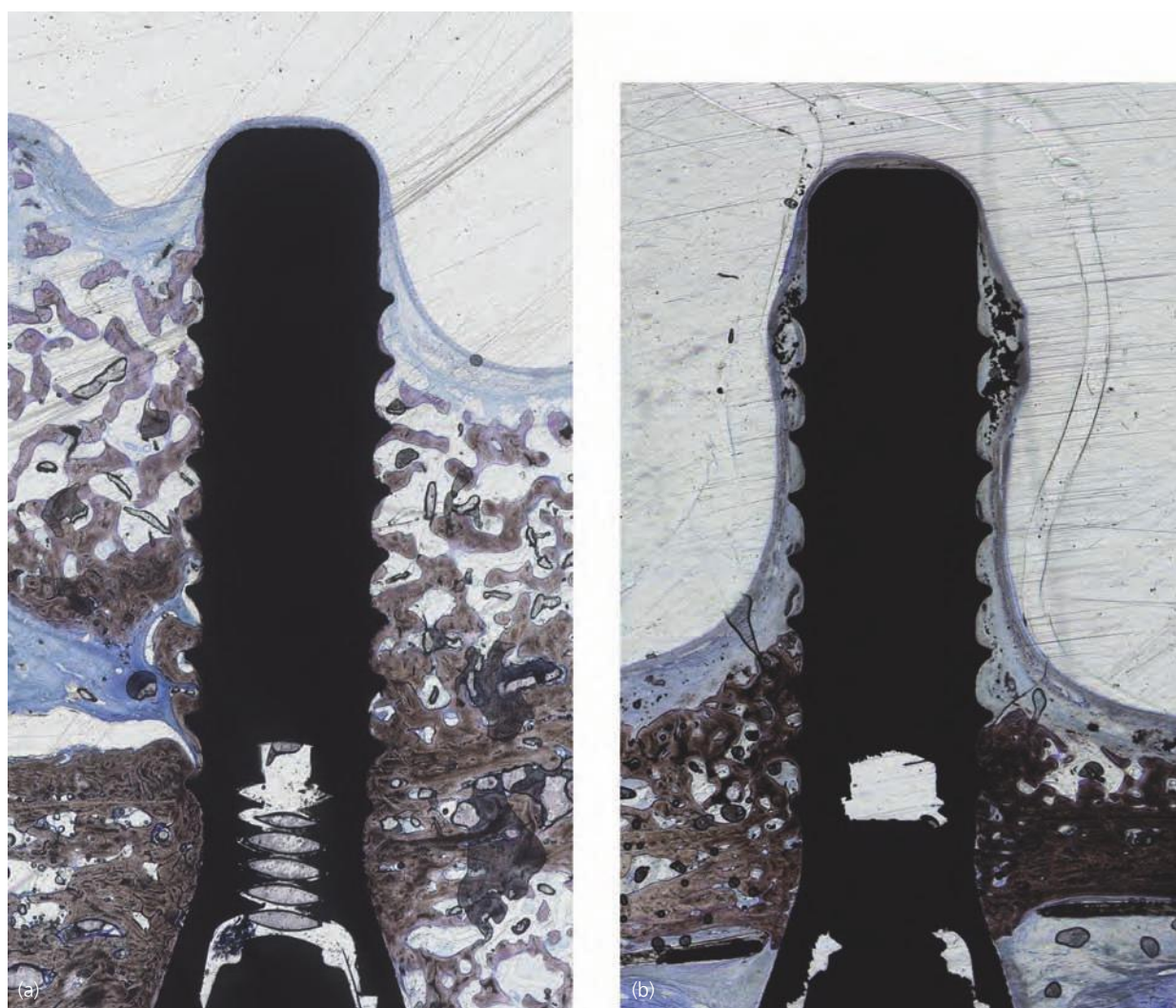


Fig. 52.15 Bone regeneration in sinus floor augmentations with TCP and GDF-5. Significantly enhanced bone volume in sides with GDF-5 (a) compared to sides with the carrier only (b). (Reproduced with permission from Gruber R, Ludwig A, Achilles M, Poehling S, Merten HA, Schliephake H. Sinus floor augmentation with recombinant human growth and differentiation factor-5 (rhGDF-5): a histological and histomorphometric study in the Goettingen miniature pig. *Clin Oral Implants Res* 2008; 19: 522–9. Copyright © 2008 John Wiley & Sons, Inc.)

vehicles for BMP. The carrier that has been approved by the FDA is collagen which has some shortcomings with respect to mechanical strength and release characteristics. Now that the proof of principle has been shown in clinical trials, current research is focused on the mode of delivery in order to make the application of BMPs more effective and reliable.

Delivery vehicles and controlled release

As growth factors are applied to enhance the biological process of tissue regeneration, the tissue-specific levels of cellular communication need to be addressed in an appropriate way. The intensity of the biological signal, i.e. the concentration of the respective growth factor, is supposed to be available at the temporal sequence and the spatial pattern required for the target cells to respond effectively. Unfortunately, neither the temporal sequence of individual growth factor concentrations during tissue repair nor their interaction is close to being clarified. So far, the majority of applications therefore have implanted growth factors that were soaked into various carrier materials with unknown release characteristics with rather arbitrary dosages. Compared to the natural amount of growth factors present during tissue repair, the doses of growth factors applied has always been several orders of magnitude higher. Dosages of growth factors vary also between different animal species. Particularly with respect to BMPs, dosage studies have shown

that moving up the phylogenetic ladder was associated with an increase in dosage required to elicit a biological response. Finally, moving from the preclinical environment into the clinical arena was associated with another huge increase in the amount of BMPs necessary to induce a reliable tissue response.⁵⁷ A recent study about sinus floor augmentations in patients has shown that up to 24 mg/ml of BMP2 per sinus had to be used to produce bone of sufficient quality and quantity to accommodate implants in a second stage operation.⁹⁷ In contrast to this, the native content of BMP in fresh bone tissue is approximately 7 ng/g demineralized bone matrix.¹⁰⁰ The difference between the natural amount of BMPs present at the site of repair and the dosage of BMPs required for clinical use thus is represented by a factor of a magnitude of 10^7 – 10^8 . The need for such excessive doses in clinical therapy has implications with respect to safety and costs on the one hand and has prompted the search for slow-release carriers that could provide a more physiological release profile of the growth factor on the other. As carriers for growth factors also have a mechanical function, in that they should have adequate strength to withstand pressure from overlying or surrounding soft tissue and provide volume for the regenerating tissue, the key question for the clinical application of growth factors is the selection of a carrier that allows for a controlled and retarded release of growth factors and at the same time has the mechanical characteristic required to provide space for tissue regeneration.

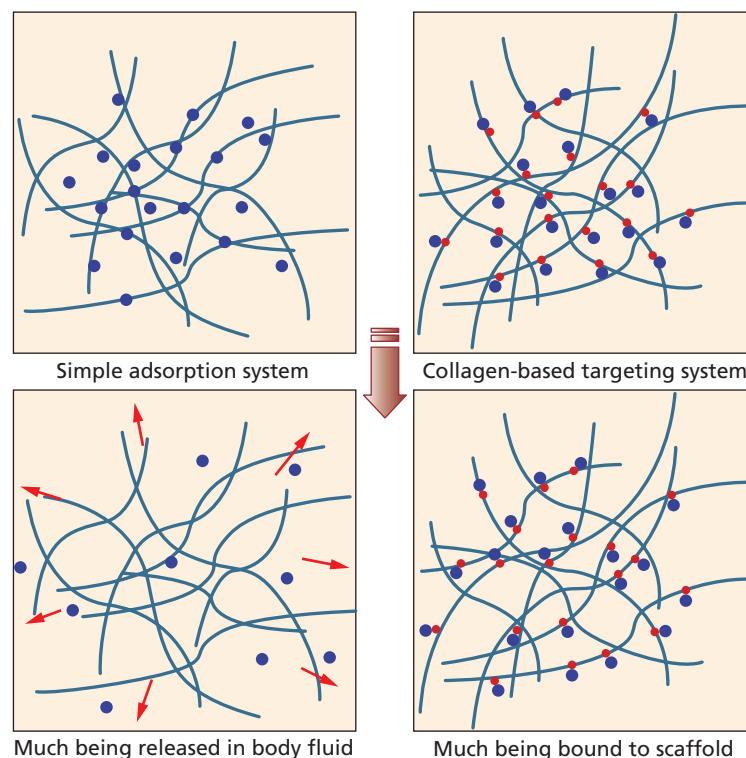


Fig. 52.16 Principle of an engineered growth factor (BMP2) that has been modified by a collagen binding site at the N-terminal end of the molecule. (Reproduced with permission from Chen B, Lin H, Wang J, *et al.* Homogenous osteogenesis and bone regeneration by demineralised bone matrix loading with collagen-targeting bone morphogenetic protein-2. *Biomaterials* 2007; 28: 1027–35. Copyright © 2007 Elsevier.)

The majority of delivery vehicles that have been developed and tested in the oral maxillofacial area were used for the application of BMPs. Most of these studies have employed collagen sponges as carriers, on to which BMPs were loaded by soaking the sponge into the growth factor solution. Unfortunately, collagen neither allows for retarded or controlled growth factor delivery nor fulfills the mechanical requirements of a spacemaking device to provide volume for bone regeneration. Delivery studies in collagen carriers have shown that soak loading with BMP is associated with rapid delivery of up to 80% of the growth factor content after implantation within the first 48 hours.^{101,102} As most growth factors carry a heparin-binding domain, binding of heparin to collagen has improved the release characteristic of the collagen carriers towards a more retarded delivery.¹⁰³ Also, addition of a collagen-binding domain to BMP by molecular engineering has significantly improved the efficacy of collagen carriers with respect to bone formation (Fig. 52.16).¹⁰⁴ However, as the mechanical properties still remain insufficient, other carriers have to be taken into consideration.

When delivery vehicles for growth factors are considered, the material characteristics are important for the strategy of growth factor loading and binding to the carrier. Non-resorbable carriers will have to have the growth factor anchored to the material surface, where it can be addressed by approaching cells and/or from where it is released from a chemotactic gradient that attracts target cells and tissue. In contrast, binding of growth factors only to the carrier surface is problematic in resorbable carriers because commencing degradation on the material surface will rapidly release the growth factor together with the superficial layers of the carrier material, leaving behind an entirely passive scaffold that is unable to enhance tissue regeneration during the subsequent period of continued carrier degradation and tissue ingrowth.

Binding of growth factors to the surface of non-resorbable carriers may be considered as biofunctionalization (see earlier in this chapter). Adsorptive loading of biomaterials with growth factors is very dependent on the surface characteristic of the carrier. Physisorption of BMP2 to titanium surfaces has resulted in release of 96–99% of activity within the first hour depending on the surface microstructure.¹⁰⁵ In contrast, adsorption of BMP2 to nanocrystalline diamond-coated titanium surfaces was associated with firm binding of the BMP molecule to the surface with almost no subsequent release.¹⁰⁶ An alternative way of binding growth factors to carriers is the application of linker molecules (chemisorption). Phosphonate anchors that attach to the oxygen of oxide groups on the material surface can be used to bind to the amino groups of growth factor molecules using succinimide chemistry, whereas thiol anchors bind to cysteine residues of organic molecules using maleimide chemistry. The nature of the carrier material and the molecular structure of the growth factor

to be attached frequently determine the choice of linker molecules. Phosphonate anchors are commonly used to couple BMP2 to a carrier surface because the seven cysteine residues are engaged in disulfide bonds within the molecule and in the intermolecular disulfide bond of the dimeric protein.¹⁰⁷ However, both phosphonate and thiol anchors have been shown to significantly enhance the retention of BMPs on the surface of metal, silk, and chitosan compared to untreated controls.^{107–109}

Incorporation of growth factors into resorbable carriers has been explored on several avenues. Soft and flexible delivery vehicles derived from native precursors, such as collagen, silk, or chitosan, have been soak loaded, and coupled to growth factors by covalent binding of heparin groups to collagen,¹⁰³ and by chemisorption using succinimide and maleimide chemistry in silk and chitosan scaffolds.^{106,107} Purely synthetic resorbable carrier materials that have been used as delivery vehicles are PEG and poly- α -hydroxy acids, such as PLA and PGA. PEG is a rather soft and gel-like material that has been successfully tested in conjunction with parathormone and BMP2 but would not fulfill the mechanical requirements to maintain tissue volume and shape in an unprotected defect. PLA and PGA polymers have a much higher stability but are difficult to load with growth factors as they are solid at room and body temperature and would only be amenable to surface coupling of growth factors. If the polymers could be liquefied, growth factors could be mixed with the liquid polymer to provide efficient incorporation. However, the glass transition temperature of these polymers (i.e. the temperature at which the polymers become liquid) is higher than the temperature at which the proteins denature and lose their biological activity. This problem has been addressed by applying organic solvents that are mixed with the polymer granules and the growth factor. After evaporation, a solid implant occurs with the growth factor incorporated into the polymer matrix. This solvent technology has been applied with BMPs¹¹⁰ and has been improved by adding growth factor binding molecules such as heparin¹¹¹ at the preclinical level. However, concerns have been raised in conjunction with this technique about residues of organic solvent that could cause toxic reactions and lead to tissue irritation *in vivo*. An alternative way of incorporating growth factors into the polymer matrix is gas foaming. During this process a lyophilized mixture of polymer granules and growth factor is submitted to high carbon dioxide pressure in a mold. This leads to liquefaction of the polymer at body temperature with homogeneous distribution of the growth factor.^{112,113} During decompression, the liquid polymer transforms into a solid foam that fills the mold. Implants produced with this technology have been successfully tested as slow-release devices for BMP2 *in vitro* and preclinically (Figs 52.17–52.19).^{114–116} As no other agents are added for coupling of the growth factor to the delivery vehi-

cle, the technology may have the potential to produce anatomically shaped degradable implants that are able to release a growth factor in a retarded manner and thereby regenerate tissue in a defined shape. Nevertheless, the promising preclinical results are still awaiting preclinical evaluation in larger animals and clinical testing for use in daily routine.

Cell-based approaches

The strategy of cell-based approaches is to obtain a small number of cells or a small tissue portion through a minimally invasive procedure and to expand these cells *ex vivo* to a volume that can be expected to form the desired amount and type of tissue. In order to produce some 10^8 – 10^9 cells, the initial population of

cells has to be highly proliferative and at the same time inducible into the desired phenotype.

Cell-based approaches are the most complex approaches of all endeavors in tissue engineering because, other than with the application of growth factors, both the host tissue and the biological quality of the seeded scaffolds that are implanted are variable biological success factors. The functionality of these biohybrid constructs depends very much on the survival and the performance of the implanted cells. Thus, besides the local conditions at the recipient site, both the initial cell source and the *in vitro* handling during cultivation of the constructs are important for successful applications.

Cell sources

In vivo, the cell sources for tissue repair are stem cells that are not yet terminally differentiated and have a high proliferative potential. In contrast to embryonic stem cells, which are pluripotent and can differentiate into a large range of different tissues, adult stem cells are already committed to a certain type of tissue such as mesenchymal stem cells. These stem cells can

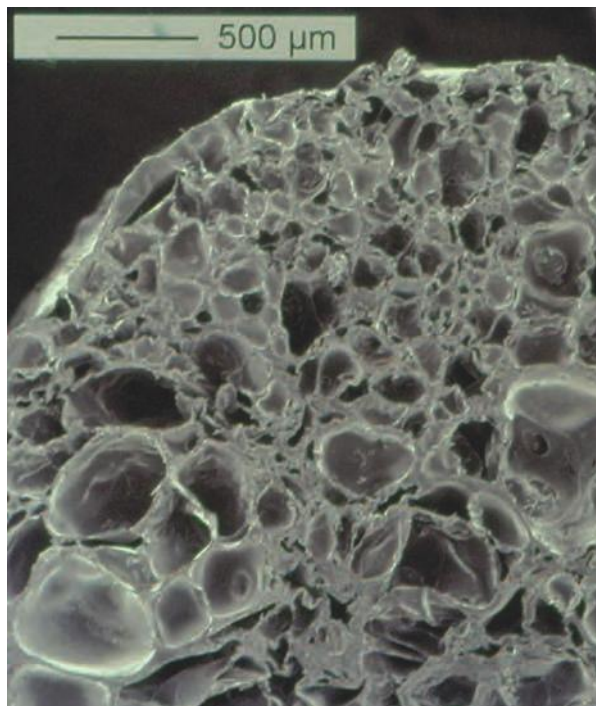


Fig. 52.17 SEM image of a gas-foamed poly DL-lactic acid implant.

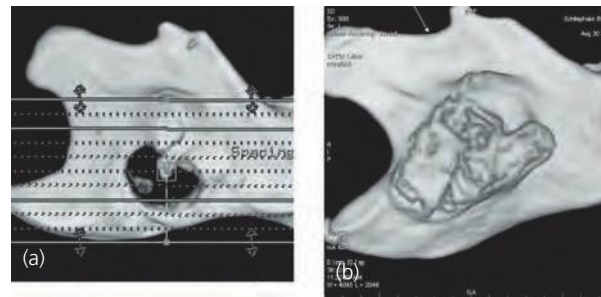


Fig. 52.19 Empty defect in the rat mandible after implantation of a blank PLA implant (a) versus bone regeneration after implantation of an implant containing 96 μg of BMP2 (b). (Reproduced with permission from Schliephake H, Weich H, Dullin C, Gruber R, Frahe S. Mandibular reconstruction by implantation of rhBMP in a slow release system of polylactic acid. *Biomaterials* 2008; 29: 103–10. Copyright © 2008 Elsevier.)

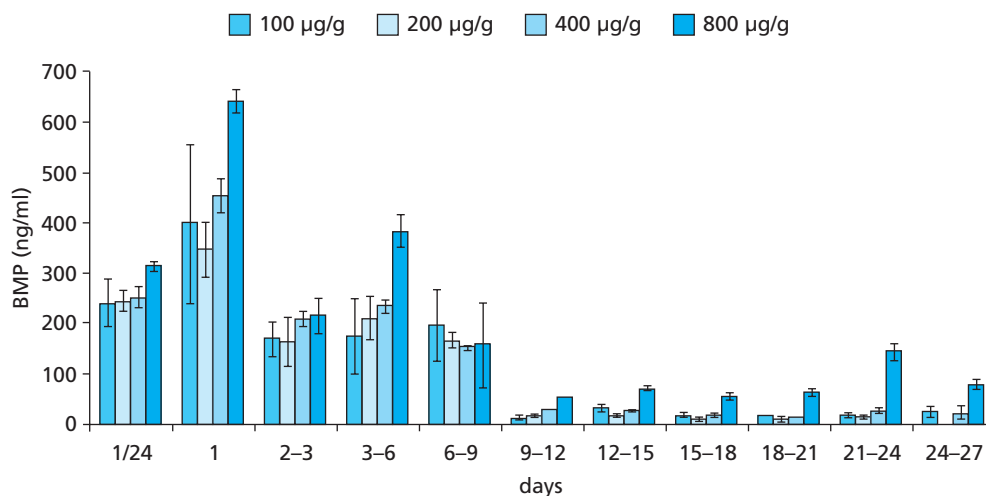


Fig. 52.18 Release kinetic of BMP2 from gas foamed PLA implants. (Reproduced with permission from Schliephake H, Weich H, Schulz J, Gruber H. In-vitro characterization of a slow release system of polylactid acid and rhBMP-2. *J Biomed Mater Res* 2007; 83: 455–6. Copyright © 2007 John Wiley & Sons, Inc.)

still differentiate into bone, cartilage, or fat cells, but are limited to tissues of the connective tissue lineage and, thus, are considered to be only multipotent. Several tissues, such as epithelium, bone marrow, liver, and fat tissue have stem cell niches. From these niches the stem cells can be obtained, isolated, and expanded *ex vivo* by *in vitro* technology. Terminal differentiation after sufficient expansion is then induced by conditioning supplements in the culture media.

Research on cell-based approaches is currently focused more or less on the regeneration of bone and cartilage tissue, thus mesenchymal stem cells are of particular interest. Mesenchymal stem cells are commonly obtained from bone marrow aspirates but can also be retrieved from fat tissue. The term mesenchymal stem cell is not quite correct for this type of adult stem cells and has undergone rephrasing several times. Currently, adult stem cells derived from bone marrow are called multipotent mesenchymal stroma cells (MSCs).¹¹⁷ Stem cells derived from fat tissue are referred to as adipose tissue-derived stem cells (ADSCs) and are considered to have very similar properties to bone marrow-derived stroma cells. Stroma cells are isolated as mononuclear cells from bone marrow aspirates by density gradient centrifugation and are separated from hematopoietic stem cells, which are always retrieved simultaneously, by their ability to adhere to the plastic bottom of the culture dishes (Fig. 52.20). Expansion is commonly done by passaging the cells before growth becomes subconfluent and subsequently replating them at low density. Under these conditions MSCs maintain a spindle-shape phenotype and form separate colonies of fibroblastoid cells (Fig. 52.21). The number of these colony-forming units (CFUs) is considered as a measure for the proliferative potential of the cell population. When the expanding cells are passaged at this stage up to 50 population doublings can be achieved without terminal differentiation.¹¹⁸ In mixed cultures, MSCs can be identified by surface markers such as CD106 or STRO1. These surface markers can be used

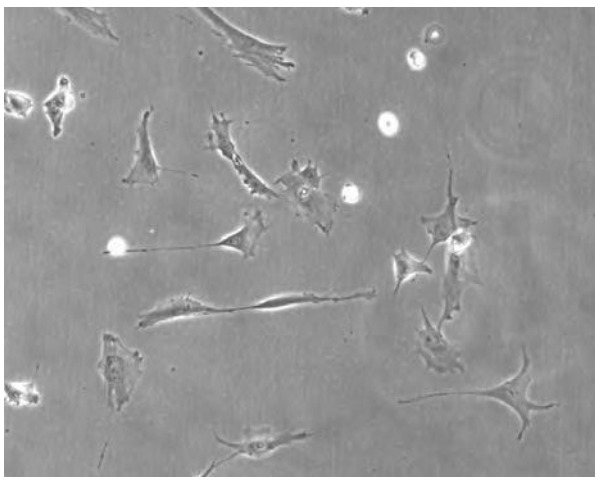


Fig. 52.20 Bone marrow stroma cells attached to the bottom of the petri dish 4 days after plating.

to further separate MSCs from the population of adherently growing cells by magnetic cell sorting (MACS) (Fig. 52.22) or by fluorescence activated cell sorting (FACS) (Fig. 52.23). After sufficient expansion

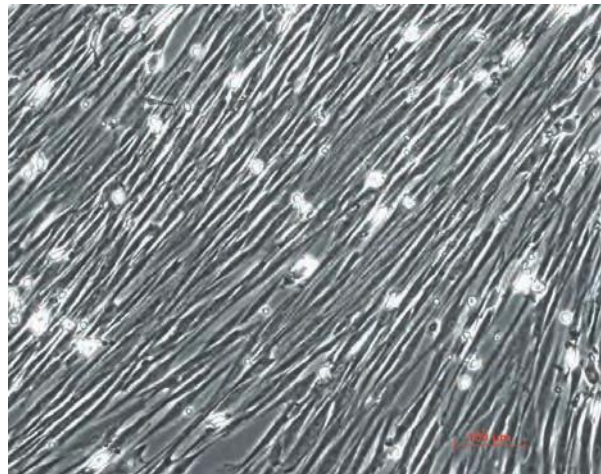


Fig. 52.21 Bone marrow stroma cells after 10 days with subconfluent growth of fibroblastoid cells.

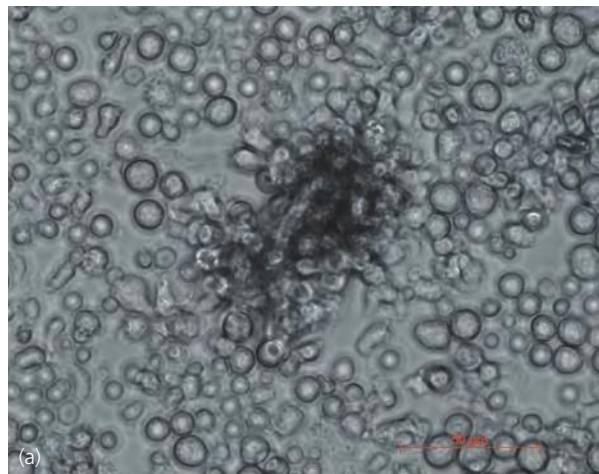


Fig. 52.22 (a) Rounded cells after incorporation of magnetic beads. (b) Cells are then sorted in a column in a magnetic chamber.

sion, osteogenic (ascorbic acid, dexamethasone, glycerophosphate) or chondrogenic supplements (ascorbic acid, dexamethasone, proline, pyruvate) are individually used to induce the respective differentiation of the expanded cells, depending on the tissue that is supposed to be regenerated.

Fat tissue for isolation of ADSCs is commonly derived by liposuction. The technique of tissue harvesting has considerable impact on the viability of retrievable ADSCs. The aspirate undergoes washing and subsequent digestion using collagenase treatment. After centrifugation and resuspension of the resulting pellets, the cells are plated at low density of (100 cells/cm²). CFUs resulting from this procedure can be expected to range between 0.1 and 5% of the retrieved nucleated cells.¹¹⁹

Epithelial stem cells are more difficult to isolate and expand. Gingiva epithelial cell isolates have been derived from trypsin digestion of gingival tissue and stem cell clones identified by size gated FACS after immunolabeling against $\beta 1$ integrin¹²⁰ or by MACS using antibodies against neurotrophin receptor p75.¹²¹ Clones with high colony-forming efficiency (CFE) are retrieved by these measures that are able to produce up to 30 population doublings.¹²⁰

Cell-based approaches have been tested *in vivo* in a large number of preclinical studies. In oral and maxillofacial surgery they focus on the regeneration

of bone, cartilage, and epithelial tissue. In bone tissue engineering, cell-seeded scaffolds have been evaluated using autogenous cells in various animal models and using human cells in a xenogenic model in immunocompromized rodents. While the autogenous models have almost consistently shown an enhancing effect on bone formation when compared with non-seeded scaffolds,^{122–127} xenogenic models using human cells have produced ambiguous results in that some reports have been unable to show that bone formation in scaffolds seeded with native bone marrow stroma cells has been superior to control scaffolds undergoing osteoconductive bone ingrowth.^{128–131}

Cartilage tissue engineering in oral and maxillofacial surgery is limited very much to the temporomandibular joint (TMJ). Some recent reports have dealt with tissue engineering of nasal cartilage^{132,133} but most of the current work is directed towards replacement of the TMJ disc and condylar cartilage.^{134–136} One of the major problems to overcome in tissue engineering of TMJ cartilage is the specific structural requirements of tissue engineered TMJ cartilage to withstand the *in vivo* forces as well as the fixation and connection of remaining ligaments. The mechanical properties of this type of cartilage have to enable the tissue not only to take pressure but also a high level of tension.¹³⁶ Thus, the characteristics of common hyaline cartilage that is used by many tissue engi-

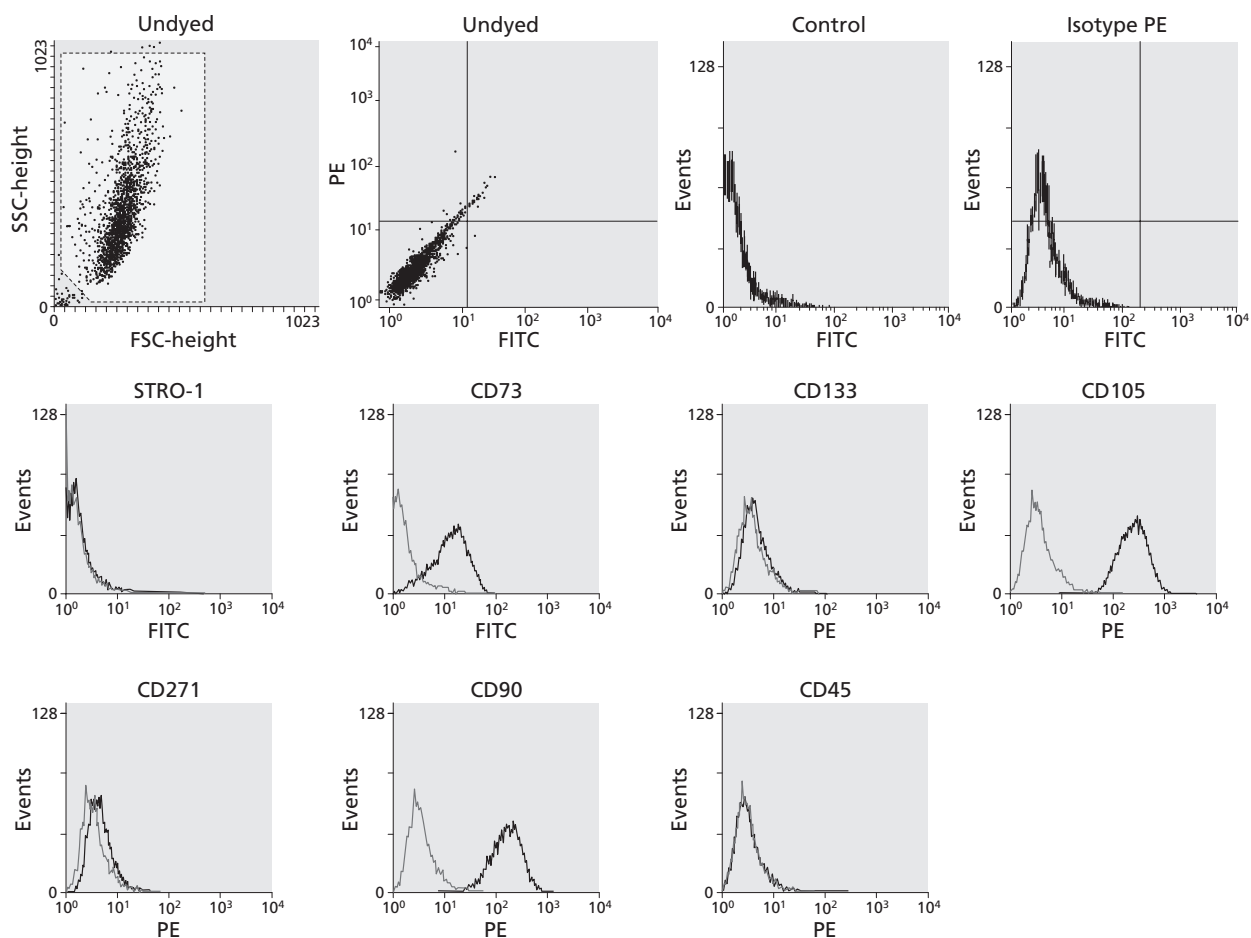


Fig. 52.23 Plot from a FACS analysis showing separation of multifunctional stroma cells according to their surface markers.

neering approaches to replace joint cartilage will not be appropriate to replace TMJ cartilage.¹³⁴

Engineering of epithelial tissue has moved from the use of epithelial monolayer sheets in the 1980s^{137,138} to more complex constructs that contain a stratified epithelium, a continuous basement membrane, and a fibrous connective tissue layer that supports the epithelium and gives three-dimensional stability during handling and healing. These engineered full-thickness oral mucosa constructs are commonly referred to as *ex vivo* produced oral mucosa equivalent (EVPOME).¹³⁹

Clinical applications of cell-based approaches in oral and maxillofacial surgery have been limited to the repair of bone tissue and epithelial defects. In bone tissue engineering, periosteal cells from the mandibular ramus and bone-derived cells from the maxillary tuberosity have been used to produce bone in sinus lift procedures and lateral rim augmentations in preimplant surgery.^{140,141} However, only 18 of 27 sinus lifts with periosteal cells seeded in polymer fleeces resulted in sufficient bone formation for secondary implant placement.¹⁴⁰ Moreover, resorption of tissue-engineered bone from periosteal cells in polymer fleeces was significantly higher than after sinus lifts using autogenous bone.¹⁴² In contrast, bone marrow stroma cells suspended in a fibrin matrix containing PRP as "injectable bone" without defined three-dimensional scaffold have been successfully used to enhance bone formation in alveolar bone distractions and in sinus floor augmentations.^{143,144} The fact that all patients in whom PRP and cells were used had been successfully treated compared to only 18 of 27 patients treated with polyglycolic scaffolds and cells alone suggests that the presence of growth factors may enhance the functionality of cell-based approaches, in that proliferation and vascularization of the seeded cells is enhanced after implantation in the *in vivo* environment. Cell-based approaches in combination with growth factors – in particular BMPs – have been used for flap prefabrication in major reconstructive procedures by implantation into the latissimus dorsi muscle and subsequent revascularized transfer to a segmental defect of the mandible (Fig. 52.24).¹⁴⁵ These procedures may not be cost



Fig. 52.24 Reconstruction of the mandible with a precultivated piece of bone in a titanium cage. (Reproduced with permission from Warncke PH, Springer IN, Wiltfang J, *et al.* Growth and transplantation of a custom vascularized bone graft in a man. *Lancet* 2004; 364: 766–70. Copyright © 2004 Elsevier.)

effective yet when compared to the established methods of revascularized tissue transfer for mandibular reconstruction, but they add to the knowledge of tissue engineering and may help to gather experience of how the principles of tissue engineering can be accommodated in the current strategies of maxillofacial reconstruction.

Clinical tissue engineering of epithelium using EVPOME (Fig. 52.25) preparations has been employed in intraoral applications such as vestibuloplasty, repair of superficial postablative mucosal defects, and for prelamination of free radial forearm flaps with subsequent transfer to the oral cavity.^{146–148} Clinical results have shown a good to excellent take rate with vascular ingrowth from the recipient bed with the total wound healing time being shorter than in the control defects covered with unseeded scaffolds only.^{118,148,149}

Recombinant cells

One of the weaknesses of cell-based approaches is the fact that the implanted cells have to rely on the host tissue to provide the biological signals and environmental factors that are necessary to revascularize the implanted constructs as rapidly as possible and at the same time maintain the biological quality of the implanted cells inside the scaffolds. This has prompted the idea to use gene transfer to the seeded cells that would allow for overexpression of the required growth factors which make them less dependent from the level of host tissue factors. Thus, the functionality of the implanted devices could be increased and the therapeutic outcome improved.

Gene transfer requires complex delivery strategies because extracellular DNA is not readily incorporated and not easily put into function in the target cells. The gene that is supposed to be transferred has to be incorporated into a so-called vector and this vector has to pass through the cell membrane. The most

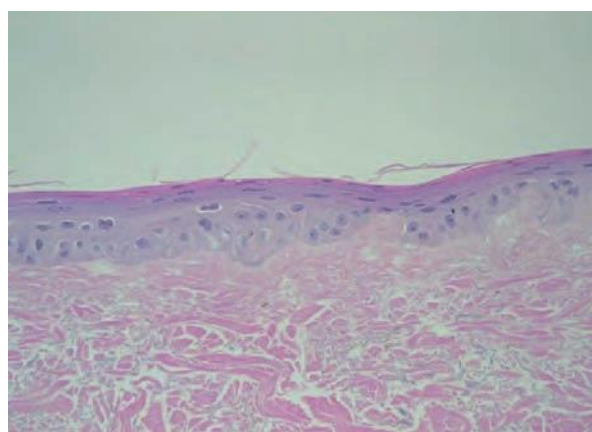


Fig. 52.25 EVPOME showing stratified epithelium on the surface after 11 days in culture. (Reproduced with permission from Izumi K, Feinberg SE, Ida A, Yoshizawa M. Intraoral grafting of an *ex vivo* produced oral mucosa equivalent: a preliminary report. *Int J Oral Maxillofac Surg* 2003; 32: 188–97. Copyright © 2003 Elsevier.)

frequently used delivery vehicles for genes are plasmids – small circular molecules of DNA. The DNA that encodes for the protein of interest is cloned into the plasmid. This plasmid commonly also contains a promoter area that activates the gene and an additional reporter gene such as green fluorescent protein (GFP) or β -galactosidase. These reporter genes encode for proteins that allow for following the plasmid after delivery (Fig. 52.26).

The introduction of plasmids into the cells is one of the main hurdles during gene transfer. The strategies for incorporation can be divided into non-viral delivery and viral-mediated transfer. Non-viral delivery is accomplished using chemical supplements such as calcium phosphate complexes that facilitate passage through the cell membrane. Alternatively, lipofection has been employed which uses liposomes that encase the plasmid vectors to pass through the cell membrane. Liposomes bind to the cell membrane and are incorporated through endocytosis. After intracellular digestion of the vesicle membrane the vector is delivered. Non-viral gene transfer has the advantage of being rather inexpensive, less complex and safer with respect to biological hazards than viral gene delivery strategies. However, transfection efficacy has been reported to be rather poor due to a low degree of integration of the transferred gene and – if successful integration has been accomplished – a rather short transient gene expression period.

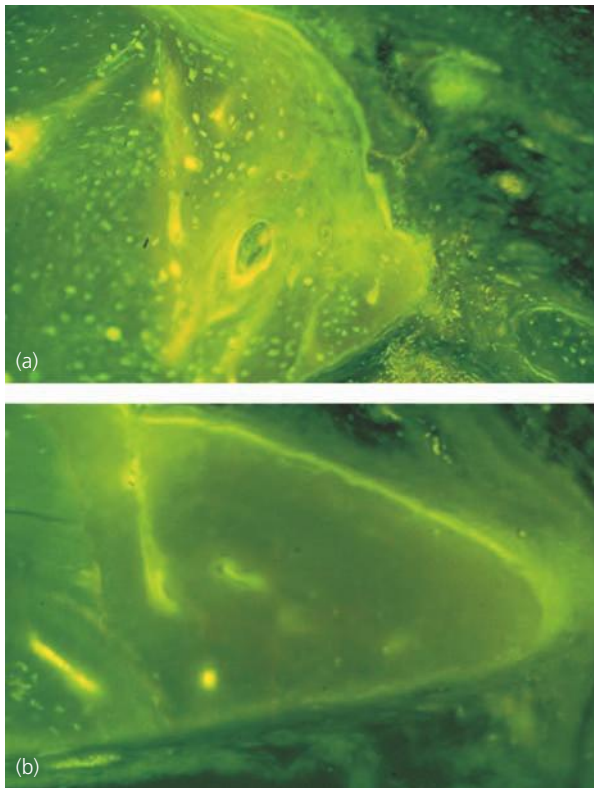


Fig. 52.26 Fluorescence microscopic image of rat calvarial defect edges showing cells positive for green fluorescent protein (GFP) after injection of medium containing adenoviral vectors containing the BMP2 gene and the reporter gene for GFP into the defect (a) compared to negative signals after injection of medium only (b).

Viral gene transfer uses replication-defective virus particles to manage the cell membrane passage and the functionalization of the transferred gene. After removal of pathogenic gene sequences from these virus particles, their ability to deliver genetic material to host cells is used to transfect the target cells with the respective gene. A wide range of viral vectors is available, including adenoviruses (AVs), adeno-associated viruses (AAVs), and retroviruses such as lentiviruses (LVs). While adenoviral vectors remain in the cytoplasm of the target cells, retroviral vectors integrate their DNA permanently into the genome. In this way, viral gene transfer using retroviral vectors is more effective than using adenoviral vectors. However, concerns have been raised that random integration of the transferred gene into the genome of the target cells might activate oncogenes and increase the biological hazard of this approach.

The use of gene transfer in tissue engineered devices in oral and maxillofacial surgery has been performed so far only in a few approaches at the pre-clinical level. Experimental studies have used genetically modified human marrow-derived stroma cells¹²⁹ and mesenchymal stem cells¹⁵⁰ overexpressing BMPs. In these studies, the transfected cells produced significantly increased amounts of bone compared to the native cells and the reporter gene-transfected cells in both ectopic¹⁵⁰ and orthotopic sites.¹²⁹

In TMJ cartilage tissue engineering, successful attempts have been made to engineer an osteochondral condylar implant in ectopic sites in rodent models using biphasic scaffolds, transduced fibroblasts (BMP-7), and chondrocytes.¹⁵¹ Also, adenovirally stimulated (AdBMP-2) mesenchymal cells from periosteum and bone marrow have been used successfully to repair TMJ cartilage defects.¹⁵²

Epithelial cells have been transduced using lentivirally mediated gene transfer¹⁵³ and particle bombardment (gene gun), not for tissue engineering purposes, however, but as a therapeutic *in situ* approach for diseased skin and neoplasms.

In vitro technology

The successful *ex vivo* production of a tissue equivalent is not only based on the isolation and expansion of the respective populations of cells with specific properties but also on the appropriate *in vitro* technology to arrange and distribute these cells inside the three-dimensional scaffolds. Moreover, nutrient supply and removal of metabolic end products have to be provided during extended periods of cultivation. The use of *in vitro* technology is based on the specific requirements of the type of tissue equivalent that is intended to be produced. The production of mucosa equivalents, for example, requires simultaneous cultivation of epithelial cells and gingival fibroblasts in thin scaffolds and conditions of cultivation have to allow for the occurrence of a stratified epithelial layer on the surface. On the other hand, the thin constructs

do not depend on an active media exchange within the scaffold as they can rely on diffusion for metabolic exchange. Cultivation of EVPOME is, therefore, performed on permeable membranes floating at an air/liquid interface for a period of 7 days after they have formed a continuous epithelial monolayer on the scaffold during submerged cultivation for 4 days.¹⁴⁸ In this way, multilayered constructs can be produced that quite closely resemble native mucosa in terms of morphological, histochemical, and genetic patterns.^{154,155}

Production of seeded scaffolds for bone tissue engineering is different as it is commonly performed in large volume scaffolds that are seeded with only one type of specialized cells. Currently, a uniform

distribution of these bone-forming cells inside the scaffolds is considered to be advantageous for *in vivo* osteogenesis.¹⁵⁶ Technical support is required, therefore, to achieve complete seeding of the whole scaffold volume and also to provide continuous exchange of culture media in the center of the seeded devices when long-term cultivation is intended to produce a mature tissue equivalent.

In general, the strategies for seeding and cultivation of cells in scaffolds can be divided into static and dynamic approaches. Conventional static seeding of biomaterials with cells has been performed by droplet seeding followed by static culturing with media exchange in intervals of 2–7 days. However, in bone tissue engineering, droplet seeding of mesenchymal cells has been shown to result in non-homogeneous distribution of cells inside the scaffolds (Figs 52.27 and 52.28);¹⁵⁷ subsequent static cultivation had to rely on diffusion for the exchange of culture media in the central portions of the scaffolds, which resulted in apoptosis of cells in these scaffold areas. In order to improve the distribution and the viability of seeded cells across the entire scaffold volume, technologic approaches for dynamic cell seeding and culturing were developed. Low pressure or vacuum have been employed during seeding to improve cell penetration into the scaffolds and increase cell density in the central parts of the scaffold.^{158,159}

Dynamic culturing has been developed in many ways and individual applications have been used to satisfy the requirements of the specialized cells of the various tissues or organ composites to be produced. Traditional methods of agitated cultures using rotary vessels or spinner flasks^{160,161} are being replaced by perfusion bioreactor systems, which produce a constant medium flow through the scaffolds, that provides continuous nutrient supply and removal of

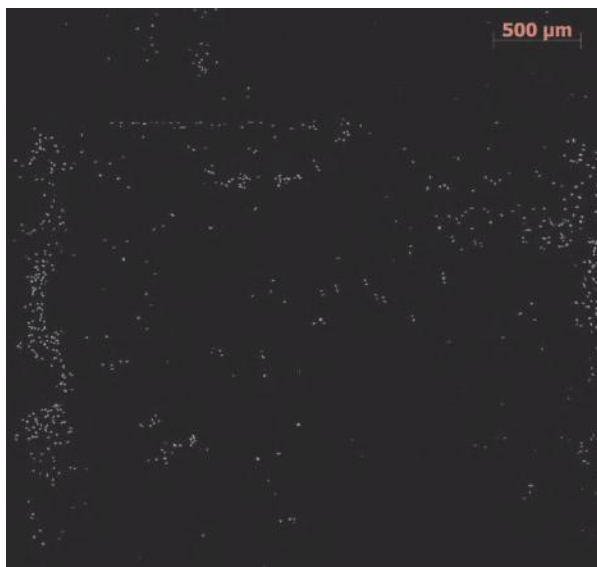


Fig. 52.27 Distribution of MSCs seeded on to a porous CaCO_3 scaffold with non-homogeneous distribution of the cells in a cross-section through the scaffold. Cell nuclei are stained blue.

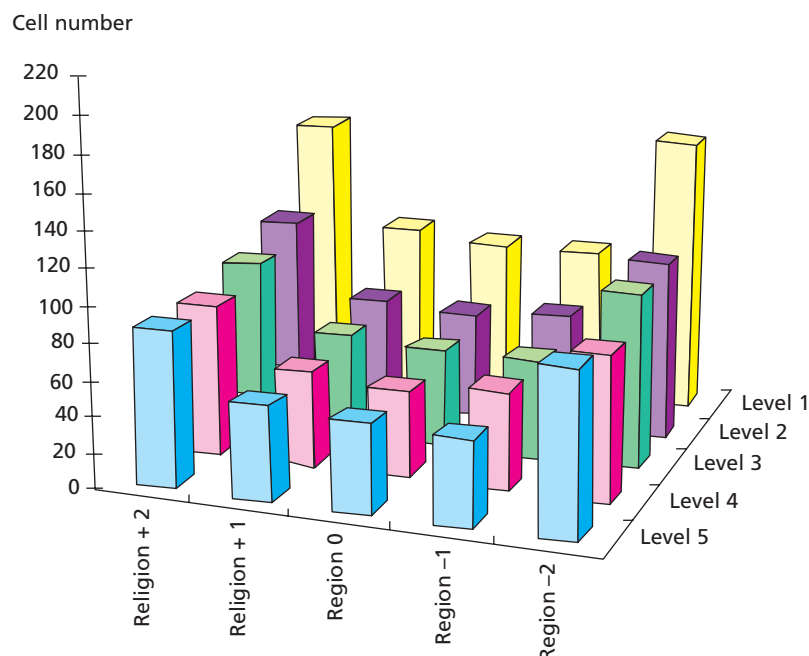


Fig. 52.28 Numerical evaluation of cells in a CaCO_3 scaffold. Note concentration of cells at the edges of the block with a relatively void center.

metabolic end products (Fig. 52.29).¹⁶² Recent developments include micromechanical manipulations in advanced reactor models.^{163,164} Dynamic seeding and culturing of human mesenchymal stem cells in perfusion bioreactor systems has produced more uniformly distributed cells and ECM proteins than static culturing.^{165,166} Dynamic culturing in spinner flasks has been reported to enhance the activity of alkaline phosphatase as a marker of bone-specific activity and other markers of osteogenic differentiation^{167–169} compared to static conditions. Culturing of bone marrow stromal cells in flow perfusion bioreactors has improved mineralized matrix deposition by increasing fluid shear forces^{167,168} and increased the content of VEGF.¹⁶⁹ Too high flow volume, however, can also be detrimental for cell survival and differentiation.¹⁷⁰

Beyond the beneficial effect of dynamic seeding and culturing on cell functions *in vitro*, the efficacy of these techniques with respect to *in vivo* bone formation has not been clearly proven yet. Experimental studies on static vs dynamic culturing of small volume scaffolds with human trabecular bone cells have shown that static vs dynamic culturing conditions did not affect the degree of bone formation,¹⁷¹ whereas dynamic seeding of rat bone marrow stroma cells produced more bone *in vivo* than static culturing.¹⁵⁸ The positive effect of dynamic culturing on cell survival inside large-volume scaffolds has been recently shown *in vitro*.¹⁷² Preclinical studies using this type of advanced *in vitro* technology now have to show that cell survival and graft function are also improved *in vivo*.

Future perspectives

A number of basic questions about the application of tissue engineering in clinical therapy are still unanswered. For cell-based approaches these questions

relate to the simulation of tissue-like conditions with respect to scaffold nature, biological signaling for cell/tissue development during *ex vivo* cultivation, and nutrition and cell survival after implantation into the *in vivo* environment. Major leaps in *in vitro* technology will be required to construct a tissue-like structure that has the quality of an autogenous graft, which is still the gold standard. Moreover, in situations that require revascularized tissue transfer, it is even more difficult to imagine the construction of a tissue-engineered scaffold that is equivalent to the composite flaps of today's standard, including vessels, bone, and soft tissue. Looking back, the vision of getting cells and scaffolds together to form a tissue may have been too simplistic. Future efforts in the clinical application of cell-based approaches in oral and maxillofacial reconstruction will have to be more complex by combining more than cells and scaffolds and trying to create active scaffolds that resemble the function of ECM more closely. By designing engineered microenvironments that bring physical regulatory factors into play, novel approaches can be developed that provide adequate interactive stimuli for organic tissue development. Research in biomaterials that allow for cell attachment and growth, and at the same time can store and release biological signals at a physiological level, may help to form constructs that take part in the natural remodeling activity during regeneration and support survival and function of the seeded cells. In this way, the quality of autogenous grafts could be improved.

The complexity of problems with the use of cells has resulted in the fact that the application of cell-based approaches is currently less advanced in clinical tissue engineered reconstruction than the use of growth factors. Recombinant growth factors will probably have the widest clinical application in bone reconstruction in the immediate future. Their effective use in terms of costs and safety will be linked to

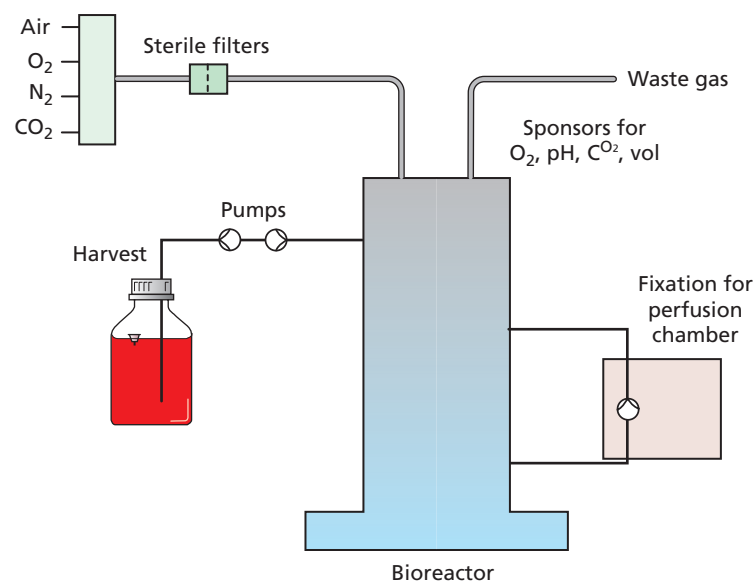


Fig. 52.29 Schematic drawing of a bioreactor.

the development of appropriate delivery vehicles. Additionally, the simultaneous use of multiple growth factors that act synergistically and over-additively in tissue regeneration will help to lower the amount of individual growth factors to be released. Future research will have to elucidate this network of interactions and simulate their interplay with the help of controlled-release carriers.

"Still, to date tissue engineering has not lived up to its promise. In a very real sense, it has underdelivered, although the potential is still there."¹⁷³ This statement makes clear that from the clinical perspective not many of the numerous promises that were anticipated in the early years of tissue engineering have yet become reality in our daily routine. However, with ongoing research the growing body of knowledge creates a much better understanding of tissue repair and how tissue function can be enhanced or supported. Nevertheless, all researchers and clinicians involved in this process are aware that this will be a slow-growing evolutionary process rather than a revolutionary breakthrough.

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Chapter 53

Cosmetic Facial Surgery

Joe Niamtu

The chapter is a comprehensive overview of different cosmetic surgical procedures in the maxillofacial region to enhance the soft tissue esthetics. Minimally invasive techniques as well as more demanding cosmetic surgical procedures are described and illustrated. The author claims that over the last four decades dentistry has been transformed from a specialty primarily treating disease to a specialty with focus on esthetics and reconstruction.

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The expansion of this topic in oral and maxillofacial surgery has been exponential. It would probably not have been appropriate to include this topic in an oral and maxillofacial surgery text 15 years ago, but much has changed in this specialty in a decade. To appreciate this change we must understand the changes that have occurred in dentistry. Over the past four decades dentistry has transformed from a specialty that primarily treated disease (dental caries) to a specialty with the main focus of esthetics and reconstruction. Orthodontics, periodontal plastic surgery, cosmetic dentistry, and implantology all share the common focus of esthetics.

Oral and maxillofacial surgery has also progressed and includes cosmetic facial surgery in its contemporary scope.¹ In the USA cosmetic facial surgery represents core curriculum in most oral and maxillofacial surgery programs, is part of the board exams, and is covered by oral and maxillofacial surgeons' malpractice insurance.

Although the progression has been rapid, it has been hard fought with competing specialties.² Despite

this opposition, oral and maxillofacial surgeons have won the privilege to perform cosmetic surgery in virtually every state in the USA in which it was contested by competing specialties. With this successful legislation and the contemporary training, many oral and maxillofacial surgeons have included cosmetic facial surgery in their practices.

Most oral and maxillofacial surgeons will not choose to limit their practices, and this chapter is designed as an overview of contemporary and common techniques. Due to the limited scope of this chapter it is impossible to cover all cosmetic procedures in great detail. The main goal of writing this chapter is to provide a brief overview of common cosmetic facial procedures to elucidate the basic techniques and provide advice on avoiding complications. The descriptions are time tested in the author's practice and have produced a low complication rate and happy patients. These descriptions are subject to the individual surgeon and obviously vary greatly from surgeon to surgeon. There are many ways to approach any procedure and each practitioner must decide personally what works best.

Minimally invasive cosmetic facial surgery procedures

As technology has progressed, minimally invasive cosmetic procedures have surpassed surgical procedures on an international basis. Botox and injectable fillers represent the most commonly requested cosmetic procedures.

Botox (botulinum toxin A)

The application of Botox in cosmetic facial surgery represented a true paradigm shift where significant effacement of facial lines and wrinkles could be treated non-surgically. Botox causes temporary paralysis of skeletal muscle by preventing the release of acetylcholine at the motor end plate.² This effect will persist about 90 days in the average patient.

There are multiple botulinum toxin preparations worldwide, and differences exist in dilution and units given; this chapter will only describe Botox Cosmetic as marketed in the USA. A Botox vial contains 100 units of toxin and can be diluted with various amounts of saline. The author dilutes each vial with 5 ml of preserved saline. This amount makes five syringes that contain 20 units each, which is the average amount used for a single treatment area.³ It is important to keep in mind that it is the number of units delivered and not the actual dilution that is critical.

The most common areas treated are the frown lines (brow depressors), horizontal forehead lines (frontalis), and crow's feet (lateral orbicularis oculi). The single biggest tip to heed when injecting Botox is to not inject closer than 1 cm to the orbital rim. This provides a safety margin to prevent the toxin from diffusing into musculature that it is undesirable to affect.

Treating the brow depressors

The author uses a Leur-Lock 1 ml tuberculin syringe with a 32 gauge needle for all injections. To treat scowl lines a total of 20 units is injected for the average patient: 5 units are injected into the procerus area,

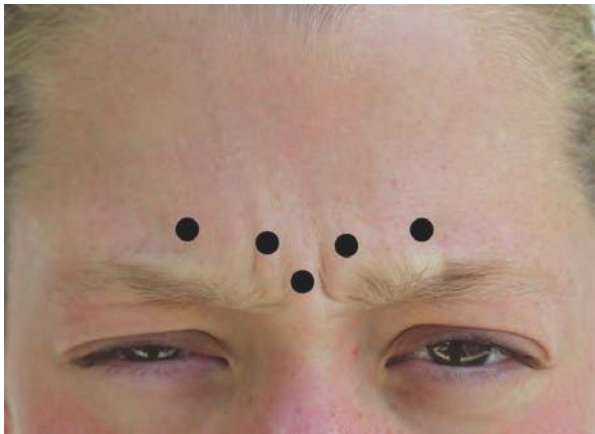


Fig. 53.1 Five injections across the brow depressors (a total of 20–25 units) adequately treat the “frown lines” in most patients.



Fig. 53.2 Multiple injections for an average of 20–25 units will adequately prevent the horizontal forehead wrinkles when flexing the frontalis muscles.

4 units are injected into each corrugator supercili area, and 3 units are injected into each lateral orbicularis oculi area (Fig. 53.1).

The frontalis is treated by injecting 2–3 units across the areas of the forehead that wrinkle with animation (Fig. 53.2). Since Botox will diffuse 10–15 mm circumferentially, the goal is to treat the entire forehead by spacing the injections. It is advisable to reduce the treated area above the lateral brow in patients with redundant eyelid skin (dermatochalasis). Failure to do so can accentuate the droopy lid skin because the frontalis deactivation prevents the patient from being able to lift the brow. In these patients it is better to taper out the treated areas so the area above the lateral brow is very lightly treated. If the patient desires less movement, more Botox can be injected at a later date. Men (having more muscle mass) or women with high hairlines may require more than 20 units for successful treatment.

Crow's feet wrinkles are treated by injecting 3 units of Botox into the lateral orbicularis oculi in three areas in a semi-lunar fashion (Fig. 53.3). It is important to avoid the superficial vessels in this area to prevent bruising.

Perioral Botox

Using Botox around the mouth is very different to its use in the upper face and the results are much less dramatic. In addition, overtreatment of the orbicularis oris musculature can cause significant dysfunction. The primary use of perioral Botox is to reduce vertical rhytids: 1–2 units of Botox are injected very superficially (almost intradermally) in the area across the upper and/or lower lip (Fig. 53.4). The patient is first asked to pucker to show the wrinkles that need injection. Since this area is so sensitive to overtreatment, it is advisable only to inject a single unit in each area, then have the patient return for reinjection a week later. The author most commonly injects this area in conjunction with filler treatment.



Fig. 53.3 Three injections of 3 units each in the lateral canthal region will improve “crow's feet” wrinkles in the average patient.



Fig. 53.4 Improvement of the upper and lower lip rhytids after 8 units in each lip performed in two sessions of 4 units across each lip.

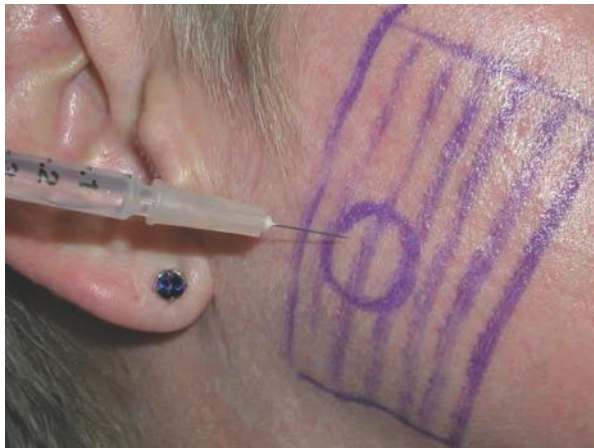


Fig. 53.5 5–15 units of toxin are injected in the most evident palpable muscle mass during clenching. This is repeated every 2 weeks until the result is achieved.

Botox for masseteric hypertrophy

Botox injection is commonly used for esthetic and functional treatment of the masseter muscles. The treatment involves injecting the muscle mass until the size and/or bite force are decreased. Prior to injection, the patient is asked to clench their teeth, then the surgeon palpates and injects the most prominent areas of palpable and visible mass. Usually 5–10 units are injected in several areas on each side, then the patient returns 2 weeks later for reinjection if necessary (Fig. 53.5). This is continued over several sessions until the desired end point. A 32 gauge, 13mm needle is used, burying the entire length for deep injections.

Figs 53.6–53.10 show typical before and after Botox treatments.



Fig. 53.6 A patient before and 1 week after Botox injections to the brow depressors to improve the frown lines.



Fig. 53.7 This patient is shown elevating her brows before and 2 weeks after 20 units of Botox across the frontalis muscles.



Fig. 53.8 This male patient is shown before and 2 weeks after 12 units of Botox in each lateral canthal region.



Fig. 53.9 This patient is shown before and 1 month after multiple injections of Botox into each masseter. The patient is clenching in both pictures.



Fig. 53.10 This female patient is shown before and 1 month after two injection sessions of 4 units across each lip. The total of 8 units in each lip has reduced the severity of the wrinkles.

Injectable facial fillers

Differences exist in the availability of fillers in various countries. Europe, Canada, and Australia have pioneered fillers that have only recently become available in the USA, and currently use others that are not yet available in the USA. A decade ago, bovine-based collagen fillers were the principle fillers in use, but there are now many more options of injectable materials. The scope of this chapter prohibits in-depth description of each filler. Every filler has specific advantages and disadvantages. It is important to understand the material composition of each filler, its longevity, and most importantly the proper tissue plane for injection. The latter is perhaps the most critical factor as different fillers are intended for superficial dermal injection, mid to deep dermal injection, and subdermal injection.

Filler technique for nasolabial folds

Cutaneous injection can usually be performed with topical anesthesia only, although some patients may require supplemental local infiltration.⁴ Patients may think that the filler will make their folds disappear so it is important that they understand that the filler will blunt the folds and make them less noticeable, but it won't eliminate them.

Since many tissue planes come together at the nasolabial fold, injected filler sometimes has a tendency to flow laterally; if this occurs it can actually make the fold larger. If the surgeon notices lateral flow it is important to withdraw the needle and continue medial to the fold.

Correct tissue placement of most fillers is in the mid to deep dermis. When the filler is in the correct plane the wrinkle or fold should visibly improve as the filler is injected. Failure to see immediate correction may mean that the needle is too deep and the filler is spreading laterally in the subcutaneous plane instead of filling the dermis. Filling techniques include serial puncture, which involves multiple sticks with filler deposition along the fold or wrinkle, or linear

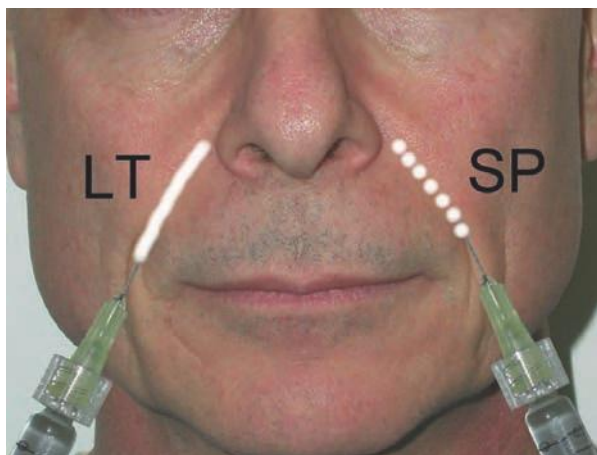


Fig. 53.11 Linear threading and serial puncture techniques are common methods used in filling facial lines, wrinkles, and folds.

threading, which involves a single puncture, threading the filler as a line while withdrawing the needle.⁵ Fig. 53.11 shows the described filling techniques and Fig. 53.12 shows a before and after picture of nasolabial filling. Individual skin wrinkles are treated basically the same way, but usually more superficially in the dermis.

Massaging any injected area (wrinkle or lip) helps coalesce the injected filler and thereby reduces the lumpy feeling or appearance. All but the youngest patients usually require a syringe for each fold. One problem that exists is that for financial reasons many patients will only want to pay for a single syringe split between both folds. Failing to use the proper amount of filler will result in a subclinical result and an unhappy patient.

Filling the lips

This section will provide a basic discussion on filling the lips. The main concerns of lip rejuvenation are restoration of definition and volume, and improvement of vertical rhytids (lipstick lines). Obviously, other applications exist, such as perioral filling, and filling the commissures and mentolabial folds, but these are beyond the scope of this chapter.

The youthful lip has pleasing definition with an angular Cupid's Bow, a plump white roll, and defined philtral columns. The aging lip loses this definition and becomes flat and devoid of definition.⁶ Every patient is different when it comes to lip rejuvenation. Younger patients may only require slight plumping, whereas older patients may need plumping, Cupid's bow enhancement, and vertical rhytid filling.

Defining the vermillion/cutaneous junction

The youthful upper lip has a well defined Cupid's bow area with the shape of a lazy "m" configuration and the lower lip has more of a curvilinear vermillion/cutaneous junction. The upper lip constitutes one third of the total lip volume and the lower lip has the ideal proportion of two thirds of the total lip volume (Fig. 53.13).

To reproduce the white roll the filler needs to be injected into the potential space that exists between the lip mucosa and orbicularis muscle. It takes some experience to appreciate this tissue plane. When the needle is in the proper submucosal potential space,



Fig. 53.12 This patient is shown before and after 1ml of Jevoderm injected intradermally into each nasolabial fold.

the filler will flow antegrade and retrograde with little syringe plunger pressure. If increased plunger pressure is required or if the filler creates a lump instead of flowing freely, the needle is in the incorrect plane (Fig. 53.14).

The filler should be injected conservatively in the center of the lip to create the “v” inferior to the philtrum. Next, the lateral limbs of the upper lip white roll are augmented. The filling of this area does not usually require filling all the way to the commissure areas. Most females look best with only the central third of the lip (the “pucker” area) augmented. The curvilinear white roll of the lower lip is likewise filled in the same manner (Fig. 53.15).

Creating pout: restoring lip volume

Simple volume is created in the lips by injecting “pillows” of filler deeper in the lip (Fig. 53.15). The target for volume filling is to inject the filler one third to one half the thickness of each lip. This is done at the level of the wet/dry line. For the average patient a single syringe will sufficiently fill and outline both lips, but some patients will require additional product.

Treating vertical lip rhytids

Some patients present with the desire to fill lipstick lines but do not desire bigger lips. The individual lines can be filled but the surgeon must be careful not to create a series of “speed bumps” across the lips. When filling solitary lines a less viscous filler such as Cosmoderm or Juvederm (Allergan Inc, Irvine, CA)

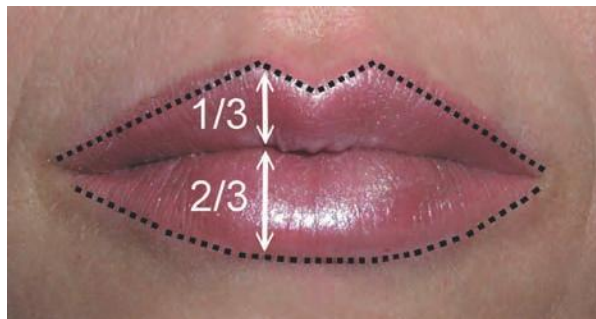


Fig. 53.13 The esthetic upper lip is about one third of the lip volume while the lower lip represents about two thirds of the total volume.



Fig. 53.14 (a) The correct needle position for filler injection in the potential space under the lip mucosa. Injection in this plane should be free flowing with minor syringe pressure. (b) “Lumping” of the injected filler in the incorrect plane. Increased syringe pressure without free filler flow signals incorrect placement.

may be used. These less viscous fillers can be injected with great accuracy and can fill isolated rhytids without gross lip volume. By employing the white roll definition technique mentioned earlier, the lipstick lines are often simultaneously improved from the subtle stretching of the lip skin.

Figs 53.16–53.19 show before and after pictures of lip enhancement techniques.

Cervicofacial liposuction

Liposuction is not a procedure to reverse obesity but rather to deal with isolated fat deposits of the neck and face. The submental, jowl, and lateral neck regions are the areas most commonly treated. Patient selection is critical for successful outcomes. Younger patients with isolated submental fat and tight neck skin are the best candidates. Patients with skin laxity

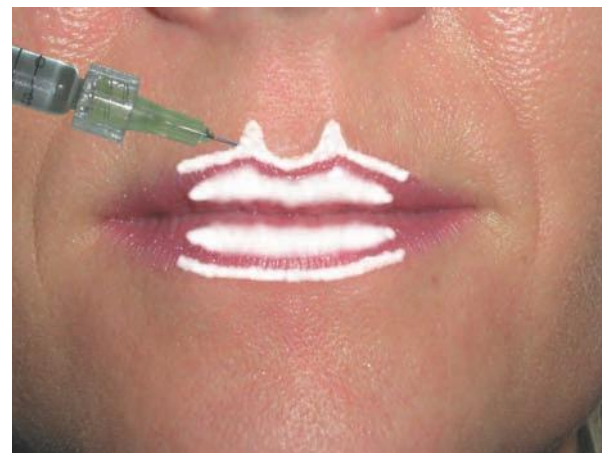


Fig. 53.15 The areas of filler injection used in the lips. Outline of the white roll, philtral column bases are illustrated as thin lines whereas the deeper fill for pout is shown as larger depositions.



Fig. 53.16 This patient was treated with one syringe of Cosmoplast in each lip.



Fig. 53.17 This patient was treated with a single syringe of Restylane split between both lips.



Fig. 53.18 This patient was treated with a single syringe of Juvederm Ultra split between both lips.



Fig. 53.19 The pictured patient was treated with 1.6 ml (two syringes) of Juvederm Ultra Plus.

of the neck must be approached with caution as the redundant skin may become more pronounced after fat removal. Although liposuction can induce skin tightening, it is much less dramatic in older patients. Patients that exhibit significant neck skin excess are candidates for facelift surgery. Another caveat of liposuction is overly aggressive removal of fat. Fat is often viewed as a cosmetic enemy but oversuctioning the neck will cause extremely unattractive and difficult to correct changes in the anterior neck. Removing too much subcutaneous fat or not leaving an adequate fat cushion on the dermis can cause the skin to tether down on the platysma muscle, producing a webbed deformity. The author prefers to stay on the subcutaneous plane when performing neck liposuction. Some surgeons advocate removal of subplatysmal fat and it can, at times, be beneficial but can also result in a submental concavity known as a “cobra” deformity. Removal of subplatysma fat should be reserved for experienced surgeons.

Procedure

The procedure can be performed with local anesthesia only or with supplemental intravenous sedation. The patient is marked in an upright position with a surgical marker to outline the fat deposits in the anterior neck and jowls as well as the inferior border of



Fig. 53.20 The subcutaneous fat deposits in the superficial jowl and submental region are characterized in this diagram.



Fig. 53.21 Tumescent local anesthesia is injected in the subcutaneous plane. This provides anesthesia, hemostasis, and hydrodissection. After waiting 10–15 minutes, the fat is suctioned.

the mandible and the submental crease. Fig. 53.20 shows the usual fat deposits and surgical markings for cervicofacial liposuction.

Tumescent anesthesia is mixed to a concentration of 1% lidocaine and 1:1 million epinephrine. This can be made up by adding 1 ampoule of 1:1000 epinephrine and 50 ml of 2% lidocaine to 1 liter of normal saline. The solution is injected in the subcutaneous plane around all areas to be treated. Generally 40–100 ml are injected in the submental area and about 20–30 ml in the jowl area (Fig. 53.21). The solu-

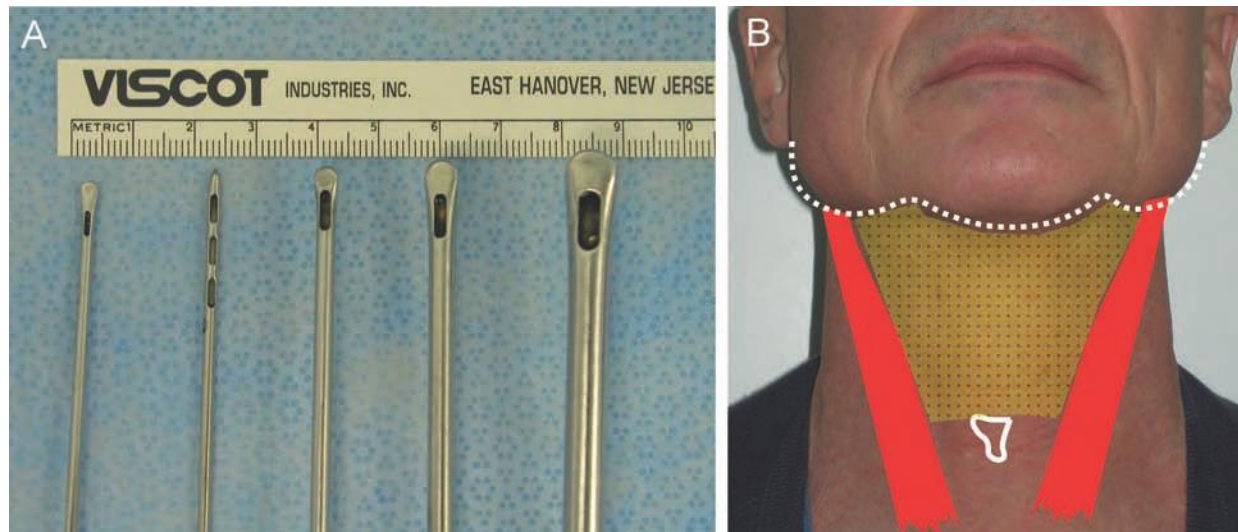


Fig. 53.22 The common cannulas used in facial liposuction and the approximate borders of the submental fat deposits.



Fig. 53.23 Jowl fat being suctioned from an earlobe incision. This is a safe surgical area in the subcutaneous plane.

tion is injected until the tissue turgor is “as hard as wood” and the operator needs to wait a sufficient time until the tissues are well blanched before beginning.

A stab incision is made in the submental crease with a number 15 scalpel blade to the subcutaneous plane and the liposuction cannula is inserted. The author prefers to begin with a 1 mm multiport cannula and progress to larger sizes, and uses simple wall suction for the face. The actual liposuction process involves a rapid and reciprocating in and out movement of the cannula. The process should also involve a criss-cross technique where the areas are suctioned from multiple vectors to increase the efficiency of fat removal. The area traditionally suctioned is from the mandibular border to the thyroid cartilage, between the medial borders of the sternocleidomastoid muscle (Fig. 53.22). This area is quite safe if the surgeon stays in the subcutaneous plane. The incision is then closed with several 5/0 gut sutures.

The safest means of approaching the jowl fat deposits is from an earlobe incision. A stab incision is made at the junction of the inferior lobe and cheek skin. The cannula is inserted in the subcutaneous plane to the jowl area to be treated (Fig. 53.23). This area must not be overtreated and patient expectations must be minor to moderate. Figs 53.24 and 53.25 show patients treated with liposuction only. The author frequently performs chin implant surgery on liposuction patients, which can greatly enhance the profile result (Fig. 53.26).

Midface implants

The midfacial area is perhaps the most overlooked area of facial rejuvenation. As we age, the midface becomes ptotic and gaunt and what was the cheeks becomes jowls.⁷ Although there are numerous ways to rejuvenate the midface, facial implants carry

numerous advantages. They come in many anatomic shapes and sizes, are a permanent solution, are reversible, are easily placed and removed, and look and feel natural. In general, most patients require



Fig. 53.24 This patient was treated with submental and jowl liposuction only.



Fig. 53.25 This patient was treated with submental and jowl liposuction only.



Fig. 53.26 This patient was treated with submental and jowl liposuction in addition to a silicone chin implant.



Fig. 53.28 The subperiosteal dissection for cheek implants includes the anterior maxilla and crosses the malar region to taper off over the zygomatic arch.

augmentation in one of three areas or a combination of these areas. For the patient that wants more of a zygomatic, “high cheekbone” look the malar shell implant is considered. For those patients requiring a more maxillary augmentation the submalar type implant is recommended. When patients would benefit from both malar and submalar augmentation, the combined submalar implant is used (Fig. 53.27).

Cheek implant technique

Cheek implants can be placed with local anesthesia and/or intravenous sedation. They are always placed in the subperiosteal plane, although it is common for the lateral portion of the implant to lie over the masseteric tendon. Local anesthesia is infiltrated over the anterior and lateral maxilla and over the medial zygomatic region from a transcutaneous approach. Intraorally, several milliliters of local anesthetic are also infiltrated in the mucosa above the canine tooth. An incision is made in the sulcular region above the canine tooth to the subperiosteal plane. Using a periosteal elevator, dissection is performed over the anterior maxilla and the malar region with the dissection tapering off over the zygomatic arch (Fig. 53.28). Care is taken to avoid and protect the infraorbital nerve. The dissection should taper off as it reaches the zygomatic arch.



Fig. 53.27 The common implants used by the author include (a) the submalar implant for anterior maxillary fill, (b) the malar shell implant (for “high cheekbones”), and (c) the combined submalar implant for augmentation of the anterior maxillary area as well as the malar area.

The larger implants require inferolateral dissection to accommodate the implant. It is not uncommon to require dissection over the origin of the masseteric tendon at the lateral malar region, and it is not a problem for a portion of the implant to cover this area. The dissection pocket need only be slightly larger than the actual implant. Making the pocket too large increases mobility. Although chin implants should always be fixated, this is rarely done with cheek implants as experience has shown that it does not affect the result. After dissection, the pocket is irrigated with an antibiotic solution and the implant placed in the pocket (Fig. 53.29). It is critical to make sure the implant lies passively and the tail is not folded or bent. The incision is closed with 4/0 gut suture. The patient is asked to refrain from significant oral function for several days and is warned that smiling, puckering, or speaking may be slightly impeded for 1–2 weeks. Figs 53.30–53.32 show before and after pictures of patients with midface implants.

Cosmetic blepharoplasty

The periorbital tissues frequently show signs of aging earlier in life than does the lower face. Eyelid aging



Fig. 53.29 The implant pocket should be only large enough to accommodate the implant which is placed through the intraoral incision. Too large a pocket can cause implant mobility.



Fig. 53.30 This patient underwent facelift, laser skin resurfacing, and placement of a medium submalar silicone cheek implant.

changes are manifested by pseudoherniation of periorbital fat, loose and wrinkled lid skin, lateral hooding, and brow ptosis.⁸ If brow ptosis is significant, it should be addressed with a brow lift. The generalized goal of blepharoplasty is to reduce excess skin and fat and sculpt the eyelid by redefining the youthful lid crease.

It is extremely important to have a firm understanding of the anatomy of the periorbital area as well as the basic tenets of blepharoplasty surgery. Due to the fact that people only have two eyes and vision is a most precious sense, utmost care must be exercised in cosmetic blepharoplasty which is an elective procedure. The goal of cosmetic blepharoplasty is to reduce the excess skin, muscle, and fat, and to produce a sculpted eyelid complex that replicates that of youth. This is a surgical procedure where it is mandatory to be conservative as it is not hard to produce irreversible changes that can lead to corneal damage, visual problems, and, in extreme cases, blindness.

Evaluation of the blepharoplasty patient

As we age, the eyelid skin becomes redundant, crepe like, and sun damaged. These skin changes of the eyelid skin are known as dermatochalasis. In addition,



Fig. 53.31 This patient underwent placement of medium combined submalar shell silicone cheek implants.



Fig. 53.32 This patient underwent placement of medium submalar silicone cheek implants with a mini-facelift.

tion, the forehead and brow complex undergo ptotic changes which contribute to the lateral eyelid ptosis known as hooding. Finally, the periorbital fat undergoes a pseudoherniation and pushes against the weakened orbital septum causing noticeable fat bags (Fig. 53.33).

First the brow position is evaluated. If a patient has significant brow ptosis, a brow and forehead lift must be considered and offered to the patient as a surgical option. The normal female brow is above the height of the superior orbital rim and the normal brow position in males is at the superior orbital rim. If the brows are low and the patient has significant lateral hooding, a brow and forehead lift is the proper operation, assuming the patient desires elevated brow. This not only elevates the brow and forehead, but opens up the look of the eyelids and redrapes the excess skin and fat, sometimes without any additional eyelid surgery. If the eyelid changes are severe, a brow and forehead lift and upper lid blepharoplasty may be necessary. Younger patients can often have their upper lids improved with brow and forehead lift only. If a ptotic brow and forehead go undiagnosed and the ptotic eyelid soft tissues are addressed, it can actually accentuate the problem and cause further lowering of the brows. The advice here is that all blepharoplasty patients must be evaluated for the need for brow and forehead lift and given the option. If they are candidates for the brow lift but refuse the procedure it must be well documented that they were given the option.

Cosmetic blepharoplasty is a procedure where the importance lies not in what is taken away, but rather what is left behind. If too much fat is removed, the patient can end up with a skeletonized appearance and if too much skin and muscle are removed, the patient may be unable to close the eyelids, which can lead to corneal damage and other significant functional problems. Needless to say, a patient that presents for an elective cosmetic procedure should not end up with serious cosmetic or functional problems.



Fig. 53.33 A patient with pseudoherniation of the periorbital fat pads, producing an aged and tired appearance.

Potential eyelid surgery patients must be evaluated closely as in some cases their vision can be compromised. If any patient is known to have ophthalmologic problems, they should receive a complete eye exam by their ophthalmologist with a written report back to the surgeon clearing the patient for surgery. Diabetes, thyroid conditions, uncontrolled hypertension, and other disease processes can contribute to blepharoplasty problems. All patients must also have a vision test with a Snellen chart before their procedure.

Tear production

It is paramount that patients with dry eyes be closely evaluated and conservatively treated. Should lagophthalmos (inability to close the lids completely) occur after surgery in a patient with decreased tear production, severe corneal damage could occur. When in doubt, a Schirmer test should be performed.

Potential bleeding problems

Although some surgeries can be performed with minor bleeding, blepharoplasty is not one of them. Bleeding around or behind the globe can lead to retrobulbar hematoma which can cause retinal artery occlusion and blindness.⁹ Although a rare problem, it is a reality and can be catastrophic. Patients on any medications (the list is long) that can affect coagulation must have their medications regulated. Many aspirin-containing compounds exist and the patient's co-medications must be carefully reviewed. Also, homeopathic medications and herbs such as ginkgo, ginseng, and garlic can affect hemostasis. Coagulation studies are obtained preoperatively on all blepharoplasty patients within 2 weeks of planned surgery.

Postoperative activity is also related to bleeding. Patients can induce bleeding by overactivity in the first week after surgery. It is important to have clear postoperative instructions that stress the importance of avoiding any activity that may raise blood pressure, including coughing, bending over, lifting, vomiting, etc. Patients must remain "couch potatoes" for the first 48 hours and keep cold compresses on the eyes for that time.

Eyelid laxity

Older patients or those with lax eyelids can be very problematic for the blepharoplasty surgeon, especially with lower eyelid surgery. Ectropion and associated eyelid malposition can lead to esthetic and functional problems in patients with lid laxity.

The pull test is a maneuver where the lower lid is pinched in the midline by the examiner and pulled away from the eye. A normal lower lid should stretch less than 8mm. A lower lid that pulls away from the globe 10mm or more can indicate potential problems and may require canthopexy for tightening

(Fig. 53.34). The snap test involves pulling down the lower lid with the examiner's index finger and the lid should snap back to normal position (just above the lower pupillary limbus) within 1 second. A lid that does not return to normal or one that takes longer than 1 second may indicate problematic lower lid laxity (Fig. 53.35). Novice blepharoplasty surgeons should avoid operating on patients with loose eyelids due to the potential complications.

Corneal examination

It is of utmost importance to protect the cornea at all costs during blepharoplasty procedures. Preoperative examination includes testing the corneal reflex. When the cornea is touched with a cotton-tipped applicator a blink reflex should be initiated. The eye should also be evaluated for Bell's phenomenon. When the patient closes their eyes and the examiner pries open the lower eyelid, one should see sclera, not pupil. This is a protective reflex in which the globe rolls superiorly when the eyes are shut to protect the cornea. With positive Bell's phenomenon one sees white (sclera) when the closed lid is pried open. If the patient exhibits a negative Bell's phenomenon, they are in danger of corneal damage if lagophthalmos occurs.

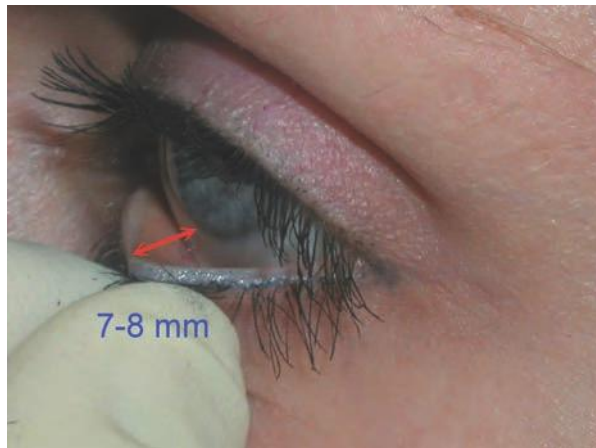


Fig. 53.34 The pull test measures lower lid laxity. The lower eyelid should not pull more than 7–8 mm from the globe in a normal lid.



Fig. 53.35 The snap test is another means of evaluating lower lid laxity. When pulling the lower lid inferiorly with the finger (left image), the lid should return to normal position within 1 second (right image). Failure to return to normal position or taking longer than 1 second can indicate lower eyelid laxity.

Photographic documentation

A complete preoperative photographic series is made on all blepharoplasty patients. Most patients pay little attention to their eyes until they have surgery, then they scrutinize them continually. A frontal image, bilateral oblique images, an image with the eyes closed, and an image with the patient looking superior form the minimum preoperative series. If the patient has asymmetries or other problems, pointing them out and discussing them preoperatively can save much discourse in the postoperative period. Also it is not uncommon for a patient to blame the surgeon for some problem that they actually had preoperatively, and showing the preoperative images can calm down an anxious patient.

Blepharoplasty anesthesia

Although many ophthalmologists prefer to perform blepharoplasty with an awake patient and local anesthesia, the author prefers to use intravenous sedation. It is difficult enough to operate on the eyes and having a fidgeting patient can make for dangerous surgery. Advocates of local anesthesia cite the fact that they can gauge skin removal and other indicators by asking the patient to open or close the eyes.

Blepharoplasty procedure

Many surgeons agree that learning how to properly mark the patient is actually harder than the actual surgery. As was stated previously, blepharoplasty surgery is about what is left rather than what is taken away, and this underlines the importance of correct preoperative marking.

Upper eyelid marking

The biggest challenge for novice blepharoplasty surgeons is learning how to mark eyelids properly. Once one understands the marking procedure, the surgery is relatively straightforward. If one understands correct marking technique, one understands blepharoplasty. There is no cookbook technique for marking the upper eyelid because each lid (even on the same patient) is different. It is imperative to mark the eyelids with the patient relaxed in the upright position. When the patient is lying down, the brows and lids are not in a natural position and the markings can be inaccurate. It is important to remember to take preoperative pictures before marking the patient. The lids are wiped with alcohol or acetone before marking so the ink adheres better, which preserves the markings through surgical scrub and manipulation.

The first task is to decide where to locate the upper lid crease. Most men (non-Asian) have an upper lid crease 8 mm above the lashes, and most women (non-Asian) have an upper lid crease 10–12 mm above the lashes. Generally the lid crease marking is drawn on

the patient's existing lid crease. Too high a crease in a man results in a feminine appearance, and too low a crease in a woman results in a masculine appearance. Women desire a high lid crease to have a significant lid shelf on which to apply eye shadow. The position of the upper lid crease can be discussed preoperatively; the author prefers 11 mm for women and 8 mm for men. The crease is marked by having an assistant elevate the brow to a normal position (at the orbital rim for men, above the orbital rim for women) and asking the patient to open and close the eyelid to visualize the crease. The crease is then marked from the lateral canthus to the lacrimal punctum with a fine tip surgical marker (Fig. 53.36). Generally the center of the crease is at the 8–10 mm mark, and the ends of the crease taper to 4–5 mm high, which creates an arc.



Fig. 53.36 The patient's natural eyelid crease is marked with a fine tip surgical marker. Most females have natural creases at 10–12 mm superior to the upper lid margin.

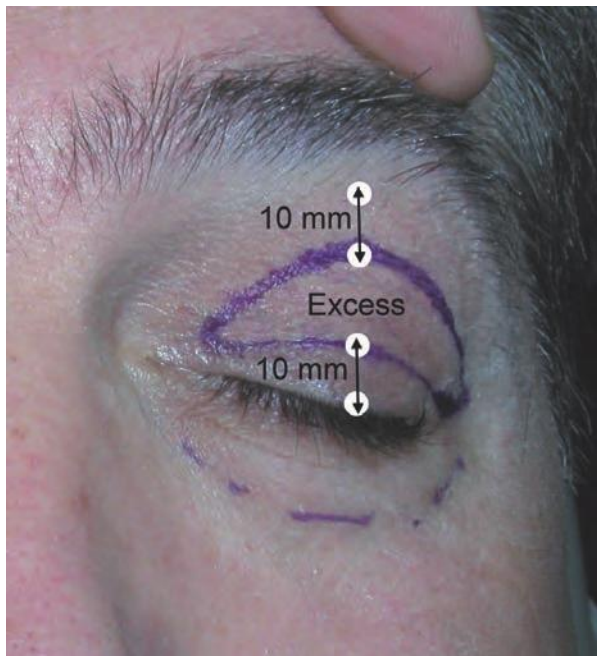


Fig. 53.37 The upper lid incision line is marked 10 mm inferior to the change in skin consistency from forehead skin to orbital skin. The lower incision line is marked 10 mm above the inferior lid margin. The skin remaining between these markings can usually be safely removed without compromising eyelid closure as long as 20 mm of lid skin is left intact.

The upper portion of the upper eyelid marking is made approximately 10 mm from the junction of the forehead and eyelid skin. If one closely examines the skin inferior to the eyebrow, one sees that there is an area (generally corresponding to the bony orbit) in which the smooth, thicker forehead skin meets the thinner crinkly upper eyelid skin. This is usually just below the finest hairs of the eyebrow. A mark is made 10 mm below this junction to define the upper extent of the skin excision (Fig. 53.37). If the surgeon leaves 10 mm from the lash to the lid crease and 10 mm from the forehead/eyelid skin junction, this gives a total of 20 mm of upper eyelid skin preserved to enable lid closure. This is the most critical point of successful upper eyelid blepharoplasty. Failure to do so can result in overexcision of upper eyelid skin and permanent lagophthalmos. One should leave at least 20 mm of upper eyelid skin intact.

The final step is to connect the incision at the lateral canthal area. If simultaneous brow lift is planned, lateral hooding of the upper lid is corrected and a “Napoleon’s hat” incision is used for the lid skin (Fig. 53.38). Because the brow lift elevates the lateral lid skin, no lateral lid skin must be excised. If a brow lift is not planned, then a “bird beak” incision is used with a lateral extension of extra lid skin excised to correct hooding (Fig. 53.39). These nicknames are used by the author to teach and are not official nomenclature in the ophthalmologic literature.

After the lid markings are made, a “pinch test” is made to check that the lids still can be closed with the prospective skin excised. When pinched, the lashes should just barely elevate, but the eyes still should be able to remain closed (Fig. 53.40). This test should be performed while elevating the brow to its normal position. This is especially critical when simultaneous brow lift is planned. Fig. 53.37 shows a summary of the planned incisions that are designed to leave a total of 20 mm of intact upper lid skin to ensure lid closure.



Fig. 53.38 When a simultaneous brow lift is performed, no significant lateral extension needs to be performed as the brow lift will elevate the lateral orbital skin and improve hooding. In this case, the “Napoleon’s hat” type of marking is made.

When addressing the medial junction of the upper and lower marks, it is important to avoid the multi-contoured depression lateral to the nose (Fig. 53.41). Generally the medial corner of this incision ends at the lacrimal punctum. If the excision is performed laterally on to the nose, a scar will be apparent and scar webbing of this multicontoured area can occur. Again, one must remember to elevate the eyebrow to a normal position before making the incision markings.

Upper lid incision

The author wears three-power surgical loupes and a headlight for all blepharoplasty procedures. Hemostasis is the key to safe blepharoplasty. When fat pads are recontoured, it is not unusual for a lacerated blood vessel to retract deep into the orbit and it can be challenging to cauterize it. If this occurs with a cut, bleeding vessel, it could cause a retrobulbar hematoma. Sometimes these vessels are small, and wearing loupes is a significant advantage. Many



Fig. 53.39 When no brow lift is planned, the lateral portion of the excision is usually angled up on the inferior margin and produces an angular geometric incision marking on the lateral region in order to remove extra skin to improve hooding. In this case the “bird beak” incision marking is made.



Fig. 53.40 After all markings are made, the incision margins are pinched together and the upper lash should just begin to elevate.

other small structures must be avoided, and the use of surgical magnification and concentrated headlight instruments cannot be overstated.

Incisional modalities for the upper eyelid skin include scalpel, scissors, radiofrequency microneedle tip, and laser. The author routinely uses either CO₂ laser or radiofrequency for upper blepharoplasty. Fig. 53.42 shows a picture of the 0.2mm laser handpiece for the Lumenis Encore CO₂ laser and the Ellman radiofrequency system with the Ellman Empire Micro Incision Needle. These modalities, especially the CO₂ laser, produce minimal bleeding, which translates into fewer complications and faster recovery. The author has conducted research using a CO₂ laser cutting handpiece with a 0.2mm spot size on one upper eyelid and the Ellman radiofrequency microneedle on the other upper lid. Basically, these instruments perform similarly and produce scars that cannot be differentiated in a statistically significant manner by blinded observers.¹⁰ The CO₂ laser is more efficient at hemostasis but is less effective on a large bleeding vessel. It is rare for the author to cause a cotton swab-sized amount of blood loss in routine blepharoplasty (Fig. 53.43).

Once the upper lid has been marked, prepared, and anesthetized, a full-thickness skin ellipse is excised. This skin is thin, and one must ensure that only the skin is dissected, similar to peeling a grape skin. This plane may be “predissected” by spreading a tenotomy scissors between the skin and muscle plane. Once the skin ellipse is removed, the orbicularis oculi muscle is visualized. This muscle is vascular and frequently has a superficial network of large vessels on its surface. The next step is to excise and sculpt the orbicularis muscle. Some surgeons advocate removing the same amount of orbicularis as skin. The author has found that being more conservative in muscle resection results in a better sculpted lid sulcus, faster return of eyelid function, and less bruising. Four to five millimeters of orbicularis oculi muscle



Fig. 53.41 It is important to not extend the upper incision on to the multicontoured areas (circled) of the medial orbital/nasal region or an unsightly and difficult to correct webbed scar can occur.



Fig. 53.42 The 0.2mm CO₂ laser handpiece and the radiowave surgery system are pictured.



Fig. 53.43 This author utilizes radiofrequency microneedle or CO₂ laser for blepharoplasty incision. This image shows the bloodless excision of the upper lid skin with either modality.



Fig. 53.44 The orbicularis oculi muscle in the upper eyelid being resected with laser and radiofrequency modalities.

are excised using either the CO₂ laser or the Ellman radiofrequency microneedle (Fig. 53.44).

If the orbicularis oculi muscle is dissected in the proper plane, the orbital septum is visible. It is frequently translucent, and one can see the fat pads moving under the septum during light pressure on the globe. The septum can be incised with a small rent over the central and medial fat pads or incised totally from medial to lateral. For the novice surgeon, the orbital septum may not be apparent, and there is risk

of incising through the levator aponeurosis (and deeper structures) if the incision is placed too inferiorly. When in doubt as to where the upper lid fat pads are located, one should err in the direction of the superior orbital rim. These fat pads cushion the globe between the bony orbit and anatomically are close to the orbital rim. Gentle pressure on the globe (retropulsion) accentuates the prolapsed fat pads and makes them more visible. The septum is incised with the CO₂ laser or Ellman radiofrequency microneedle

in the center of the orbit just below the orbital rim. The central fat pad generally prolapses and can be dissected to its lateral extent, which sometimes may extend to the lateral portion of the incision. The lacrimal gland lies in the superior lateral portion of the orbit and has been mistaken for fat pads. There are usually only two fat pads in the upper eyelid: a central and a medial. The lacrimal glands are pinker (similar to parotid tissue) and firmer than the yellow central fat pad. If the lacrimal glands are slightly prolapsed, they can be tucked back into the orbit. If they are significantly prolapsed, they are suspended to the orbital periosteum with a non-resorbable suture.

Many surgical techniques exist to reduce or recontour the orbital fat pads. Whatever technique is used, the surgeon must pay close attention not to pull, stretch, or retract the fat pads too much. Many vessels traverse the fat pads from deep within the orbit. If the fat pads are overstretched from the orbit, one could lacerate a deep vessel. When the fat pad is released it can retract deep into the orbit along with the bleeding vessel. If uncontrolled bleeding occurs deep in the orbit, a hematoma can form and compress the retinal artery and cause permanent blindness. Extreme caution should be used when handling the fat pads. Generally, one should excise only the amount of fat that easily protrudes with gentle pressure on the globe. The surgeon or the assistant never should “tug” on the fat pads. Sometimes a surgeon views the fat pads as the “enemy” and removes as much as he or she can find. Blepharoplasty is an operation of what tissue is left behind, not what tissue is removed. Excessive fat resection causes a hollow-looking orbit, which gives the patient an older, gaunt appearance. One should be conservative. A good general rule is to take only the amount of fat that the orbit “gives up” and not be overly aggressive in searching for or removing deep fat. Some surgeons prefer “melting” the fat pads with laser or radiofrequency instead of resecting or recontouring portions of the fat pad. Fat pad removal has been described as teasing out the protruding fat then clamping the base of the fat pad with a hemostat before resecting. Although it makes sense to clamp and crimp the base of the fat pad to assist in hemostasis, the hemostat also can exert a lever effect on the fat pad and inadvertently stretch the base of the fat pad and lacerate a vessel. This can

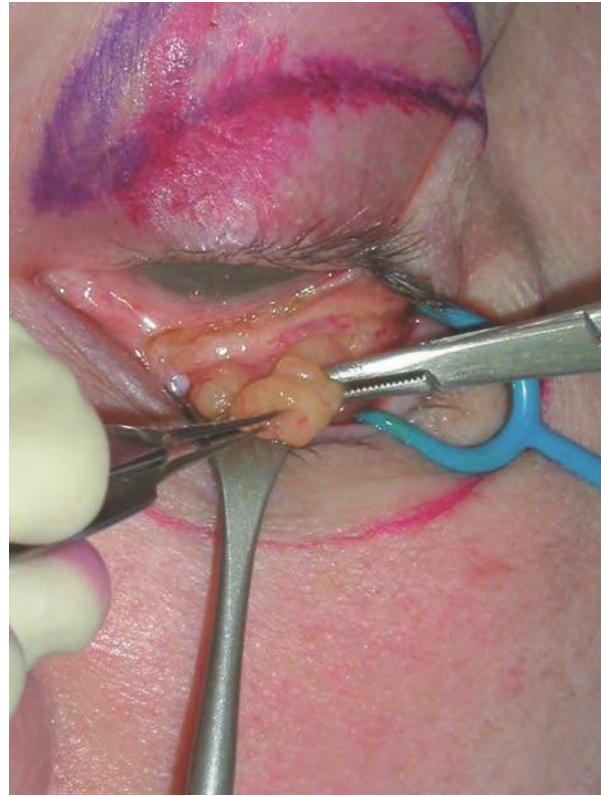


Fig. 53.46 Clamping the base of the fat pad with a small hemostat can assist hemostasis before sectioning the base of the fat.

happen when the assistant holds the hemostat while the surgeon incises the fat pad. When the CO₂ laser or the Ellman microneedle tip electrode is used, excellent hemostasis ensues and the clamp is not required (Fig. 53.45). If a surgeon wishes to excise fat with a scalpel or scissors, the use of a clamp is recommended before cutting to ensure hemostasis. Cauterizing the base of the fat pad above the hemostat also is recommended before release (Fig. 53.46).

Locating the medial fat pad in the upper eyelid can be a challenge for the novice surgeon. It usually lies more medially than the medial incision. The best means of locating the medial fat pad is to spread a small hemostat gently in the inferior medial portion of the medial blepharoplasty incision while gently pressing on the globe. This should be done delicately, with small spreading motions. Once the medial fat pad is located and teased out, it frequently balloons to surprising proportions. The medial fat pad contains a larger amount of fibrous tissue than the other

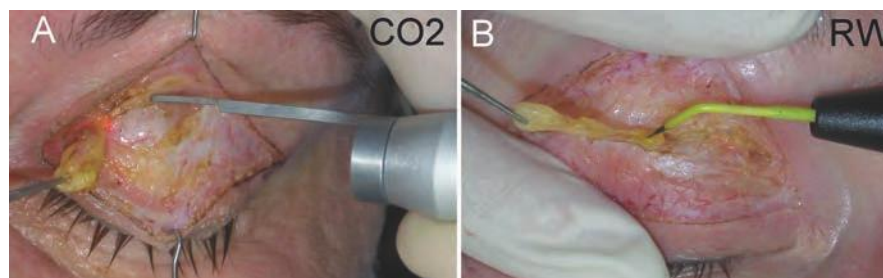


Fig. 53.45 The CO₂ laser and radiowave microneedle produce a safe and bloodless modality for fat excision in upper lid blepharoplasty.

fat pads and is usually a whiter color when compared with the butter yellow color of the other fat pads. As fat is removed from each eye, it is placed on a gauze or towel so that approximately similar volumes are removed from both eyes.

Once the medial and central fat pads are recontoured, the entire surgical site is reinspected for bleeding. Placing several drops of saline or local anesthesia in the incision site can help identify small or persistent bleeders. Once the surgeon is sure that the surgical field is dry, the incision is closed with either 6/0 nylon or 6/0 fast-absorbing gut suture in an interrupted or running fashion. If a lateral extension is used on the blepharoplasty incision, several interrupted sutures are placed to reinforce the tissue in the crow's feet areas. An interrupted or running 6/0 nylon suture is used for the remaining closures (Fig. 53.47). When performing simultaneous upper and lower blepharoplasty, the lower lids are usually done first so as not to disturb the upper incisions with retraction suture. The surgical site is then covered with triple antibiotic ointment and iced saline gauze. If nylon sutures are used, they are removed at 5 days. It is much easier to remove a single running suture than 10–12 interrupted sutures.

Lower eyelid transconjunctival technique

The transconjunctival approach for lower eyelid fat removal does not violate the orbital septum and produces less lower lid retraction. Some surgeons use the transconjunctival approach and perform a preseptal dissection, which requires septal incision and negates the septal sparing component of this approach. The surgical approach described is retroseptal. Using the thumb and index finger, the assistant retracts the lower eyelid. The surgeon gently depresses the globe with a Jaeger lid plate retractor, which prolapses the herniated fat. Technically the incision should be made approximately 4 mm inferior to the lower tarsal margin or approximately 8 mm from the lower lid margin.



Fig. 53.47 Closure of upper blepharoplasty incision with several spaced interrupted sutures followed by a running 6/0 fast-absorbing gut suture. All sutures are removed at 5 days.

(Because the lower tarsus is approximately 4 mm wide, placing the incision 4 mm below this equals 8 mm.) The incision is made directly through the middle of the conjunctival bulge that forms from the assistant's retraction and the surgeon's pressure from the Jaeger lid plate. This usually corresponds to 4 mm below the inferior tarsal margin. If in doubt, one should err in the direction of the orbital rim and not the globe. A transconjunctival incision too deep in the fornix can damage the extraocular muscles and initiate unnecessary bleeding. The incision is made from the lacrimal punctum medially to the lateral canthus laterally. Failure to carry the incision far enough laterally causes the surgeon to operate in a hole and makes surgery more difficult, especially access to the lateral fat pad. To allow access to the lateral canthal conjunctiva, the assistant must move the thumb and index finger more laterally to pull and spread the lateral canthus. Doing this provides access to the conjunctiva in the lateral corner of the eye. The CO₂ laser or Ellman microneedle is used to incise through conjunctiva and capsulopalpebral fascia (lower lid retractors) in gentle, sweeping motions (Fig. 53.48).

At this point, a retraction suture is placed. A 5/0 gut suture on a small cutting needle is passed through the superior incision margin and draped over the head and suspended with the weight of a hemostat (Fig. 53.49). This technique retracts the incision for facilitated view and instrumentation. It also can serve to protect the globe.

After each level of incision, the fine hemostat is inserted and right-to-left spreading is done gently (as opposed to anterior–posterior spreading, which could damage the eyeball). One must pay attention to staying anterior to the globe when incising and dissecting. In some patients these layers are thick and well defined, and the surgeon can incise directly to the fat pads. In other patients the anatomy is less defined, and accessing the fat is a matter of gentle incision and gentle spreading with the small hemostats. As this gentle dissection is performed, gentle pressure is



Fig. 53.48 Incision placement of the transconjunctival approach from the surgeon's view. The incision is made from the punctum to the lateral canthus about 4 mm below the lower tarsal margin or 8 mm inferior to the ciliary margin.

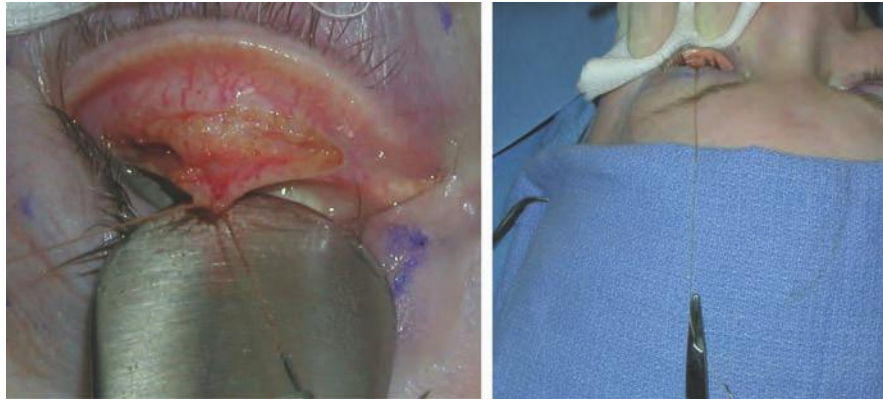


Fig. 53.49 A retraction suture is placed through the superior incision flap, secured with a hemostat and hung over the head to retract the incision.

placed on the globe and the fat pads come into view. If the surgeon becomes disoriented or unsure of his or her position within the orbit, he or she should use an instrument to sound the inferior orbital rim. This ensures that the surgeon's position is anterior to the globe. As the fat pads come into view, their capsule is incised by laser or radiofrequency, which usually allows the herniated fat to billow out of the incision (Fig. 53.50). The fat is teased out and never stretched or pulled with significant traction as a vessel may be torn or lacerated.

The lower lid fat is incised with laser or radiofrequency electrode (Fig. 53.51). The central fat pad is usually the first encountered and the easiest to reduce. The medial fat pad, as in the upper lid, frequently takes some blunt dissection to locate, but once incised it gives up an impressive amount of fat. This fat is more fibrous and a whiter color. The lateral fat pad in the lower lid may be difficult to deliver into the surgical field, and some exploration may be necessary. It is usually located more laterally than the lateral extent of the transconjunctival incision. The surgeon should look continually at the external lid skin while pressing on the globe to verify adequate fat removal. The inferior oblique muscle divides the central and medial fat pads, and this muscle is sometimes encountered in

lower transconjunctival blepharoplasty (Fig. 53.52). The muscle is best avoided, with care taken not to incise it. It is not a problem to retract the muscle gently, but damage could result in extraocular muscle dysfunction.

After the three lower fat pads have been recontoured, the surgical site is checked for hemostasis.

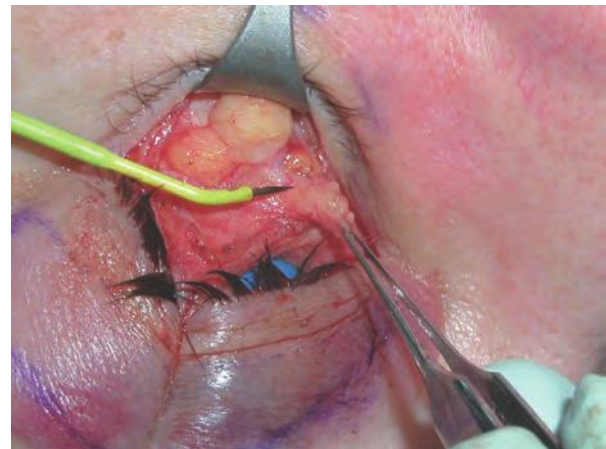


Fig. 53.51 A radiowave microneedle electrode being used to cauterize the base of the medial fat pad. The excess fat is excised slowly and conservatively in a bloodless manner with this modality or with CO₂ laser incision.



Fig. 53.50 The medial, central, and lateral lower fat pads teased out of their respective positions into the transconjunctival operative field.



Fig. 53.52 The inferior oblique muscle divides the medial fat from the central fat compartment and is frequently encountered in the transconjunctival approach.

Generally the incision is not closed unless it is gaping. Usually by picking up the incision margins with small forceps and approximating them, they stay together well and rarely, if ever, require suturing (Fig. 53.53).

It should be noted that some contemporary surgeons do not advocate any fat removal and they instead reposition the bulging fat over the inferior orbital rim. This is done to reduce the fat bags without creating hollows. The author has little experience with fat repositioning and prefers conservative fat removal.

The next step is to address the dermatochalasis of the lower eyelid. The author's preference is CO₂ laser resurfacing of the periorbital areas. The Lumenis (formerly Coherent) ultrapulsed Encore laser (Santa Clara, CA) with a computer pattern generator is used. For periorbital resurfacing, a setting of 80 MJ, 600 Hz, and a number 1 pattern (circle) size 6–7 and a density of 6 (30% overlap) is selected. A full pass is made over both eyelids and crow's feet areas, if required (Fig. 53.54). A second pass is then made on the lower lids only.

It is hard to conceal laser redness outside the confines of the orbital rims. Inside the confines of the orbital rims, the treated and untreated junction is not as obvious and can be covered with sunglasses. If a patient wants resurfacing of the crow's feet and infra-orbital areas, the author frequently converts this to a full-face resurfacing procedure to better blend the laser areas and not end up with relative hypopigmentation of the treated skin when compared with the surrounding skin. After the first pass, the char is debrided with moist gauze followed by dry gauze. A second pass is made on the lower lid at the same or lower setting. Frequently only a single pass is used on the upper lids to blend the color. This is especially true if blepharoplasty and brow lift are performed together. Adding aggressive laser resurfacing with other procedures can cause lagophthalmos. If severe wrinkling exists, two passes are used on the upper lids. It is important to understand that there is no



Fig. 53.53 The transconjunctival incision is passively approximated at the end of the procedure and does not require suture closure.

definitive laser setting and that each laser and each patient involve separate situations and must be treated individually. The second pass is then debrided with the moist and dry gauze and a petrolatum dressing is applied. The laser shields are removed, and the eyes are copiously irrigated with basic saline solution and lubricated with bacitracin ophthalmologic ointment.

When performing blepharoplasty on four lids simultaneously (or sometimes two lids), a patient may have several millimeters of lagophthalmos (inability to close the eyelids completely). This is not unusual and results from swelling, local anesthesia, local hemorrhage, and tissue traction. It usually resolves in several days or a week.

Skin pinch technique

If the laser is not used, the skin pinch technique is indicated. The author tries to avoid using a skin-muscle approach to preserve the orbital septum and prevent lower lid retraction.

The skin pinch technique is a convenient, minimally invasive procedure that deals with excess skin in the lower eyelid.¹¹ It also complements the transconjunctival approach for individuals who cannot tolerate post laser erythema. The author uses this technique mostly in men or patients with pigmented skin for whom the laser is contraindicated. Women can cover laser redness with makeup, but because erythema can persist for months, it is prohibitive in many men. African-American, Asian, Latino, or other individuals with pigmented skin tend to have post-laser pigmentation problems, and the skin pinch technique is a favorable adjunct for that population. The procedure is simple and involves crimping the excess skin with a hemostat and resecting the skin.

If intravenous sedation is used, no local anesthesia is required with this technique. In a non-sedated patient, local anesthesia infiltration is required, but it distorts the skin and makes the judgment of how much skin to remove more difficult.



Fig. 53.54 The excess periorbital skin is treated with the CO₂ laser.

The skin pinch is performed last, after completing the transconjunctival removal of herniated periorbital fat. The first step is to take cotton pliers and grasp the excess lower eyelid skin just below the lashes (Fig. 53.55). Generally, most patients require approximately 5 mm of skin to be removed (Fig. 53.56). By “test pinching” with the cotton pliers, the surgeon can judge how much skin to remove. The skin is pinched just below the lash so that the lower lid skin is tightened while the lower lashes are gently everted without retracting the lower eyelid. When satisfied with the correct amount of skin (one should be conservative), the pinching begins in the lateral one third of the lower lid.

Using a small hemostat, the surgeon pinches 4–5 mm of excess skin and crushes it in the serrated beaks of the hemostat at the nasal, central, and lateral regions (Fig. 53.57). The surgeon must grab and crush the same piece of skin three to four times. Doing this elevates a ridge of excess skin that is crushed at its base. The next step is to use a Wescott or other small scissors to sever the crushed skin ridge at its base (Fig. 53.58). Little bleeding occurs due to the fact that the base of the incision is crimped. The surgeon needs to be careful not to cut the eyelashes when trimming

the skin. After the scissor excision the crushed skin stays approximated as shown in Fig. 53.59.

The incision can be pulled apart, which exposes the orbicularis oculi muscle in the lower eyelid. If the muscle is hypertrophic, the surgeon can trim the excess or shrink it with the CO₂ laser or Ellman radio-frequency small ball electrode (Fig. 53.60).

Because the skin has been crushed and cut at several points, a jagged incision may result. The Ellman



Fig. 53.55 The excess skin of the lower lid is pinched with a forceps to estimate the amount to be resected.



Fig. 53.56 Skin excision of the pinch procedure should be very conservative so as not to cause lower lid malposition. Generally the amount of skin excised is 3–6 mm.



Fig. 53.57 Curved hemostats are used to crush the lower eyelid skin at the nasal, central, and medial regions.



Fig. 53.58 The crushed skin is then excised with microscissors at the base.



Fig. 53.59 The excised skin and the resultant wound.

Empire radiofrequency microneedle can be used to even out the incision to a semilunar shape. The final step is to close the incision with five to six 6/0 fast-absorbing gut sutures (Fig. 53.61).

Skin-muscle approach for lower blepharoplasty

Although this approach to the lower eyelid is very popular with some surgeons, the author tries to avoid it because of the increased incidence of lower lid malposition. There are times when a patient has hypertrophic orbicularis oculi muscle bulges in the lower lid and this technique is required.

The periosteum of the lateral orbital rim is contiguous with the orbital septum. Local anesthesia is injected at the lateral orbital rim, the needle is angled parallel to the lower lid margin and the plane between the septum and orbicularis muscle is ballooned with local anesthesia. An incision is made at the lateral canthal area and fine scissors are used to dissect bluntly to the periosteum over the lateral orbital rim. Once this plane is reached, the scissors are turned sideways and the ballooned plane between the septum and the muscle is dissected easily by spreading

the scissors from the lateral lid to the medial lid (Fig. 53.62).

The next step involves angling the scissors parallel to the lower lid and making a cut through the skin and muscle (Fig. 53.63). The scissors are repositioned parallel to the skin, and the skin and muscle are cut on an angle, with the outside limb of the scissors cutting the skin surface and the inside limb of the scissors cutting the muscle surface. The muscle is cut at a higher level than the skin, which preserves the pretarsal muscle structure and decreases the chance of lower lid ectropion.

When the subciliary incision is completed, the surgeon has an excellent open view of the orbital septum (Fig. 53.64). The septum is incised by the preferred modality (Fig. 53.65), and the three lower fat pads are identified and recontoured (Fig. 53.66). The final and trickiest step is trimming the excess skin and muscle from the lower incision margin. This is described in many ways by various authors and is often contradictory. The author has found the following technique to work effectively without causing lid retraction. The excess skin is grasped and pulled superiorly and slightly laterally at the lateral orbital rim area. There is more superior pull than lateral. This approach pro-



Fig. 53.60 The orbicularis oculi muscle can be contracted by using the radiowave ball electrode or the CO₂ laser. This can provide further tightening for the procedure.



Fig. 53.61 The skin pinch incision is closed with 6/0 fast-absorbing gut suture.



Fig. 53.62 The scissors are inserted above the septum and below the orbicularis muscle and the plane is easily dissected by spreading and advancing the scissors.



Fig. 53.63 The skin incision is made with scissors in a curvilinear line in the subciliary region.

duces a triangle of excess lower lid skin and orbicularis muscle. The triangle is pulled with minimum traction to prevent over resection, and the base of the triangle is trimmed (Fig. 53.67). The skin is then closed with 6/0 nylon or 6/0 fast-absorbing gut sutures. The septum is never sutured.

Postoperative care

Postoperative care is centered on keeping activity and blood pressure down. Catastrophic complications, including blindness, can occur from retrobulbar hematomas causing retinal artery occlusion. Iced saline gauze (or commercially available cold packs or gel masks) are immediately applied and left on for 24–48 hours. The author makes all patients promise to be a “couch potato” for 48 hours and sleep with their head elevated. It is imperative that they do not perform Valsalva maneuvers or otherwise increase blood pressure to the head. Retrobulbar bleeds have occurred from coughing, straining with bowel movements, bending over, sexual intercourse, and other exertion in the immediate postoperative period. Patients are instructed not to lift anything over 5 kg for 1 week. It is also important to ensure that patients refrain from any medication that may prolong bleeding. Postoperative care includes antibiotics, analgesics, tapering steroids, and ophthalmic drops (Tobradex or Oculoflox) on occasion. Patients can expect swelling and sometimes bruising. Pain is gen-

erally minimal unless corneal abrasion has occurred from eye shields. Patients should be able to see light and count fingers. Severe pain, throbbing retrobulbar pain, or frank proptosis may signal retrobulbar hematoma and should be immediately assessed because it indicates a true emergency.^{12,13} If all four lids are operated or if endoscopic brow lift or periorbital laser is performed, the eyelids may swell shut for 24 to 48 hours. Patients should receive preoperative instruction regarding that possibility.

Some patients cannot close their eyes completely after blepharoplasty; this condition usually resolves



Fig. 53.66 The exposed fat pads are conservatively excised and recontoured, in this case with the Ellman radiowave microneedle electrode.



Fig. 53.64 The orbital septum and underlying fat are visualized after the suborbicularis dissection is performed.



Fig. 53.67 The excess skin is conservatively trimmed. The novice blepharoplasty surgeon should always excise skin conservatively, so as not to cause lower lid malposition.



Fig. 53.65 The orbital septum is incised and the fat pads are accessed under direct vision.



Fig. 53.68 A patient with subconjunctival ecchymosis. The patient did not stop aspirin preoperatively as directed. The situation spontaneously resolved in 10 days.

spontaneously in several days to several weeks. One problem is dry eyes caused by lagophthalmos, which can be painful and irritating and, in severe cases, may cause corneal problems. The author uses Refresh eye drops (Allergan, Irvine, CA) for these patients during the day and Refresh Plus eye drops at night. Generally they adequately hydrate the eye until the lagophthalmos resolves. If dryness is severe, especially at night, patients can cut a piece of plastic wrap the size of their palm and cover their eye at night. Applying

petroleum jelly or lotion to the periorbital area keeps the plastic wrap in place.

Another common complication is subconjunctival ecchymosis (Fig. 53.68). This harmless complication is a result of blood under the bulbar conjunctiva. Patients should be reassured that this condition will resolve, although sometimes it can last several weeks.

Chemosis is edema of the bulbar conjunctiva and may be caused by inflammation or overactivity. The patient in Fig. 53.69 felt so good after her blepharoplasty that she went home and bent over to pull weeds, which caused severe chemosis. She was treated with prednisone, 60 mg once a day, for 5 days and warm compresses. The chemosis resolved within a week.

Periorbital ecchymosis can occur for no apparent reason, but may signal a coagulation problem. The patient in Fig. 53.70 had normal coagulation studies but a history of bruising. Periorbital ecchymosis resolves spontaneously.

Postblepharoplasty wound dehiscence can occur, and unlike other surgical wounds it usually heals without any significant scarring. If the wound separates in the first 48 hours, it can be cleansed and resutured. Late wound dehiscence is treated by wound care with irrigation and topical antibiotic ointment. Fig. 53.71 shows an early wound dehiscence that was cleansed and resutured. Fig. 53.72 shows a late wound dehiscence. At 3 months, the scar



Fig. 53.69 Chemosis is conjunctival edema that can occur from postoperative overactivity and responds to rest, heat, and corticosteroid treatment. This patient is shown preoperatively, 5 days postoperatively with severe chemosis, and 3 weeks postoperatively after rest, heat, and prednisone treatment.



Fig. 53.70 Periorbital ecchymosis can be caused from coagulation problems. Some patients are prone to such bruising despite normal coagulation profiles. Aspirin or other medications that affect platelet function can also contribute.



Fig. 53.71 Suture line dehiscence that occurs in the first several days of healing can be cleansed and reclosed.



Fig. 53.72 This late-occurring suture line dehiscence (12 days) healed by secondary intention without significant scarring.



Fig. 53.73 This male patient is shown 8 weeks after bilateral upper blepharoplasty, bilateral lower transconjunctival blepharoplasty, and periorbital CO₂ laser skin resurfacing. The significant improvement in skin wrinkling is a direct result of the laser treatment.



Fig. 53.74 This 42-year-old female was treated with bilateral radiowave assisted upper blepharoplasty.



Fig. 53.75 This 48-year-old male was treated with bilateral upper blepharoplasty, bilateral lower transconjunctival blepharoplasty, and lower eyelid skin pinch.



Fig. 53.76 This 48-year-old female was treated with bilateral upper blepharoplasty, bilateral lower transconjunctival blepharoplasty, and periorbital CO₂ laser skin resurfacing.



Fig. 53.77 This female patient was treated with bilateral laser-assisted upper blepharoplasty and bilateral lower lid transconjunctival blepharoplasty. No treatment was rendered to the lower eyelid skin.



Fig. 53.78 This patient was treated with bilateral radiowave-assisted upper blepharoplasty.



Fig. 53.79 This 63-year-old male was treated with bilateral upper blepharoplasty, bilateral lower transconjunctival blepharoplasty, and lower eyelid skin pinch.



Fig. 53.80 This female patient presented with the chief complaint of dark circles and bags of the lower lids. She was treated with bilateral laser-assisted lower lid transconjunctival blepharoplasty and lower lid CO₂ laser resurfacing.



Fig. 53.81 This 45-year-old male was treated with bilateral upper blepharoplasty, bilateral lower transconjunctival blepharoplasty, and lower eyelid skin pinch.

was undistinguishable. Should a scar persist, it can be resurfaced or excised.

Before and after images of various combinations of eyelid rejuvenation are shown in Figs 53.73–53.81.

Acknowledgment

The clinical photos in this chapter have been published previously in *Cosmetic Facial Surgery* (2010) by Dr J. Niamtu. Reproduced with permission. Copyright © 2011 Elsevier.

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Part 8: Temporomandibular Joint Disorders

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Chapter 54

Diagnosis and Non-surgical Management of Orofacial Pain

Charles McNeill and Patricia A. Rudd

This chapter will cover the classifications and descriptions of orofacial pain syndromes, both intraoral and extraoral. Appropriate imaging studies for orofacial pain syndromes and temporomandibular joint disorders will be discussed. The diagnosis of both intra-articular and extra-articular temporomandibular disorders is discussed together with non-surgical management of temporomandibular joint disorders, including behavioral therapy, physical therapy, pharmacological management, and orthopedic appliance therapy.

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Introduction

Successful orofacial pain diagnosis begins with the clinician understanding the current principles of basic pain mechanisms. In order to fulfill this important responsibility, the dentist must have a working knowledge of functional neuroanatomy, peripheral and central nervous system pain pathways, including the descending pain modulating system, and the affective or emotional aspects of persistent pain.¹⁻⁴ Pain is defined as an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage by the International Association for the Study of Pain.⁵ The definition of pain avoids relating the pain directly to the stimulus. A pain experience reported

in the absence of tissue damage is accepted as pain in the same way as pain clearly related to tissue damage.

Pain is subjective and is a personal experience that cannot be shared, involving both sensation and emotion. A stimulus is referred to as a noxious stimulus when it is one that is damaging to normal tissues. Pain responses to a given stimulus cannot be predicted with any degree of certainty. Noxious stimuli elicit a variety of behaviors that not only serve to protect uninjured tissues, but that can also cause affective or unpleasant behavioral aspects of pain. This variability is related to the activity of the pain modulation system in the central nervous system (CNS). Dysfunction of the nervous system can cause hypersensitivity and variations in pain tolerance. Pain tolerance, the

greatest level of pain which a subject is prepared to tolerate, is highly variable and depends on personality traits and situational factors. Individuals apply different meanings to pain and develop different ways to cope with pain. For example, cultural differences can lead to learned pain behavior as can other behavioral and psychological factors.

Pain can be either acute or persistent (chronic). Acute pain has a recent and usually sudden onset with short duration limited to the normal healing time. The development of acute nociceptive (normal) pain may be due to inflammation, mechanical deformation, ongoing injury, or destruction of tissue. Acute pain is usually self-limiting and does not typically present a diagnostic problem. The patient understands the cause and effect of the pain, and the clinician usually renders the appropriate intervention to treat the condition. Sometimes pain persists or even becomes worse beyond the expected time of healing. Historically the pain conditions that last longer than 3 months, regardless of the mechanism, have been referred to as chronic pain conditions or syndromes, but today the preferred term is non-malignant persistent pain. The onset of non-malignant persistent pain is less well defined than that of acute pain. Persistent pain conditions are not easily understood by the patient or the clinician and become pain without meaning.⁶

The multitude of disease entities that can present with similar pain patterns in the head, face, and neck region mandate that clinicians consider diseases unrelated to the masticatory system in their differential diagnosis of orofacial pain. A thorough diagnostic process using validated diagnostic criteria is critical, because an incorrect or omitted diagnosis is one of the most frequent causes of treatment failure.⁷ It, in fact, becomes a daunting task to correctly identify all of the possible sources of pain that may be the cause, the effect or are coincidental to a patient's orofacial pain complaints. In order to help differentiate all the possible head, face, jaw, intraoral, and neck pain conditions, it is essential to have a systematic approach. Internationally established pain classifications with operational diagnostic criteria for the various possible orofacial pain conditions serve as useful guides. Differential diagnoses can at least be based on universally accepted inclusion and exclusion criteria, even though the mechanism causing the pain may not be fully known.

Head, neck, and orofacial pain classifications

In 1988, the International Headache Society (IHS) published its landmark diagnostic system, the *Classification and Diagnostic Criteria for Headache Disorders, Cranial Neuralgias and Facial Pain*.⁸ This classification system, with specific operational diagnostic criteria, was updated and improved in 2004 and was

entitled the *International Classification of Headache Disorders II (ICHD-II)*.⁹ Two years after the publication of the second edition, the IHS launched a website edition of the International Classification of Headache Disorders (ICHD-II) (Table 54.1).¹⁰ In support of, and in conformity with this major effort, the American Academy of Orofacial Pain (known as the American Academy of Craniomandibular Disorders at the time) published a diagnostic classification for temporomandibular disorders (TMD) in 1990.¹¹ The American Academy of Orofacial Pain (AAOP) improved this classification in 1993 with the addition of more specific inclusion and exclusion diagnostic criteria.¹² In 1996 and recently in 2008, the AAOP diagnostic classification has been expanded based on the IHS classification, to include all head, face, and neck conditions that could be associated with orofacial pain.^{13,14} This classification is, as is the International Headache Society's genitor classification, a work in progress with plans to publish updated editions as new research mandates a timely transfer of science.

This chapter presents an overview of the diagnosis of orofacial pain and the specific management of temporomandibular disorders (TMD). The diagnostic range of orofacial pain includes the following three

Table 54.1 The International Classification For Headache Disorders (2nd edition: ICHD-II).

Part 1	Primary headaches
IHS 1	Migraine headache
IHS 2	Tension-type headache
IHS 3	Cluster headache and other trigeminal autonomic cephalgias
IHS 4	Other primary headaches
Part 2	Secondary headaches
IHS 5	Headache attributed to head and/or neck trauma
IHS 6	Headache attributed to cranial or cervical vascular disorder
IHS 7	Headache attributed to nonvascular intracranial disorders
IHS 8	Headache attributed to a substance or its withdrawal
IHS 9	Headache attributed to infection
IHS 10	Headache attributed to disorder of homeostasis
IHS 11	Headache attributed to extracranial pain disorders
IHS 12	Headache attributed to psychiatric disorder
Part 3	Cranial neuralgias, central and primary facial pain
IHS 13	Cranial neuralgias and central causes of facial pain
IHS 14	Other headache, cranial neuralgia, central or primary facial pain

categories: (1) medical conditions that either directly cause, refer pain to the region, or masquerade as orofacial pain; (2) intraoral pain conditions; and (3) musculoskeletal pain conditions affecting the neck and jaw (Table 54.2). Medical conditions that can be associated or confused with orofacial pain include intracranial non-vascular and vascular disorders, neurovascular pain conditions (primary and secondary headache), neurogenic (neuropathic) pain conditions, and extracranial pain disorders (e.g. ear, nose, sinus, throat conditions). Extracranial intraoral pain disorders include hard and soft tissue conditions (teeth and supporting bone), mucogingival and glossal pain, and salivary gland pain conditions. Extracranial musculoskeletal conditions involve both the neck and jaw. The jaw conditions are commonly referred to as temporomandibular disorders (TMD), and they include temporomandibular joint (articular) and masticatory muscle (non-articular) disorders.

Medical conditions masquerading as orofacial pain

Intracranial disorders

Disorders of the intracranial structures, such as neoplasia, aneurysm, abscess, hemorrhage or hematoma, and edema can usually be easily differentiated from orofacial pain.¹⁵ They should be considered first in the diagnostic process, because they can be life threatening and require immediate attention. The characteristics of serious intracranial disorders include new or abrupt onset of pain, pain that increases in severity, interruption of sleep by pain, pain precipitated by exertion or positional change, and neurologic deficits. Referred pain from primary and metastatic tumors in

particular can be extremely difficult to separate from symptoms related to temporomandibular disorders in a timely manner. The referred pain is particularly difficult to distinguish from tempomandibular disorder signs and symptoms in the earlier stages of the various disease entities.¹⁶ One vascular pain disorder, temporal arteritis, a giant cell arteritis, may be misdiagnosed as myofascial pain involving the temporalis muscle. The patient, typically at least 60 years of age, presents with a fever, anorexia, loss of weight, and severe headache. The temporal and possibly the facial arteries are swollen, torturous, and extremely tender. Arteritis is associated with a significantly elevated erythrocyte sedimentation rate, and biopsy of the temporal artery confirms the diagnosis. Treatment consists of the appropriate corticosteriod therapy for the autoimmune inflammatory process. If treatment is delayed, temporal arteritis can quickly lead to a loss of vision due to acute ischemic optic neuropathy secondary to inflammation of the ciliary artery.¹⁷

Neurovascular headache disorders

Headache is a common complaint reported by patients suffering from musculoskeletal jaw disorders. Neurovascular headache disorders and jaw disorders can share common nociceptive pathways, therefore clinicians must be aware of the characteristics of primary and secondary headache and their potential association with orofacial pain.¹⁸

Primary headache

Primary headache disorders associated with orofacial pain include migraine headaches, migraine-variant headaches, cluster headaches, chronic paroxysmal hemicrania, and tension-type headaches. Migraine headache is divided into migraine with aura (classic) and migraine without aura (common migraine) headache. Migraine with aura headaches are characterized by unilateral (60–70% of the time), throbbing or pulsating pain lasting 4–72 hours with frequent accompanying nausea and/or vomiting, phonophobia, and photophobia. They typically have a prodromal phase with visual aberrations. Migraine without aura is similar to classic migraine, but proceeds into headaches without a prodromal phase. Tension-type headaches are believed to be a type of chronic or episodic migraine-like headache. They are bilateral, mild to moderate in intensity, and characterized by a non-pulsating, pressing, or tightening feeling in the head. The headaches are not related to the use of the jaw such as chewing hard foods, and may be associated with pericranial muscle tenderness. It is important to distinguish between tension-type headaches and localized myofascial pain of the temporalis muscles. The symptoms of these conditions can be very similar, but the source of the pain can be quite different requiring very different management strategies.¹⁹ It is reported that orofacial pain patients have greater

Table 54.2 Management of orofacial pain: diagnostic range.

Medical conditions masquerading as orofacial pain
Intracranial and vascular pain disorders
Neurovascular headache disorders:
Primary headache
Secondary headache
Neuropathic pain disorders:
Proxysmal pain disorders
Continuous pain disorders
Headache attributed to associated extracranial pain disorders:
Eye, ear, nose, sinuses, and throat disorders
Extracranial intraoral pain disorders
Teeth and periodontal disorders
Mucogingival, tongue, salivary gland disorders
Musculoskeletal pain disorders
Cervical disorders
Temporomandibular disorders:
Articular disorders
Muscular disorders

prevalence of primary headaches with a greater disability impact than non-orofacial pain subjects.²⁰ Future headache therapeutic targets need to address genetics and neurophysiologic changes in the neurons and brain and also help to control immune systems including the glia that support neuronal cell function, protecting and aiding the repair and regeneration of damaged neurons.²¹

Secondary headache

Secondary headache disorders are defined as headaches that develop secondary to another condition or mechanism. These could include physical exertion, cold stimuli, trauma, infection, metabolic disorders, or substances or substance withdrawal. Orofacial pain can commonly be associated with rebound headache. Patients who have misused or abused medications, including over-the-counter analgesics and non-steroidal anti-inflammatory drugs, can suffer from rebound headache. When prescription medications are either prescribed or taken inappropriately, or when patients develop tolerances to the medications, breakthrough pain can become a major problem. There is usually a history of daily or near-daily headache and medication use, associated depression and sleep disturbance, and occasional severe migraine-like attacks. The possible mechanism of rebound headache is related to interference of the descending pain modulation system and neuroplasticity changes in the CNS by inadequate pain control from improper pharmacotherapeutic strategies.²²

Neuropathic pain disorders

Neuropathic pain is a disorder resulting from injury, peripheral and/or central, in the pain transmission system and is usually present in the absence of an ongoing primary source for the pain.²³ Neuropathic pain disorders are divided into either paroxysmal (episodic) or continuous painful conditions.²⁴

Paroxysmal disorders

The paroxysmal conditions associated with orofacial pain include the following: trigeminal neuralgia, glossopharyngeal neuralgia, and the more rare conditions of nervous intermedius neuralgia and superior laryngeal neuralgia. Occipital neuralgia involving the greater and lesser occipital nerves with occasional persistent pain between the paroxysmal episodes can be a source of headache. The pain sensations follow the distribution of these different nerves and are characterized by brief electric shock-like (lancinating or jabbing) pains lasting only seconds to minutes with pain-free intervals. One of the distinguishing aspects of trigeminal neuralgia and glossopharyngeal neuralgia is that the pain is evoked by trivial stimuli including use of the jaw (e.g. talking, swallowing, or even brushing the teeth) as well as just lightly touching the

face or mouth. Typically the second and third divisions of the trigeminal nerve are affected; the first division is only affected in 1–2% of the patients. The unilateral pain is severe with remissions lasting for days to years. Glossopharyngeal neuralgia pain is also a severe, transient, stabbing or burning pain located in the ear, base of the tongue, tonsillar fossa, or beneath the angle of the mandible. The paroxysms of pain are provoked by swallowing, chewing, talking, or yawning. Glossopharyngeal neuralgia is 50–100 times less common than trigeminal neuralgia.

Continuous disorders

The continuous neuropathic pain disorders associated with orofacial pain are primarily deafferentation pain syndromes related to compression or distortion, demyelination, infarction, or inflammation of the cranial nerves. Acute herpes zoster, chronic post-herpetic neuralgia, diabetic neuropathy, and neuromas are examples of continuous neuropathic pain syndromes. Burning mouth syndrome (BMS) is considered a neuropathic pain. The pain is persistent without major visible signs of spontaneous onset. Idiopathic persistent facial pain, historically referred to as atypical facial pain, is likely related to partial or complete deafferentation.²⁵ The previously mentioned atypical odontalgia or idiopathic odontalgia (historically referred to as phantom tooth pain) is a continuous, but variable, dull ache that sometimes is also described as a burning pain that typically follows dental treatment.²⁶

Headache attributed to associated extracranial pain disorders

Extracranial pain disorders associated with orofacial pain include pain related to the eyes, ears, nose, sinuses, throat, intraoral structures, neck, jaw, and cranial bones (Table 54.3).²⁷ The site of pain in the head, neck, and orofacial region is often not the primary source of the pain, making differential diagnosis extremely difficult. Referral of pain from one structure to seemingly another site is very common and is explained in part by the convergence of noxious input in the subnucleus caudalis.²⁸ Extracranial intraoral disorders and musculoskeletal cervical and temporomandibular disorders (TMD) will be discussed in more detail later in this chapter.

Pain in and around the eyes is relatively common, but seldom is it a result of noxious stimuli originating in the eye, extraocular muscles, or optic nerve. Rather, pain is commonly referred to the eye from other structures; however, pain referral to the face and mouth from the eye is uncommon. Pain perceived to be emanating from the ear is also very common. Although the source of the pathology may be from the ear (e.g. otitis externa and media, mastoiditis, eustachian tube disorders, neoplasia such as acoustic neuroma) (Fig. 54.1), again, the majority of ear pain is

Table 54.3 IHS 11 headache or facial pain attributed to extracranial disorders.

IHS 11.1	Headache attributed to disorders of cranial bones
IHS 11.2	Headache attributed to disorders of the neck
IHS 11.3	Headache attributed to disorders of the eyes
IHS 11.4	Headache attributed to disorders of the ears
IHS 11.5	Headache attributed to rhinosinusitis
IHS 11.6	Headache attributed to disorders of teeth, jaws, or related structures
IHS 11.7	Headache or facial pain attributed to TMD
IHS 11.8	Headache attributed to other extracranial structures

referred from another source. Pain in the nose and paranasal sinuses can arise from inflammation, infection, and malignant disease. The cause is primarily traction or distention of pain-sensitive structures leading to pain in the affected area. As with the above extracranial structures, the nose and sinuses commonly refer pain to adjacent structures, such as the teeth. Due to the broad sensory innervation of the throat, pain is referred to and from both adjacent and distant structures.

Intraoral pain disorders

Intraoral pain disorders include odontogenic pain, and pain conditions associated with mucogingival tissues, tongue, and salivary glands. Odontogenic pain is defined as pain associated with the teeth and periodontium. Tooth pain includes reversible and non-reversible pulpitis and pulpal necrosis. Teeth often refer pain to other teeth as well as to distant areas in the head, neck, and jaw. Pain conditions

associated with the supporting tissues of the teeth include acute apical periodontitis, acute apical abscess, and acute periodontal abscess. Mucogingival and glossal pain disorders may be localized or generalized throughout the mouth. The previously mentioned neuropathic burning mouth syndrome (BMS), also known as stomatodynia or oral dysesthesia, is characterized by burning mucosal, glossal, and/or palatal pain sometimes with associated taste sensations. Psychological disorders including anxiety and depression, systemic and localized disease, allergies, dietary deficiencies, hormonal changes, and xerostomia; all have been considered as possible etiologic possibilities. BMS is most prevalent in postmenopausal women. One concept is that chorda tympani hypofunction may play an important role in the pathology.²⁹

Localized pain conditions include acute necrotizing ulcerative gingivitis, recurrent aphthous stomatitis, herpes simplex candidiasis, and injury from trauma. Generalized pain conditions can result from physical, chemical, or drug-induced causes including chemotherapy, radiation therapy, dermatologic disorders, and systemic disease including pain secondary to HIV. Pain disorders associated with salivary disease or dysfunction can result from trauma, neoplasia, infection, e.g. mumps, inflammatory disorders such as acute suppurative and chronic recurrent sialadenitis and sialolithiasis, or salivary calculi or "stones".

Most intraoral pain disorders are familiar and routinely screened for by dental health professionals. However, atypical odontalgia, discussed above as a persistent neuropathic pain, is not familiar to general dentists. Even though the prevalence of atypical odontalgia is not known, studies suggest 3–5% of endodontically treated teeth may develop this persistent pain condition.^{30–32} Due to the lack of understanding of CNS pain mechanisms and the complexities of persistent pain syndromes, atypical odontalgia is a commonly mistreated diagnosis (Fig. 54.2). Imaging studies and clinical examination of the tissues appear normal with no obvious source of local pathology. However, the constant pain continues at the site for months and years.³³ The condition typically occurs subsequent to dental treatment and is thought to be peripherally and even centrally generated with possible alterations in the descending inhibitory pathways. This is consistent with the concept that non-responding persistent pain disorders, irrespective of their peripheral location, may be regulated centrally and have similar neuropsychologic impacts.³⁴ Also, there is some evidence that the sympathetic nervous system might also be involved in the maintenance of this continuous pain condition.³⁵

Atypical odontalgia is defined as a continuous, variable, diurnal tooth or tooth site pain of greater than 4 months' duration, with no obvious source of local pathology. It is often described as an aching, burning, and/or pressure sensation with a history of



Fig. 54.1 Acoustic neuroma can initially present as face and jaw pain.



Fig. 54.2 Panoramic radiograph revealing multiple unnecessary endodontic procedures for persistent neuropathic intraoral (atypical odontalgia) pain.

dental treatment, or trauma resulting in deafferentation of peripheral nociceptive nerve endings. The pain is located in a region where a tooth has been endodontically or surgically treated. Local provocation with temperature or loading and local diagnostic anesthetic injections are equivocal. Most atypical odontalgia patients have other comorbid pain conditions and higher scores for depression and somatization. Significantly lower scores on quality-of-life measures are also found.³⁶ Unfortunately, this diagnosis is often mistreated with repeated root canal therapies, apicoectomies, and even extractions. The result is worsening pain and irreversible harm to the patient. Treatment typically consists of tricyclic antidepressants, gabapentin and other membrane stabilizers, tramadol and topical lidocaine.

Musculoskeletal cervical and temporomandibular disorders

Musculoskeletal conditions affecting the jaw (TMD) and neck are the major cause of non-odontogenic pain in the orofacial region.³⁷ They include cervical spine and temporomandibular joint (articular) disorders; and cervical and masticatory muscle (non-articular) disorders. As with other musculoskeletal disorders, both neck and jaw symptoms wax and wane. Even though they are not life-threatening, they can significantly affect quality of life. They are defined as a collection of quite disparate musculoskeletal dis-

orders, articular or non-articular, that affect the neck and jaw, often with similar signs and symptoms.

Cervical spine disorders

Pain disorders associated with the cervical spine can involve the muscles, ligaments, facet joints, bones, discs, and neural tissues.³⁸ The traditional classification of these soft tissue and articular disorders includes diagnostic terms such as myositis, cervical sprain/strain, fibrositis, facet syndrome, osteoporosis, spondylosis, and osteoarthritis; articular hyp- and hypermobility; discogenic disease, and cervical nerve disorders. Eight percent of the US population seeks treatment for cervical spine disorders. Prevalence generally increases in frequency and intensity with age up to the fifth decade of life and is higher among women than men. A common traumatic cause of cervical symptoms is the damage that occurs during acceleration–deceleration (extension–flexion) injuries to the neck referred to as “whiplash” injuries.^{39,40} As with the various TMDs, the etiology of many of the cervical disorders is not well understood. Therefore, in the absence of fracture and disease, another classification has been proposed. It is modeled after the guidelines for low back pain and focuses on signs and symptoms to facilitate better communication among health professionals (Table 54.4).

Symptomatically, the cervical region overlaps with the craniofacial region, because the upper cervi-

Table 54.4 Cervical spine subclassification scheme.

Category 1	Neck symptoms without musculoskeletal and neurologic signs
Category 2	Neck symptoms without neurologic signs; musculoskeletal signs are present with a decrease in cervical range of motion and/or tension, plus pain on palpation of the cervical muscles
Category 3	Neck symptoms with musculoskeletal and neurologic signs that may include decreased or absent deep-tendon reflexes, weakness, and sensory deficits
Category 4	Radiation of symptoms or cephalic symptoms in addition to symptoms of category 1, 2, or 3; cephalic symptoms may include headaches, dizziness/unsteadiness, nausea/vomiting, tinnitus, visual problems, dysphagia, memory/cognitive problems, and reduced/painful jaw movements

cal nerves, discs, facet (zygapophyseal) joints, and muscles are potential sources of referred pain. The second and third cervical nerves innervate the angle of the jaw, the region inferior to the temporomandibular joint (TMJ), and parts of the ear, neck, and back of the head. Therefore, any irritation and/or dysfunction of these nerves can be associated with facial pain. As was discussed previously, convergence of input from the upper cervical nerves (C1, C2, and C3) with input from the trigeminal nerve in the trigeminocervical complex in the subnucleus caudalis in the brainstem can result in incorrect cortical discrimination of the actual source of the pain.⁴¹ Therefore, the patient may not be able to determine whether the source of the pain is in their neck, head, or jaw. Additionally, the upper cervical discs,⁴² as in discogenic disease; and facet joints,^{43–45} as in spondylosis and osteoarthritis, are other common sources of referred pain to the craniofacial region. Lastly, trigger points (tight bands) in cervical myofascial pain can be another

confusing source of referred pain to other cervical regions as well as the craniofacial region (Fig. 54.3).⁴⁶

Cervical findings are very prevalent in university-based orofacial pain centers. In a recent study at University of California San Francisco Center for Orofacial Pain, 86% of all patients seeking treatment had cervical findings and 42% had moderate to severe cervical muscle tenderness (Fig. 54.4).⁴⁷ It is the responsibility of the dentist to determine if the cervical findings are a primary source of the patient's orofacial pain complaint or a concomitant finding. A simple screening question can help with this differentiation. If chewing or other uses of the jaw do not change the patient's primary orofacial pain complaint, the dentist should be suspicious of a cervical spine disorder. The dentist should screen the cervical region for proper range of motion and soft tissue tenderness. If there is any significant mobility dysfunction or soft tissue tenderness, it needs to be investigated by a medical colleague with orthopedic training. Unfortunately in many cases, cervical diagnoses go undetected by the community dentist and patients are improperly treated.

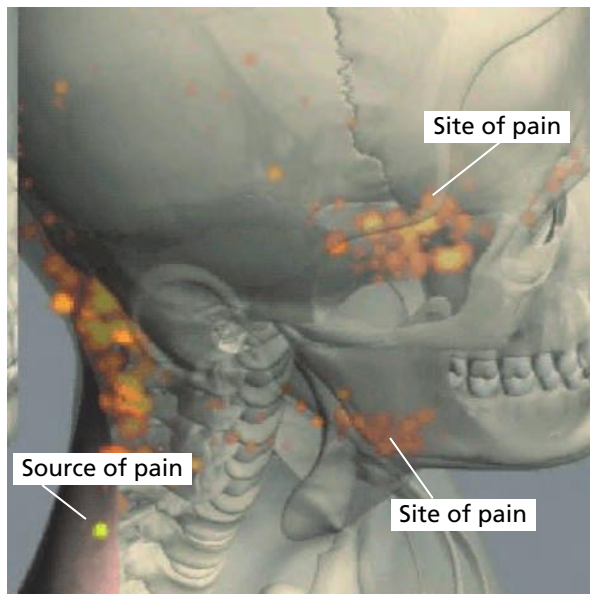


Fig. 54.3 Noxious input from the upper cervical nerves (C2 and C3) converges with noxious input from the trigeminal nerve primarily in subnucleus caudalis that can result in incorrect cortical discrimination of the actual source of the pain.

Temporomandibular disorders

Temporomandibular disorders (TMD) were referred to as a syndrome in the past. Common terms were "TMJ", "TMJ syndrome", or "TMJ pain-dysfunction syndrome". Presently they are considered a collection of various distinct articular or muscular conditions affecting the jaw, many times with similar signs and symptoms, but different underlying mechanisms. The common clinical presentation is any combination of jaw, face, head, or ear pain, TMJ noises such as clicking, popping or crepitus, or grating, and/or limited jaw opening, jaw catching, and locking.⁴⁸ Related symptoms, without proven cause and effect, include global headaches, neck pain, tinnitus, ear fullness or perceived hearing loss, and dizziness. Pain in the TMJ region is reported in approximately 10% of the population over 18 years of age (8–15% for women and 3–10% for men).⁴⁹ Recent studies have shown that these reported symptoms and clinical signs rarely

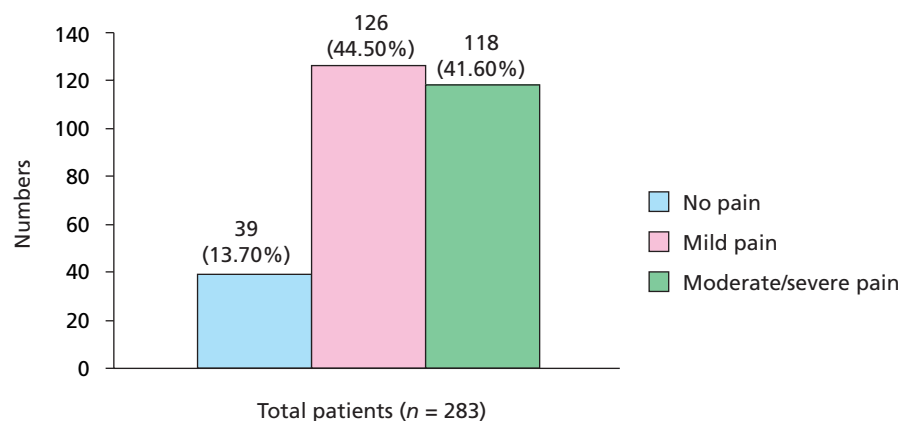


Fig. 54.4 Cervical muscle tenderness in patients seeking treatment at a university-based orofacial pain center.

become progressively more severe or disabling; however TMD-related pain is the most common persistent orofacial pain with a prevalence of about 10–15% worldwide. Epidemiological studies reveal that females seek treatment more than males. The gender ratio varies between cross-sectional studies from anywhere from 4:1 to 2:1 female to male. The peak age is approximately 35–45 years (the child-bearing years of females).⁵⁰

Although the etiology of the various subsets of TMD was in the past thought to directly relate to occlusal discrepancies and improper jaw relationships, presently the number of related contributing factors for each specific diagnosis is uncertain and many times unknown. Recent systematic reviews of randomized controlled trials (RCTs) have concluded that malocclusion is not directly correlated to the various subsets of TMD.⁵¹ Contributing etiologic factors include trauma, possibly parafunction, gender and hormonal factors, systemic factors, overuse of the masticatory system, and psychosocial and behavioral factors.⁵² Recent evidence suggests a specific gene expression, primarily in females, implicated as a risk factor for persistent orofacial pain.⁵³ The number of comorbid conditions is substantial in many patients seeking treatment, especially when the orofacial pain condition has become persistent.⁵⁴

Classification of articular disorders

The American Academy of Orofacial Pain’s 2008 classification of TMD includes a disparate group of articular and non-articular conditions.⁵⁵ TMJ disorders include congenital, developmental, or acquired disorders, disc derangement disorders, condylar dislocation, inflammatory disorders, non-inflammatory disorders, ankylosis, and fracture of the condylar process (Table 54.5).

Table 54.5 Temporomandibular joint disorders (modified from the American Academy of Orofacial Pain’s classification).

Congenital, developmental disorders and acquired disorders
Disc derangement disorders:
Disc displacement with reduction
Disc displacement without reduction
Temporomandibular dislocation
Inflammatory disorders:
Synovitis and capsulitis
Polyarthritides
Osteoarthritis (non-inflammatory disorders):
Active
Stable
Ankylosis
Condylar fracture

Developmental and acquired disorders

Developmental disorders of the TMJ include: agenesis (lack of development), aplasia (faulty development), hypoplasia (incomplete or under-development of the condyle), and hyperplasia (non-neoplastic over-development of the condyles).^{56,57} Acquired disorders include benign (e.g. osteoma, chondromas, synovial chondromatosis),⁵⁸ malignant or metastatic neoplasms (e.g. squamous cell carcinomas, primary nasopharyngeal tumors).⁵⁹ Approximately 3% of malignant neoplasia metastasize to the mandible, usually to the body or ramus.⁶⁰

Disc derangements

Disc derangement disorders represent an abnormal anatomical relationship or misalignment of the articular disc and condyle. Recent studies are revealing, however, that there is a great deal of variation in the disc position even in individuals without joint pain, joint noise, or jaw dysfunction. Magnetic resonance imaging (MRI) studies of asymptomatic volunteers have revealed that about one third of these individuals had displaced discs.^{61,62} Most of the volunteers had a partial disc displacement that reduced on opening with a small number not reducing on opening.^{63,64} Stretched or torn collateral discal ligaments are thought to be the reason why discs become displaced, although there is still uncertainty regarding the natural history of these derangement conditions.⁶⁵ Articular discs are typically displaced anteriorly and medially, but can also be positioned laterally,⁶⁶ or even posteriorly.⁶⁷

Derangement disorders occur as disc displacement with reduction, disc displacement without reduction, and disc adhesion. The most common disc disorders are disc displacements with reduction. The reduction occurs when the condyle moves into a more normal position with an unstable disc during translation, usually creating a joint sound (i.e. clicking or popping) at the time of the improved relationship of the condyle with the disc. On closing, the condyle moves posterior of the disc resulting in a closing click. Jaw clicking occurs in 50–55% of the adult population, representing a relatively common biologic variation. Disc displacement can be either asymptomatic (non-painful) or symptomatic (painful) at the time of the click. When a momentary hesitation or locking (catching) occurs, analogous to a shoulder impingement, the displacement is referred to as a disc impingement disorder. Symptomatic disc displacement and impingement disorders require management.

Disc displacement without reduction (disc fails to re-establish an improved anatomical relationship) can either be acute (less than 3 months’ duration) or chronic (3 months’ or greater duration) (Fig. 54.5). The acute condition is often quite painful with marked

reduction in condylar translation; however, it can on occasion be painless. The chronic condition is usually less painful with a normal or near-normal range of condylar translation over time. In the chronic stage, as the tissues adapt, pain-free range of motion increases, but articular changes may be seen on imaging due to the change in force loading.^{68,69} Lastly, disc adhesion is a disc disorder created by the disc adhering to the temporal component of the fossa creating a static disc position and altered joint mechanics. With disc adhesion, the condyle translates under the posterior band of the disc causing a click and then continues to move beyond the anterior band of the disc resulting in a second click. During jaw closure, the clicking occurs at the same two places as it did during opening.⁷⁰

Condylar dislocation

Condylar dislocation or open lock is a hypermobility condition of the jaw.⁷¹ It occurs when the condyle inadvertently becomes positioned anterior and superior to the articular eminence, during jaw opening or protrusion, and is unable to return to a closed position. It can be caused by trauma, extended periods of mouth opening such as a long dental appointment, or can be a manifestation of joint hypermobility. This condition requires the condyle to be manually dis-

tracted below the crest of the articular eminence, so the condyle can freely return to a closed position in the fossa. It is called an open lock or dislocation if a health provider has to reduce the anteriorly positioned condyle. This same condition is referred to as subluxation when the patient is able to self-manipulate the jaw back to a closed position.

Inflammatory and non-inflammatory disorders

Inflammatory joint disorders can occur as an inflammation of the synovium (synovitis) and/or joint capsule (capsulitis). This may be the result of trauma, infection, or cartilage degeneration, or the sequelae of a systemic polyarthritic or collagen disease (rheumatoid arthritis, lupus, Reiter's syndrome).⁷² Inflammatory joint conditions typically present with localized joint pain that limits jaw movements. Non-inflammatory joint disorders include primary and secondary osteoarthritis. Osteoarthritis (OA) is defined as a non-inflammatory degenerative condition of the joint characterized by deterioration and abrasion of articular tissue and concomitant remodeling of the underlying subchondral bone due to overload of the remodeling mechanism.⁷³⁻⁷⁷ In susceptible individuals mechanical overload can involve the production or release of free radicals, cytokines, fatty acid catabolites, neuropeptides, and matrix-degrading enzymes, resulting in a degenerative disease state.⁷⁸

OA is classified as primary OA when the etiology is unknown and secondary OA when an etiologic event or factor can be identified (e.g. gout, Cushing's disease, osteonecrosis, infections, Charcot's neuropathic pain). It can be further categorized into active OA or stable OA, sometimes referred to as osteoarthritis. Active OA is related to an active change or degeneration in the articular tissues, whereas stable OA refers to the recortication of the articular osseous structure with a lack of any further structural change (Fig. 54.6).

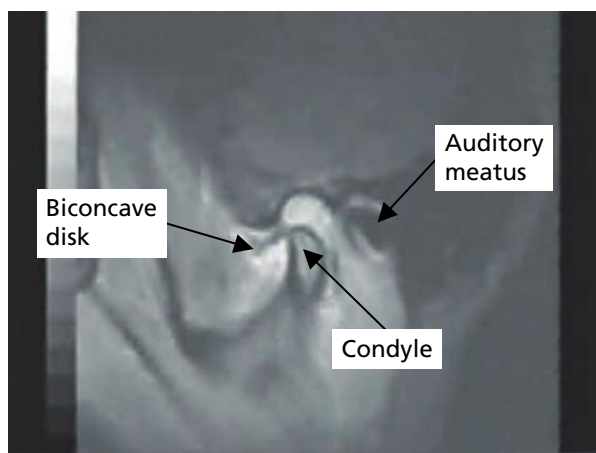


Fig. 54.5 MRI sagittal view of an anterior disc displacement without reduction.

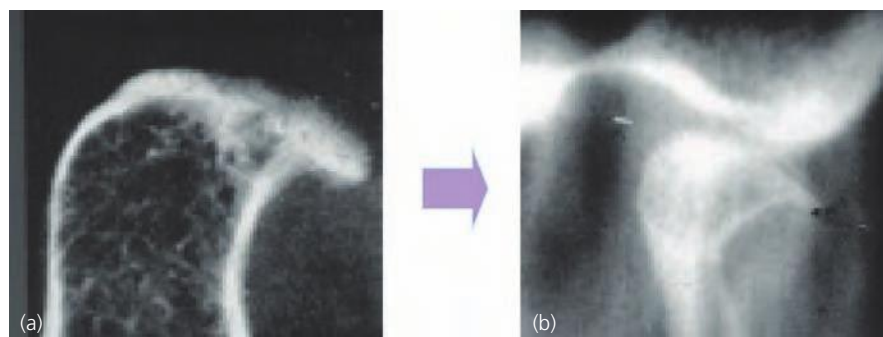


Fig. 54.6 (a) Active osteoarthritis is an active change or degeneration in the articular hard tissues. The cortical outline is no longer intact and there are erosions and subcondral bone cyst formation. (b) Stable osteoarthritis is a recortication (smooth surface) of the articular hard tissues and a probable end to any further structural change.

Ankylosis and fracture

Ankylosis is a hypomobility condition of the jaw when either fibrous or bony adhesions restrict condylar translation thus limiting jaw mobility and function (Fig. 54.7). Ankylosis is often the sequelae of trauma, including condylar fracture, infection, significant inflammation, or adhesions resulting from surgical intervention. The last articular diagnosis is fracture of the condyle. This condition usually results from direct trauma to the mandible. Fracture can be idiopathic or even iatrogenic, when secondary to another pathologic process. Ankylosis, condylar degeneration, or OA are possible sequelae.⁷⁹

Muscular disorders

The underlying mechanisms that cause masticatory muscle pain are similar to those that cause skeletal muscle disorders throughout the rest of the body. Some mechanisms thought to be related to muscle pain include overuse, localized ischemia, spontaneous activity of deep nociceptors, sympathetic nervous system hemodynamic perfusion changes, and changes in descending anti-nociceptive modulation.⁸⁰⁻⁸² Endogenous substances such as bradykinin, serotonin, prostaglandins, neuropeptides, substance P, and others are thought to sensitize the peripheral nociceptive nerve endings resulting in pain in the muscle.⁸³ Deep tissue inflammation can upregulate nociceptive behaviors which may not be effectively modulated by neuropeptide antagonists, resulting in more pain.⁸⁴

The concept that bruxism is a major cause of masticatory muscle pain is being questioned in that most subjects that brux do not have muscle tenderness.⁸⁵ Also, the historic dental view that masticatory muscle pain is related to occlusal interferences is no longer a

viable concept. Systemic conditions that can produce muscle pain include polymyalgia rheumatica, polymyositis, lupus erythematosus, and fibromyalgia. It is also important to distinguish between the cranial muscle tenderness associated with the various headache types and primary masticatory muscle tenderness, because even though the symptoms of these conditions can be very similar, the source of the pain is quite different, requiring very different management strategies.⁸⁶ Masticatory muscle disorders include local myalgia, myofascial pain, centrally mediated myalgia, myospasm or trismus, myositis and tendonitis, myofibrotic contracture, and neoplasia (Table 54.6).

Local myalgia

Local myalgia is characterized by localized or regional dull ache and stiffness in the masticatory muscles. There is typically little to no pain at rest. The pain increases with jaw function and often results in a decreased active jaw opening that increases with passive stretch. The patient often reports muscle weakness, fatigue, and increased pain when eating hard foods, yawning, and with prolonged opening. Local myalgia can include such conditions as protective muscle splinting as a result of TMJ pain, muscle fatigue, and delayed-onset muscle soreness. Delayed-onset muscle soreness or postexercise muscle soreness is a painful muscle condition related to intense or unaccustomed use of a muscle.⁸⁷ The overuse results in interstitial inflammation and a delayed pain in the muscles 8–24 hours later.

Myofascial pain

Myofascial pain is characterized by a regional or local dull, aching muscle pain that increases during function. Clinically there are localized tender sites or trigger points in the muscle, tendon, or fascia. Palpation of the trigger point provokes pain referral to a distant site such as the teeth, ear, or head and this must be present to meet the criteria for myofascial pain (Fig. 54.8).^{88,89} Patients may also report muscle stiffness, ear symptoms such as tinnitus, decreased mouth opening that can be passively stretched by more than 4 mm, and hyperalgesia in the region of the referred

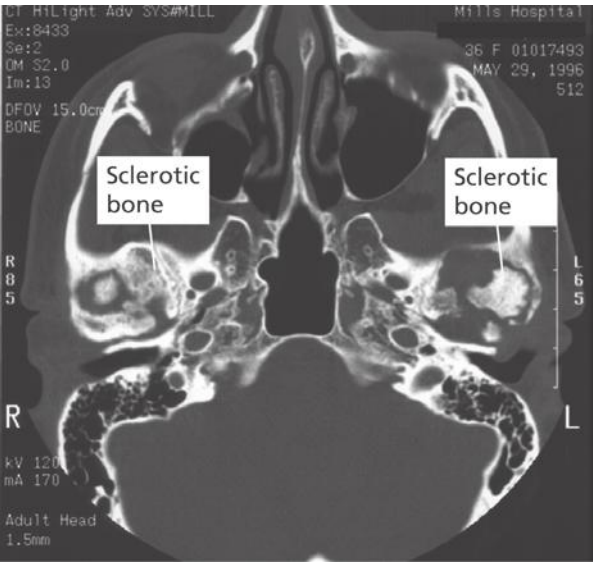


Fig. 54.7 CT scan of bony ankylosis following temporomandibular surgery.

Table 54.6 Masticatory muscle disorders (modified from the American Academy of Orofacial Pain’s classification).	
Local myalgia	
Myofascial pain	
Centrally mediated myalgia	
Myospasm	
Myositis and tendonitis	
Myofibrotic contracture	
Neoplasia	

pain. Myofascial pain is not considered an inflammatory process; whereas tendonitis is an inflammation and/or soreness in the tendinous attachments of masticatory muscles.

Centrally mediated myalgia

Fibromyalgia, a type of general or global muscle pain involved with CNS upregulation, can be confused with local or regional muscle pain if the clinician is not comprehensive with the history taking and physical assessment process.⁹⁰ Fibromyalgia is characterized by a continuous, aching pain in many areas of the body. It is associated with generalized fatigue, chronic headache, irritable bowel syndrome, sleep disturbance, and emotional distress including anxiety, depression, and somatization. The American College of Rheumatology has developed a screening examination for fibromyalgia. They have identified 18 points (9 pairs) on the body that are normally non-painful with palpation. If 11 or more of these points are painful to palpation and there is pain in three out of four quadrants of the body for at least 3 months, this suggests a positive fibromyalgia screening.⁹¹ Patients suspected of fibromyalgia should be referred to rheumatology for further evaluation. Centrally mediated muscle pain is thought to be associated with the upregulation of central mechanisms. The pain may result from prolonged nociceptive input to the CNS resulting in antidromic effect on the afferent peripheral neurons. This can result in the release of pain-modulating substances such as bradykinin and substance P that cause pain. Chronic exposure to emotional stress and other sources of deep pain input, such as chronic upregulation in the autonomic nervous system, are thought to be possible underlying sources of the centrally mediated pain.

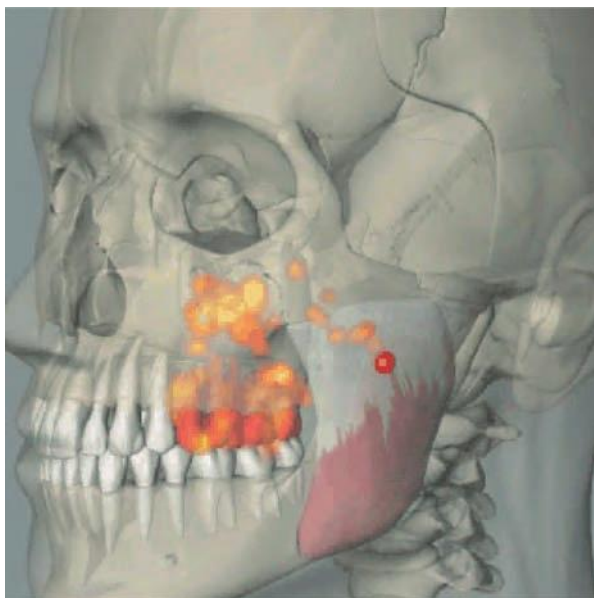


Fig. 54.8 Localized tender sites or trigger points in the muscle (red dot), tendon or fascia with referral to a distant site such as the teeth.

Myospasm

Myospasm or trismus is an acute muscle disorder characterized by a sudden, involuntary, tonic contraction (fasciculation) of a muscle. The acute pain is present at rest as well as during function, and function is significantly limited. The jaw cannot be manually stretched open beyond the patient's voluntary opening (hard end-feel) unlike local myalgia or myofascial pain. Myospasm involves the entire muscle and produces a dramatic increase in electromyography (EMG) activity in the muscle similar to maximum voluntary clench, again unlike the slight increase associated with myalgia or myofascial pain.⁹² Myospasm is a relatively rare muscle disorder of the masticatory muscles. One common cause of myospasm of the medial pterygoid is a mandibular nerve block. The risk of myospasm increases when the injection is repeated several times to achieve profound anesthesia. This inadvertently may cause local trauma, bleeding, or the introduction of intraoral organisms into the muscle resulting in myospasm.

Myositis and tendonitis

Myositis is defined as a true inflammation of muscle usually due to direct trauma and/or infection. Swelling, erythema, and increased temperature over the entire muscle are common. Continuous severe pain and diffuse tenderness result in increased pain with function resulting in significant limitation in the range of motion. Ossification of a muscle can occur secondary to inflammation which leads to myositis ossificans.⁹³ The inflammation may occur in the tendinous attachments of the muscle referred to as tendonitis or tendomyositis. When the tendinous attachments become inflamed, there is severe pain with use and palpation. This inflammatory condition often requires aggressive anti-inflammatory management.

Muscle contracture

Muscle contracture or myofibrotic contracture is a painless shortening of a muscle as a result of fibrosis or scarring of the supporting tendons, ligaments, or muscle fibers. Jaw opening is limited and usually not painful except when the muscle is extended beyond its functional length. When the opening is stretched there is an unyielding firmness or hard end-feel. This muscle disorder can occur following a long period of limited range of motion, such as with extended use of intermaxillary fixation.

Muscle neoplasia

Muscle neoplasia is defined as a new, abnormal, or uncontrolled malignant or benign growth of muscle tissue, and may or may not be associated with pain. The tumors can be present within the muscle or more

commonly are extensions from adjacent structures or metastases from remote sites. Swelling, trismus, paresthesias, and possibly pain are common features of these rare tumors. Confirmation with imaging and biopsy is required when a tumor is suspected.^{94,95}

Assessment of musculoskeletal disorders

Screening history and examination

The collection of baseline records and indicated diagnostic tests is fundamental to the proper management of jaw disorders. The extent to which any or all of the elements of evaluation are pursued depends on the magnitude of the presenting complaints and the potential for the problem to progress physically and psychologically. Screening for jaw disorders is an essential part of all routine dental and orofacial pain examinations.⁹⁶ Basic assessment of all patients should also include behavioral and psychosocial screening by the dentist during the history-taking process. The history should include questions to evaluate behavioral, social, emotional, and cognitive factors which may initiate, sustain, or result from the patient's condition. If significant findings are identified and recorded, a comprehensive history and examination should be conducted.⁹⁷ Appropriate imaging studies of the jaw and craniofacial structure, as well as other diagnostic tests including blood and urine chemical analyses are important diagnostic tools in specific cases. Also, diagnostic somatic and sympathetic nerve blocks, intra-articular TMJ injections, and trigger point injections are extremely valuable as diagnostic aids.

Comprehensive history and examination

The gold standard for diagnosis of TMD is still the interpretation of findings from a comprehensive history and comprehensive physical examination.^{98,99} The comprehensive history parallels the traditional medical history and review of systems, and consists of the chief complaint(s), history of the complaints, medical history, dental history, and personal history (social and family). It is important for the dentist to not get lost in multiple complaints.

The complaints should be rank ordered beginning with the chief complaint by the patient. The history should include a chronological history for each complaint. The comprehensive physical examination consists of a general inspection of the head and neck, including: a visual inspection and palpation; cursory evaluation of the cranial nerves; a comprehensive orthopedic evaluation of the TMJ and cursory evaluation of the cervical spine; a masticatory and cervical muscle evaluation; and an intraoral evaluation.

Behavioral and psychosocial assessment

With pain being defined at the beginning of this chapter as an unpleasant sensory and *emotional* experience associated with actual or potential tissue damage or described in terms of such damage, it is readily apparent that a biopsychosocial model is required to manage orofacial pain. This assessment becomes especially important when these pain conditions become persistent. It has long been established that persistent pain disorders result in a number of biologic and psychologic changes. Persistent pain patients exhibit significant anxiety, depression, somatization, high utilization of health services, and frequent use of pain medications.¹⁰⁰ Because patients develop maladaptive patterns that prolong suffering and prevent effective symptom management, the comprehensive history needs to include an evaluation of the behavioral, social, emotional, and cognitive factors that either can sustain or result from their pain complaints.

Psychological and psychosocial assessment can be effectively incorporated as part of the pain history with standardized self-report questionnaires. A dual-axis system for physical and psychological assessment for TMD referred to as the Research Diagnostic Criteria for TMD (RDC) was developed by an international team of clinicians–researchers in 1992.¹⁰¹ In 2005 they established a website with Axis I physical diagnoses, viable for research diagnostic classification standardization, and Axis II for psychological assessment.¹⁰² The psychological assessment measures include a visual analog pain scale, mandibular function questionnaire, depression and somatization system checklists, and a psychosocial functions graded chronic pain scale.

Self-report questionnaires are numerous and can include the Holmes and Rahe Scale for life changes,¹⁰³ the Interactive Microcomputer Patient Assessment Tool for Health (IMPATH),¹⁰⁴ and the TMJ Scale¹⁰⁵ as screening devices. These tests may indicate the need for a more extensive evaluation from a psychologist or psychiatrist. They may consider the need for other psychological inventories such as the Symptom Checklist-90-Revised (SCL-90-R), Post-traumatic Stress Disorder (PTSD) Civilian Checklist, Minnesota Multiphasic Personality Inventory (MMPI), Hamilton Depression Scale, West-Haven-Yale Multidimensional Pain Inventory, McGill Pain Questionnaire, and the Million Behavioral Questionnaire.^{106,107}

Imaging

Imaging of the TMJ and orofacial structures may be necessary to rule out structural disorders of the jaw and other medical conditions that may be masquerading as a jaw disorder.¹⁰⁸ Imaging should primarily be ordered after a comprehensive examination suggests some form of joint pathology or when there is a

suspicion of some other serious non-musculoskeletal pathology such as an infection or tumor. Corrected tomography has been the choice of imaging for hard tissue pathology in the TMJ.¹⁰⁹ Cone-beam computed tomography (CBCT) is the most accurate method for radiographically examining patients with suspected TMJ degenerative disease or other osseous pathology and structural aberrations (Fig. 54.9).¹¹⁰

MRI has diverse capabilities for the examination of suspected TMJ soft tissue disorders and pathology, i.e. disc displacement, effusion, and tumors. As the resolution improves and technical advancement occurs, MRI is clearly becoming the study of choice for complex problem solving. MRI studies on autopsy series of oblique sagittal and coronal views has been found to be approximately 95% accurate in determining disc position.¹¹¹ For the study of routine jaw disorders, MRI is rarely indicated for non-surgical TMD management, because the study does not usually change the treatment approach. However, if surgical intervention is a consideration, a MRI study can be a critical diagnostic aid for surgical treatment planning.

Additional diagnostic tests

A variety of additional diagnostic studies are available for use in select cases to assist in confirming a physical diagnosis. Diagnostic tests may include laboratory tests (blood chemistries) for systemic arthritides; neural blockade, somatic, and sympathetic nerve blocks; diagnostic specific injections of TMJ and trigger point injections of the cervical and masticatory muscle; and spray and stretch to determine if a soft-tissue trigger point is a source of pain. A physical

therapy evaluation is helpful to determine the appropriateness of a specific rehabilitation program, to evaluate the cervical spine as a source of orofacial pain, and to perform a fibromyalgia screening examination to determine the scope of the pain presentation (Fig. 54.10).



Fig. 54.10 Palpation of the lateral epicondyle region by a physical therapist as part of the fibromyalgia screening.

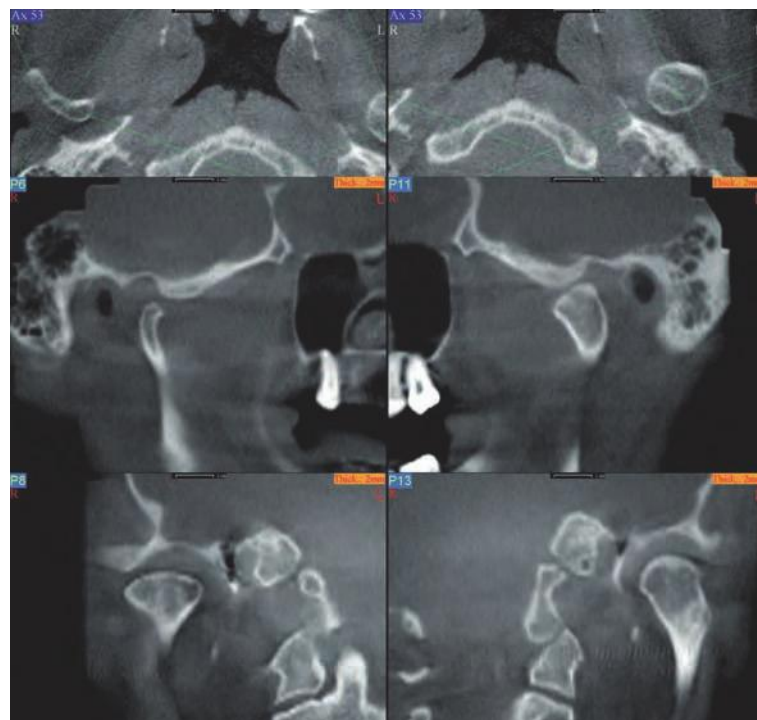


Fig. 54.9 Cone-beam CT revealing hyperplasia of left condyle and subtle degenerative changes in the right condyle.

Adjunctive diagnostic devices

There are a number of electronic devices marketed to diagnose jaw disorders (TMD) including electromyography testing, jaw tracking, thermography, sonography, and vibration analysis. However, peer-reviewed articles have questioned the sensitivity (percentage of correctly diagnosed patients) and reliability (percentage of correctly diagnosed normals) of these technical diagnostic TMD tests. There is insufficient evidence that vibration analysis of the TMJ can diagnosis disc displacement with reduction any more accurately than the use of the stethoscope and palpation.¹¹² Many of the devices lack research support and are subject to great biologic variability.^{113–116} The risk of an incorrect diagnosis often based on over-interpretation of insignificant or normative physiological data can result in mistreatment or over-treatment of the patient. Jaw tracking instrumentation, that provides additional measurement data regarding mandibular movements, does not justify treatment of the occlusion for TMD patients. The “high-tech electronic devices” that record jaw relationships and jaw movement are often recording biologic variations that can be misleading. The interpretation that these data are supportive evidence that the occlusion needs to be altered from a TMD management standpoint is not in agreement with evidenced-based research. Jaw tracking can have technical benefit for the dentist that wants to use a semi-adjustable or fully adjustable articulator when comprehensive restorative, prosthodontic or implant dentistry is being contemplated for dental treatment.

The US Food and Drug Administration (FDA) and the American Dental Association (ADA) used the term “adjunctive diagnostic devices” when they approved jaw-tracking, EMG, and sonography devices *only* for the measurement of clinical jaw signs.¹¹⁷ Both organizations did not approve the devices for having the ability to make a diagnosis,^{118,119} with the ADA stating “... the interpretation of the test results rests with the dentist”. Also, the literature to date on the use of thermography for the diagnosis of orofacial pain has revealed conflicting evidence.¹²⁰ The primary concern with using these adjunctive devices is their low degree of diagnostic specificity, resulting in a high number of false-positive diagnoses and unnecessary treatment.¹²¹

Dental casts

As mentioned above, due to the many variables involved, it has been difficult to establish significant cause and effect correlations between the occlusion and TMD.¹²² Based on the literature, treatment involving the occlusion is rarely appropriate for specifically treating TMD. Therefore, because TMD is not directly correlated to the occlusion or specific jaw relationship, diagnostic casts to study the occlusion have little value in the assessment of TMD. However, casts can

be an important assessment aid for identifying wear patterns from sleep and awake bruxism and longitudinal comparisons of jaw relationship and/or occlusion changes secondary to an articular or muscular TMD condition. Also, they are extremely beneficial during the treatment planning process for complex restorative, prosthodontic, and orthodontic including orthognathic surgery cases. Cast surgery on mounted diagnostic casts prior to surgery is helpful for both the surgeon and the orthodontist.

Management of musculoskeletal disorders

The majority of patients with jaw disorders achieve good symptomatic relief with a medical model using non-invasive management.¹²³ Long-term follow-up studies show that 85% to more than 90% of patients have few or no symptoms after conservative treatment.¹²⁴ A recent meta-analysis of TMD treatment need reports an approximate 16% need for adults.¹²⁵ Jaw disorders are similar to other musculoskeletal disorders, but currently not enough is known about the natural course of most jaw disorders and which signs and symptoms will progress to more serious conditions. As in other musculoskeletal conditions, the TMD signs and symptoms vary and may be transient and self-limiting, resolving without serious long-term effects. Recent reports on the course of untreated disc displacement without reduction suggest that a natural resolution of symptoms occurs over time. The research from Boering and co-workers also suggests a natural course for not only some disc disorders but OA, as well. The peer-reviewed literature strongly supports that a special effort should be made to avoid aggressive, irreversible therapy for articular and non-articular jaw disorders.^{126,127}

As previously mentioned, treatment of the occlusion is rarely appropriate for specifically treating the subsets of TMD, based on the recent systematic reviews of RCTs.¹²⁸ There are many testimonials and belief systems that claim that occlusion or incorrect jaw relationship is the primary etiologic factor for TMD, but scientifically a direct correlation is largely unproven.^{129–133} Thus, based on the efficacy of the non-invasive medical model for the treatment of TMD and the lack of evidence that occlusal therapy is necessary, the old concept of phase I and phase II treatment is obsolete. As a patient’s TMD signs and symptoms improve with the conservative medical management model described in this chapter (“phase I treatment”), there is no scientific evidence to support the need for subsequent definitive treatment of the occlusion (“phase II treatment”).^{134–136} When TMD signs and symptoms resolve, the only compelling reasons to proceed with treatment of the occlusion would be based on dental (tooth and/or periodontal) pathology, mobility, discomfort, or esthetic reasons. And even then, great care should be

taken with this “at-risk” patient relative to minimizing trauma to the patient’s jaw including minimal appointments, limited jaw opening, and reduced force loading. Less rather than more complex dental procedures should be contemplated, especially when considering making changes in the occlusion.

A multidisciplinary medical model that includes patient education and self-care, cognitive behavioral intervention, pharmacologic therapy, physical rehabilitation, and/or orthopedic appliance therapy is endorsed for the management of nearly all patients (Table 54.7).¹³⁷ The management goals should be diagnosis specific. The common goals are reduction of pain, reduction of adverse loading, improvement of mobility and function, and restoration of activities of daily living. The emphasis should be on conservative therapy that facilitates the musculoskeletal system’s natural healing capacity. The management requires the patient to assume responsibility for the physical and behavioral management of his or her own problem. There is a small number of TMJ disorders that require surgical intervention, e.g. developmental conditions resulting in significant structural, functional and/or esthetic problems, tumors, ankylosis or fracture, that are discussed elsewhere in this textbook.^{138,139}

Patient education and self-care

The success of a self-care program depends on patient motivation, cooperation, and adherence. A successful program begins with a thorough explanation of the patient’s diagnosis or diagnoses and a discussion about the patient’s prognosis. Once the patient understands their condition, the self-care program is more meaningful and patient adherence is higher. When the jaw disorder is mild, patient education and instruction in a self-care program may be all that is required.^{140,141} The self-care instructions should be specific for the patient’s diagnosis and clinical presentation; and should be directed toward achievable goals. These instructions typically include resting the masticatory system through correct relaxed jaw posture awareness with an emphasis on not contacting opposing teeth unless swallowing, i.e. preventing awake clenching and grinding of the teeth. Patients need to be mindful of any other oral habits that may be delaying their recovery such as jaw protrusion, cheek sucking, or tongue bracing behaviors. Patients generally benefit from a soft diet and slower mastication. If they need joint protection, as in the case of joint inflammation or disc instability, patients benefit

from chewing on the affected side, limiting incising their food and avoiding prolonged opening.¹⁴² The instructions may also include palliative recommendations such as moist heat to increase circulation and relax muscles for local myalgia and myofascial pain. On the other hand, ice is the preferable thermal modality to decrease pain and inflammation when there is an acute joint inflammation. Self-massage of the affected muscles is another method to relax tight muscles and improve local circulation. Sometimes gentle range-of-motion exercises are appropriate to lubricate the joint, reducing joint pain and increasing range of motion. Other instructions may focus on head and neck posture, ergonomics and sleep positioning, and include general suggestions for improved health habits, cardiovascular exercise, and sleep hygiene.

Cognitive behavioral intervention

Behavioral intervention is an important part of the overall medical management program for patients with jaw disorders.¹⁴³ Simply making patients aware of their jaw habits is often enough to improve jaw relaxation skills, but changing persistent habits may require a structured program with a clinician trained in behavior modification strategies. Comprehensive stress management and counseling programs using a combination of EMG biofeedback, progressive relaxation, diaphragmatic breathing, and self-directed changes in lifestyle appear to be more effective than any one behavioral treatment procedure in isolation. Unfortunately, biofeedback is not generally effective for treatment of sleep bruxism.¹⁴⁴ Patients with persistent pain or who have experienced multiple treatment failures typically require in-depth psychological evaluation and treatment by a mental health professional such as a psychologist or psychiatrist.

Pharmacologic therapy

The indicated classes of pharmacologic agents include analgesics, non-steroidal anti-inflammatory agents, corticosteroids, anxiolytics, muscle relaxants, low-dose (pain-dosing) antidepressants, and nerve membrane stabilizers. The non-opiate analgesics, such as acetaminophen, are effective for mild to moderate pain, whereas the opioid narcotics, such as codeine, ultram, hydrocodone, and demerol, should only be used short term for controlling more severe acute pain.¹⁴⁵ Opioid narcotics produce tolerance and dependence and should be used on a time-contingent basis.¹⁴⁶ Non-steroidal anti-inflammatory drugs (NSAIDs), such as ibuprofen, naproxen, or nabumetone, are effective analgesics and anti-inflammatory agents. They are prescribed for painful articular inflammatory disorders, not usually for muscle disorders. They must be prescribed at therapeutic levels over a significant period of time to achieve the desired anti-inflammatory effects. Caution should be exer-

Table 54.7 Multidisciplinary management model.

Patient education and self-care
Cognitive behavioral interventions
Pharmacologic therapy
Physical rehabilitation
Orthopedic appliance therapy

cised when prescribing these medications, as they can cause gastrointestinal irritation and bleeding. When NSAIDs are contraindicated or ineffective, corticosteroids can be considered for persistent localized moderate to severe joint inflammation. These medications can be delivered orally, e.g. with a methylprednisolone dosepak or they can be injected directly into the temporomandibular joint, e.g. prednisolone.

The benzodiazepines, such as alprazolam, lorazepam, and diazepam, are most commonly prescribed for their anti-anxiety effects. These drugs act as depressants when used long-term; thus they should only be used short-term for acute muscle pain/spasm (trismus), to relax patients prior to jaw manipulation for acute disc displacement without reduction, or for sleep disturbances associated with anxiety.^{147,148} Muscle relaxants, such as cyclobenzaprine, metaxalone, and tizanidine, are useful for acute and/or chronic muscle pain. Cyclobenzaprine, which is quite similar chemically to the tricyclic antidepressants, is one of the drugs of choice by rheumatologists and pain specialists for generalized persistent muscle pain.^{149,150} Cyclobenzaprine (5–10mg at bedtime) is reported to also improve sleep architecture and reduce sleep bruxism. Tricyclic antidepressants such as nortriptyline, doxepin, and desipramine, when used in low dosages (10–75mg) have been shown to act on the CNS neurotransmitters, improving the patient's pain regulation. They are prescribed for persistent pain patients who have neuropathic pain, persistent myofascial pain, and poor restorative sleep.^{151,152} Studies suggest that they may also reduce sleep bruxism. Membrane stabilizers or anti-seizure drugs, such as carbamazepine and phenytoin, have been replaced with improved medications like gabapentin and pregabalin for persistent pain conditions including neuropathic atypical odontalgia pain. These second-generation medications have less significant side-effect profiles than the former drugs.¹⁵³

Physical therapy

Physical therapy is recognized as an effective, conservative approach for patients with jaw disorders.¹⁵⁴ A physical therapy consultation should be performed by a physical therapist and involves a comprehensive head, neck, upper quarter orthopedic evaluation, and fibromyalgia screening. Based on these findings, a rehabilitation program is designed to restore optimal masticatory, cervical, and upper quarter function as appropriate. The rehabilitation strategies are based on the patient's diagnoses, clinical findings, and the patient's personal treatment goals. Treatment goals commonly include pain control, optimizing joint biomechanics and range of motion, restoring functional muscle strength and endurance, and restoring the ability to perform activities of daily living of the jaw, such as talking, chewing, yawning, and singing; or of the cervical and upper quarter regions such as sitting, reading, driving, lifting, and reaching.

The management begins with patient education and instructions in a structured self-care program. The next goal is to reduce pain as efficiently as possible, so the patient can begin exercising their jaw, neck, and upper quarter as appropriate. Physical agents such as moist heat, cold packs, transcutaneous electrical nerve stimulators (TENS), iontophoresis, ultrasound, and vapocoolants may be used initially for pain control. Moist heat increases local circulation and relaxes muscles. This improves the nutrition to the tissue and removes the inflammatory by-products from the area. Cold packs anesthetize the area reducing pain, and they initially decrease the circulation reducing an inflammatory response. TENS inhibits "c" fibers, thereby reducing the propagation of pain. Iontophoresis uses an electric galvanic stimulator to deliver corticosteroids through the tissues. Ultrasound is a thermal device that produces sound waves. The sound waves penetrate the skin to a depth of up to 5cm vibrating the tissue and creating a mechanical heat that results in increased local circulation and muscle relaxation (Fig. 54.11). Vapocoolants anesthetize the skin, so that a muscle with tight bands (trigger points) can be stretched, deactivating the referred pain from the trigger points (Fig. 54.12).



Fig. 54.11 Ultrasound application to the right masseter region.



Fig. 54.12 Spray and stretch technique to eliminate a masseter muscle trigger point that is referring pain to the head and teeth.

Soft-tissue mobilization and myofascial release techniques are commonly used to increase local circulation, restore normal muscle tone, and deactivate myofascial trigger points (Fig. 54.13). Once the patient has better pain control, the therapist can mobilize the joints as needed, and begin a range-of-motion and muscle conditioning program. Joint mobilization is essential for the acute disc displacement without reduction. The therapist manually distracts and translates the condyle under the posterior band of the displaced disc, restoring full condylar translation and the former condition of disc displacement with reduction. Joint mobilization is also indicated when jaw mobility is limited for other reasons. Mobilization techniques to restore rotation and translation for optimal joint biomechanics are performed before muscle stretching is initiated. This same philosophy is applied to other joints of the upper quarter. Once improved joint mechanics are achieved, range-of-motion and stretching exercises are introduced to facilitate normal movement of the joint and muscles. Lastly, muscle strengthening and conditioning ensure the safe return to previous levels of function without reinjury. Patients that only do palliative treatments are likely to reaggravate their tissues when they advance their diet, attempt to use their jaw normally, or resume other activities of daily living.

Physical therapy for persistent pain conditions requires less peripheral (local) management and involves strategies to influence the CNS. The therapist must address sleep positioning and sleep hygiene to enhance restorative sleep. It is also essential to motivate patients to be more active, establishing realistic and achievable exercise goals to improve general strength and endurance. Education about diaphragmatic breathing and relaxation training is another important component in the rehabilitation program; and lastly, stressing the importance of proper pacing of their activities, so as not to overdo it when a patient is having a better day, leading to relapse and increased pain the following day. Better restorative sleep, increased activity, improved relaxation skills, and improved pacing with activities of daily living improve the patient's central pain modulation system.



Fig. 54.13 Soft tissue mobilization of the left masseter.

Throughout physical therapy, the patient should be frequently reassessed by the physical therapist and the referring clinician. The patient should be achieving their management goals and requiring less and less treatment with the therapist. If the patient is not improving as expected, additional diagnostic testing and/or consultations must be considered. Ultimately, the patient should be discharged from treatment with an independent home management program.

Orthopedic appliance therapy

Orthopedic appliances, also referred to as orthoses, occlusal splints, bite splints, bite plates, night guards, or bruxism appliances, have been commonly used in the management of jaw disorders.¹⁵⁵ There is general agreement that some patients with myofascial pain, articular disc instability, and joint inflammation sustain their symptoms when they brux their teeth at night. These patients may benefit from appliances worn during sleep. Also, appliances have dental benefits with regard to protecting the teeth and/or restorations from wear and fracture, as well as decreasing tooth sensitivity and mobility. Appliances should only be worn during sleep in order to limit the amount of time the appliance masks the periodontal proprioceptive input that allows the patient to return to their intercuspal position when the appliance is removed. Eventually random wear at night, during periods of increased stress, should be all that is necessary.

There still is great debate about how to design an appliance for the greatest efficacy.¹⁵⁶ Systematic reviews of RCTs suggest that appliance design, i.e. specific types of occlusal interfaces and/or jaw positions, are not a very critical factor for the management of bruxism or masticatory muscle pain. Most dentists agree, however, that orthopedic appliances should cover all the teeth on either the maxillary or mandibular arch in order to prevent irreversible changes in the occlusion (Fig. 54.14). Partial coverage appliances can allow teeth to extrude or intrude and/or cause condyles to reposition themselves within the articular fossae. The type of material, hard acrylic or soft vinyl, also is no longer an important consideration. The efficacy of soft vinyl appliances, which were questioned in the past, has been reported to be comparable to that of hard acrylic appliances in recent studies.¹⁵⁷

Currently an appliance called the Nociceptive Trigeminal Inhibition (NTI) splint is being widely marketed to the dental profession and the public (Fig. 54.15). The appliance covers the maxillary central incisors allowing contact on jaw closure with only the lower incisors based on the old concept of the Lucia Jig and the Leaf Gauge by Long.^{158,159} The anterior contact reportedly decreases jaw elevator muscle (EMG) activity and associated muscle pain and headache. However, a recent study reported that the decrease in postural EMG activity in a myofascial group was short-lasting, and thus should not be

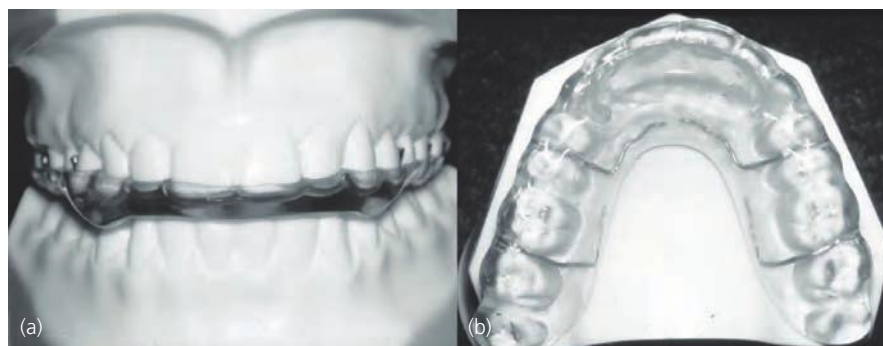


Fig. 54.14 (a, b) Frontal and occlusal views of a maxillary orthosis.

considered as evidence that the appliance has a long-term muscle relaxation effect.¹⁶⁰ Anterior tooth contact only increases TMJ loading, possibly resulting in an exacerbation of an articular condition, e.g. inflammation or OA of the TMJ. Studies have revealed no significant differences in muscle pain between a full-coverage stabilization appliance or an NTI appliance after 3 months.¹⁶¹ At 6-month follow-up, the stabilization splint was favored over the NTI splint by a number of the patients with the added advantage of less risk for occlusal changes.¹⁶²

In summary, recent scientific reviews and RCTs find no difference in efficacy between active full-occlusal coverage appliances versus palatal coverage placebo appliances for the treatment of bruxism.¹⁶³ They also have found that full occlusal coverage stabilization appliances performed no better than active, non-occluding palatal coverage appliances for the treatment of masticatory muscle pain.^{164–166} The effectiveness of occlusal appliance therapy versus conservative treatment for masticatory muscle pain as suggested in this chapter is comparable in recent studies. One systematic review concluded that there was insufficient evidence to suggest that stabilization appliances are any more effective than other treatments, such as self-care, acupuncture, biofeedback,

relaxation, and exercises, for treatment of TMD pain.¹⁶⁷

Conclusion

Managing pain and relieving suffering should be at the core of the health professional's commitment to patients. Proper pain management is mandated in order to prevent the consequences of unrelieved pain. Recent scientific and clinical advances have recognized that unrelieved pain can cause delayed healing, an altered immune system, an altered stress response, vegetative symptoms, and permanent alterations in the peripheral and central nervous systems, resulting in persistent pain syndromes. Therefore, there is every medical and ethical reason to treat pain, including orofacial pain, in a timely manner with every resource available. In doing so, we need to provide irreversible, non-invasive treatment, when at all possible, that facilitates the natural healing process and that also helps to sufficiently alleviate pain. As health providers, we must always remember the axiom – physician, do no harm!

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Fig. 54.15 Container and NTI anterior guide-plane that fits over the maxillary central incisors allowing contact only with the mandibular central incisors creating disclusion or separation of the posterior teeth.

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Chapter 55

Arthroscopy and Arthroscopic Surgery

Anders Holmlund

Arthroscopy provides unique possibilities for simultaneous intra-articular diagnosis and surgical treatment. This chapter describes the method for diagnostic arthroscopy and gives examples of the different pathological changes that may be observed in the temporomandibular joint (TMJ) during arthroscopy. Several surgical procedures, such as lysis and lavage, disc suturing, synovectomy, and restriction procedures, are described and critically reviewed, as are the postoperative course and sequela.

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Development of temporomandibular joint arthroscopy

Modern arthroscopy started with the commercial introduction of the flexible fiber-light cable in the beginning of the 1970s. Another invention, the Hopkins rod-lens telescope, also introduced commercially in the 1970s, constituted a significant improvement in the optics. The first report on temporomandibular joint (TMJ) arthroscopy was published in the *Journal of Japanese Stomatology* in 1975 by Dr Masatoshi Ohnishi, who followed the strong Japanese tradition in the field of arthroscopy.¹ In 1980, Dr Ohnishi presented his experiences with TMJ arthroscopy in English.² Other reports followed, such as those of Dr Murakami in Japan, Dr Holmlund in Sweden, and Drs McCain, Kaminishi, and Sanders in the USA. TMJ arthroscopy now has a solid position as an important aid for both diagnosis and treatment in the field of TMJ surgery.

Anatomic considerations

The functional anatomy of the TMJ is beyond the scope of this chapter. However, there are some special considerations with regard to the arthroscopic procedure which should be mentioned. The joint is divided by the disc into an upper and a lower compartment. The volume of the upper compartment is about twice as large as that of the lower compartment. The kinetics of the TMJ is a combination of rotation and sliding movements where the disc-condyle complex slides down the eminence. In most disorders of the TMJ, this sliding movement is impaired. If translation is completely impaired, as in ankylosis, the joint space is almost totally closed, making safe puncture of the joint impossible. Fortunately, in most patients this is not so and the space is large enough to permit safe puncture of the joint. In the lateral and medial areas, the capsule and disc attachment become fused and attached to the lateral and medial poles of the condyle. Puncture of the upper compartment involves

only trocar penetration of the lateral capsule, while puncture of the lower compartment involves penetration of both the capsule and the disc ligament. Thus, puncture of the lower compartment always involves the slight risk of damaging the lateral disc attachment, which may, in turn, cause displacement of the disc medially. The TMJ capsule is thin in the anterior and anteromedial parts. This must be considered while distending the joint. If irrigation is performed with too much pressure, rupture of the anterior and medial capsule may occur, with subsequent extravasation of fluid into the surrounding tissues. The lateral capsule is better developed and is usually felt as resistant by the trocar when puncturing the joint.

Occasionally, the roof of the glenoid fossa is paper thin. In the author's experience with TMJ arthroscopy over a period of 25 years, accidental penetration into the middle cranial fossa should be almost impossible if the proper technique and instruments are used.

The TMJ is surrounded by important anatomic structures, i.e. the temporal vein and artery, the anterior wall of the acoustic meatus, and the tympanic membrane posteriorly, and the facial nerve inferiorly and anteriorly on the lateral side. The chorda tympani may run close to the medial capsule and the maxillary artery is located somewhat inferior to the joint on the medial side. Puncture of the TMJ must be planned with great care in order to avoid damaging these structures. However, it is relatively safe to insert the instruments from the lateral side between the temporal vessels posteriorly and the temporal branch of the facial nerve anteriorly.

Diagnostic arthroscopy

A careful case history and clinical examination are of great importance in TMJ diagnosis and arthroscopy since other supplementary techniques can never act as a substitute for a thorough clinical examination. Moreover, supplementary techniques (radiography, computed tomography (CT), magnetic resonance imaging (MRI), or arthroscopy) are all more invasive or costly. Such methods should therefore be regarded as important adjuncts to the clinical examination and can confirm or refute a tentative diagnosis. Whether arthroscopy should be chosen is more a question of what MRI or CT cannot achieve. One definite advantage of arthroscopy is that diagnosis and therapy can be performed simultaneously.

The indications are as follows:

- disc derangements;
- osteoarthritis;
- rheumatic joint disease;
- crystal-induced arthritis;
- synovial pseudotumors.

Two clinical variants of disc derangement exist: reciprocal clicking and chronic closed lock. In reciprocal

clicking, arthroscopic examination can sometimes confirm the clicking but it usually cannot because irrigation reduces friction. The arthroscope does not permit complete closing of the mouth and therefore any displacement of the disc cannot be determined. The arthroscopic anatomy is generally normal. A slight inflammation is usually present. Therefore, the value of diagnostic arthroscopy in patients with reciprocal clicking is limited to the assessment of the degree of synovitis.

In chronic closed lock, on the other hand, a degenerative process typically develops in the posterior part of the disc and the posterior disc attachment. Arthroscopically, there is a loss of the well defined boundary between the disc and the posterior disc attachment, with softening and hyperemia of the disc. In the closed position the disc remains in the anterior recess. Synovitis is present in most cases and in about 20% of cases the inflammation is more severe with presence of synovial hyperplasia.³ Arthrotic changes of the fibrocartilage and disc frequently occur.

Regarding osteoarthritis, the late stage of the disease correlates well with clinical signs (crepitation)⁴ and radiographic features (sclerosis, flattening, erosions).⁵ However, arthroscopy more accurately reveals the severity of cartilage and disc involvement and of the associated inflammation (Fig. 55.1).⁵ This information is important for choosing intra-articular pharmacotherapy, e.g. the use of steroids.

The TMJ may also be involved in rheumatic joint disease, e.g. rheumatoid arthritis, psoriatic arthritis, and ankylosing spondylitis. An early diagnosis of TMJ involvement is important. However, clinical signs and symptoms are non-specific and proposed radiographic features (erosions) are also found in joints with osteoarthritis and are demonstrated rather late in the disease process. Arthroscopy provides reliable information as to the degree of inflammation and cartilage involvement.^{6,7} In the chronic stage, adhesions seem to be a particularly important diagnostic marker.⁷

In rarer conditions, such as crystal-induced arthritis (gout, pseudogout), pigmented villonodular synovitis, and synovial chondromatosis, arthroscopy may be the only way to make a correct diagnosis. Gout and chondrocalcinosis show typical urate crystals and deposition of calcium salts respectively. In patients with pigmented villonodular synovitis, typical synovial hyperplasia with brown pigmentation is found. In patients with synovial chondromatosis, numerous loose cartilage bodies can be visualized almost blocking the joint space. These loose bodies are not always detectable with CT and MRI.⁸

Contraindications

Absolute contraindications include:

- bony ankylosis;
- advanced resorption of the glenoid fossa;

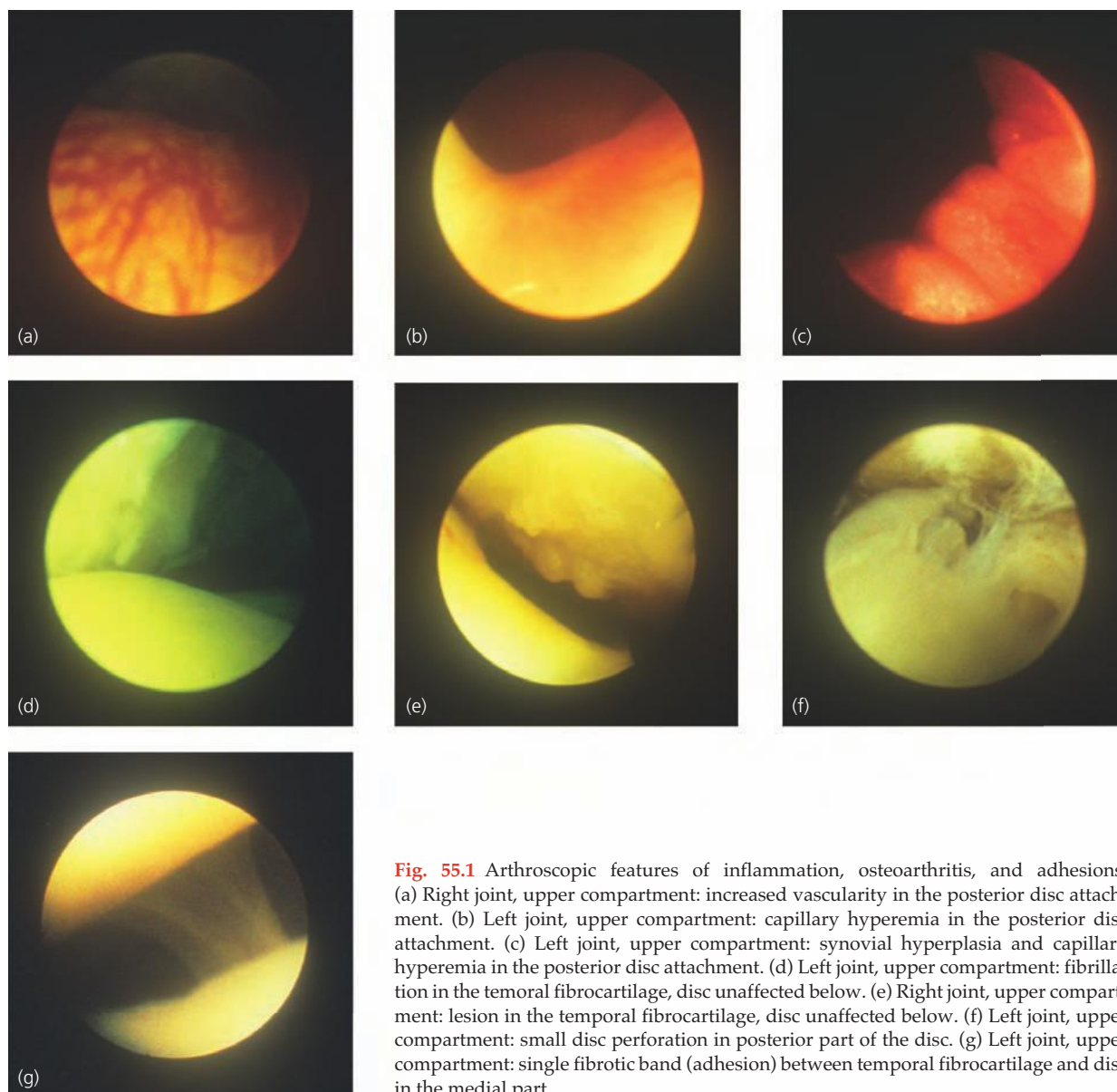


Fig. 55.1 Arthroscopic features of inflammation, osteoarthritis, and adhesions. (a) Right joint, upper compartment: increased vascularity in the posterior disc attachment. (b) Left joint, upper compartment: capillary hyperemia in the posterior disc attachment. (c) Left joint, upper compartment: synovial hyperplasia and capillary hyperemia in the posterior disc attachment. (d) Left joint, upper compartment: fibrillation in the temporal fibrocartilage, disc unaffected below. (e) Right joint, upper compartment: lesion in the temporal fibrocartilage, disc unaffected below. (f) Left joint, upper compartment: small disc perforation in posterior part of the disc. (g) Left joint, upper compartment: single fibrotic band (adhesion) between temporal fibrocartilage and disc in the medial part.

- infection in the joint area;
- malignant tumors.

Relative contraindications include:

- patients at increased risk for hemorrhage;
- patients at increased risk for infection;
- fibrous ankylosis.

Bony ankylosis is, for obvious reasons, a contraindication. In TMJs where the CT scan indicates advanced resorption of the glenoid fossa, diagnostic arthroscopy should be avoided because of the risk of accidental perforation into the middle cranial fossa.

In patients with an infection in the soft tissues surrounding the joint, diagnostic arthroscopy should be avoided, since there is a considerable risk of spreading an extra-articular infection into the joint. However, in the case of infectious arthritis, it may be desirable to perform arthroscopic puncture of the joint to establish drainage and irrigate the joint.

In cases of suspected malignant tumors, diagnostic arthroscopy should be avoided, since there is always a risk of spreading malignant cells with the irrigation fluid.

Patients at increased risk of bleeding should be carefully evaluated and prepared before arthroscopy in order to prevent this complication which may lead to hemarthrosis. Patients at increased risk of infection, e.g. immunodeficient patients, must also be carefully assessed especially as regards the need for prophylactic antibiotic therapy.

Arthroscopy equipment

Several publications have described the basic equipment for diagnostic TMJ arthroscopy.^{9,10} Fig. 55.2 shows a suitable set of instruments. Most telescopes have a diameter of about 2mm but ultrathin telescopes have recently been developed with a diameter of about 0.7mm. Such a reduction in diameter results



Fig. 55.2 Arthroscopic equipment. (a) Telescope, arthroscopic sheath, trocars, biopsy forceps, irrigation syringe, and light cable. (b) Camera and light source. (c) Arthroscopy in action.

in a loss of optical quality. There is also an increased risk that the instrument will break. However, more important than the diameter, is the quality of the optical system. A rod lens telescope has proved superior to other models. The telescope should have a direction of view of 30° . The field of vision is thereby much increased simply by rotating the instrument.

The arthroscopic sheath should fit properly on the telescope so that it just overlaps the telescope at the tip, thereby protecting the lens. The sharp and blunt trocar should fit the sheath and extend slightly over the sheath at the tip.

A 1.2mm disposable standard needle is suitable for outflow.

For tranguation purposes a twin sheath is used. This allows for switching the position of the working instruments and telescope.

A Xenon cold-light fountain provides a light with accurate color purity. The light cable that connects the telescope to the light source should be sufficiently long and flexible.

Instruments for arthroscopic surgery are:

- forceps, knives, and scissors;
- suction punch;

- mini-shavers;
- bipolar cautery;
- surgical lasers.

A mini-shaver or suction punch may be used to smooth surfaces of the disc and fibrocartilage or remove adhesions. Bipolar cautery has been used to coagulate tissue in cases of synovial bleeding or to cut the tissue.

During the last decade the surgical laser has become more popular. With the laser the wavelength of the transmitted light is most important. The energy of lasers with a wavelength in the far infrared spectrum, such as the carbon dioxide laser, is largely absorbed by water. The tissues in the joint have a high water content which would make this laser ideal, since most of the laser energy is absorbed by the superficial layers of tissue. The thermal damage zone is minimal, less than $70\mu\text{m}$. However, because of its long wavelength ($10.6\mu\text{m}$) it cannot be transmitted through a fiber-light cable without distortion. It is therefore not practical for arthroscopic surgery. The Neodymium YAG laser operates at a wavelength of $1.06\mu\text{m}$. Due to its short wavelength, the laser beam transmits easily through a flexible light cable and, as

a free beam, it also passes through water without being absorbed. The tissue penetration exceeds the tissue effect seen during surgery by 3–5 mm. The Neodymium YAG laser is excellent for coagulation. However, because the thermal damage zone is too large, it is not ideal for the TMJ. The Holmium YAG laser is a better alternative; it operates at a wavelength of 2.1 μm . The thermal damage zone is about 0.5 mm, making this laser ideal for the TMJ.

Arthroscopic procedure

Anesthesia

Routine diagnostic arthroscopy and minor arthroscopic surgery can be performed under local anesthesia. Arthroscopy under local anesthesia is more cost-effective than under general anesthesia. No assistant is required to manipulate the jaw and the patient can move the mandible normally.

Effective anesthesia of the joint area is achieved by blocking the auriculotemporal nerve posterior to the condyle and by infiltrating the subcutaneous tissue lateral to the joint (3–4 ml of lidocaine/epinephrine, 10 mg/ml).

The patient should be told that the anesthetic almost invariably affects the upper branches of the facial nerve and should be asked to remove ear-rings and contact lenses before arthroscopy. Sedation may be used if the patient so requires. In patients where advanced arthroscopic surgery is planned, general anesthesia is to be preferred.

Puncture

The arthroscopic examination should be performed with the patient in a supine position to reduce the risk of inducing a vasovagal reaction. The technique

for puncture has been described in several publications.^{9,10}

First, the upper compartment is distended with 2 ml of lidocaine which is injected slowly until resistance is felt. The additional injection of the anesthetic blocks any sensory input from branches of the posterior deep temporal and masseteric nerves supplying the anterior part of the TMJ. If no resistance is encountered after injection of 3 ml, the distension should always be stopped because it suggests the occurrence of leakage through the capsule into the surrounding tissue.

The so-called inferolateral approach gains good access to the posterior part of the upper compartment of the TMJ (Fig. 55.3). With this approach, access to the anterior recess is somewhat limited. However, from a diagnostic point of view, it is particularly important to examine the posterior part of the joint. This is also the best approach for puncturing the lower compartment. Alternative approaches to the upper compartment may be the endaural and anterolateral approaches (Fig. 55.3). The former may give a better view of the lateral part of the upper compartment; the latter provides better access to the anterior recess of the upper compartment. The anterolateral and endaural approaches are mainly used when arthroscopic surgery is intended.

It is questionable whether the lower compartment should be punctured at all. Even if successful, only the posterior non-functional parts of the disc and condyle are visualized and, moreover, it always involves at least some risk of damaging the lateral disc attachment. Routine puncture of the lower compartment should therefore be avoided.

Correct placement of the arthroscope should be confirmed through the telescope. An outflow portal is then created (inferolateral approach) about 5 mm anterior to and slightly below the arthroscopic sheath. Continuous irrigation is performed using isotonic

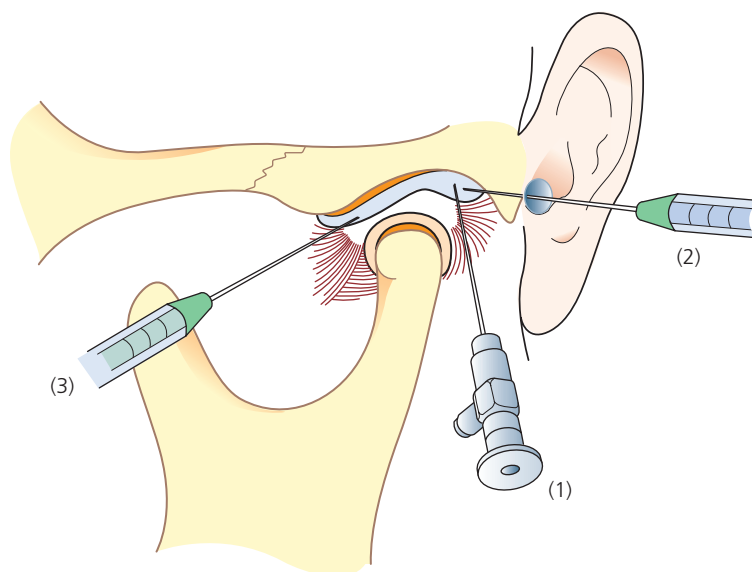


Fig. 55.3 Puncture directions for the TMJ. (1) Inferolateral. (2) Endaural. (3) Anterolateral.

saline solution. For more prolonged arthroscopic surgery, Ringer's solution may be a better alternative because it has been found to protect the chondrocytes and maintain the synthesis of proteoglycans.

Arthroscopic examination

The arthroscopic examination begins with identification of the arthroscopic landmarks – i.e. the boundary between the disc and the posterior disc attachment, the medial capsule, and the inferior part of the eminence. The arthroscope should always be moved slowly and gently between the various landmarks. Rotation of the arthroscope increases the field of vision considerably. The outflow should always be checked because if the needle becomes obstructed, considerable extravasation may occur in a short time. The irrigation pressure should be kept constant and low. In a small joint, such as the TMJ, this is best performed with a syringe by the assisting nurse. A total irrigation volume of 30–50 ml is usually sufficient for diagnostic arthroscopy.

Another problem may be the magnification of the structures by the telescope. The amount of magnification depends on the distance between the object viewed and the arthroscope. A ratio of 1:1 is obtained at a distance of 20–25 mm, which means that magnification is always present in a small joint such as the TMJ.

In the upper compartment the examiner should first identify the posterior disc attachment and the posterior part of the disc. The posterior disc attachment is the predominant location for inflammation and this part is therefore thoroughly examined for signs of inflammation, such as increased vascularity, capillary hyperemia, villi, or synovial hyperplasia.

The temporal cartilage and disc surface should then be evaluated. Signs of osteoarthritis, such as fibrillation, lesions in the fibrocartilage, and denuded subchondral bone, may be mainly located on the posterior slope of the eminence. Disc perforations are usually detected in the lateral, central, and posterior parts.

In some cases, the lower compartment can be examined through a large disc perforation from the upper compartment.

Arthroscopic surgery

In the field of orthopedic surgery, arthroscopic procedures have increasingly replaced open joint surgery. The duration of the postoperative period and the frequency of complications have thereby been reduced.

However, arthroscopic surgery of the TMJ is more difficult. The anatomic position limits the possibilities for surgery and the small size and the anatomy of the joint often give limited access for the instruments. The introduction of new instruments with fine dimensions and the surgical laser has increased the possi-

bilities for arthroscopic surgery of the TMJ. However, one should keep in mind that the increased access to the joint provided by small instruments does not necessarily imply that the procedure is effective. Many new instruments are ineffective as regards working capacity. An increased risk of broken instruments must also be considered. The complexity of the procedure can also make it rather time-consuming and some advanced arthroscopic procedures may take even longer than open surgery.

Technical aspects

Minor surgical procedures, such as lysis and lavage or partial synovectomy, can be performed under local anesthesia with or without sedation. More complicated procedures are best performed under general anesthesia. Draping is the same as for open TMJ surgery. The assistant can mobilize the mandible with the thumb through the drape behind the incisors or by using a towel clamp placed at the angle of the mandible.

The surgical procedures are best performed under direct visual control either with a double cannula or with triangulation. The double cannula needs one portal but the diameter of the instrument is rather big, about 4 mm, limiting the access to the anterior part of the joint. Triangulation is therefore frequently used instead (Fig. 55.4). It requires two portals but gives better access. Triangulation demands great skill by the surgeon, however, and a well trained assistant is needed. Training on cadavers and experience of open TMJ surgery are prerequisites.

The following surgical procedures can currently be performed:

- biopsy;
- lavage;
- lysis;
- disc repositioning;
- synovectomy;
- debridement and abrasion;
- restriction;
- intra-articular pharmacotherapy.

Synovial biopsy

Although arthroscopy is very accurate for diagnosing synovitis, recent studies indicate that a synovial biopsy may also be of value in the diagnosis.⁷ Current indications for synovial biopsy include chronic inflammatory conditions or benign tumors.

In the knee, biopsy under direct arthroscopic control has been shown to be the best because of the wide variation within the same joint. It is therefore necessary to use a second portal for the biopsy forceps. In small joints, such as the TMJ, a so-called semi-blind technique seems sufficient.^{11,12} First the area for performing the biopsy must be selected, then the arthroscopic sheath should be fixed in that position

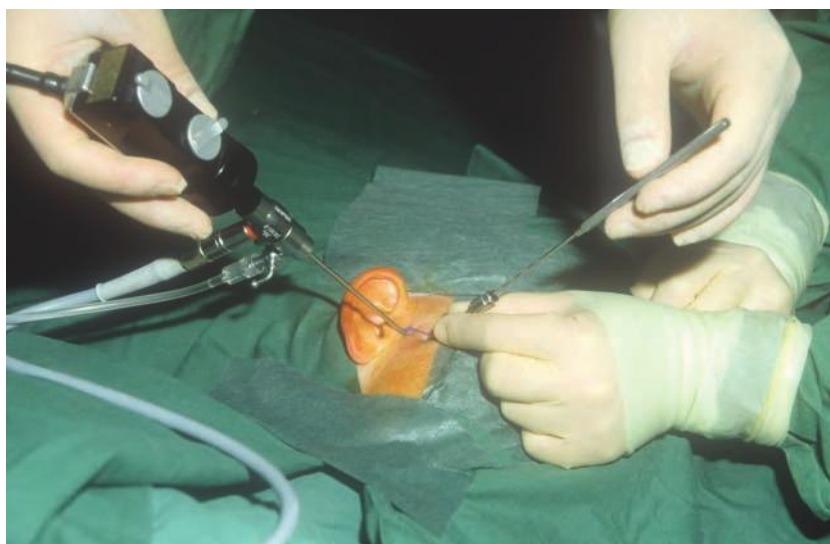


Fig. 55.4 Triangulation using inferolateral and anterolateral approaches.

and the telescope removed. The biopsy forceps are then inserted, the sheath slightly withdrawn, the forceps opened, and the biopsy is then performed. The correct location should then be confirmed through the telescope. Recent studies in the TMJ have shown that the semi-blind technique has the same high accuracy as biopsy under direct visual control.¹² The type of biopsy forceps used seems to be more important. The forceps must be cup-shaped with sharp edges, in order to cut the tissue precisely, without compressing the specimen.

Lavage (arthrocentesis)

In patients with osteoarthritis of the knee, irrigation of the joint with isotonic saline solution or Ringer's solution has been found effective for giving pain relief and improving function. In osteoarthritis and synovial joint disease, there is an accumulation of microcartilagenous debris and inflammatory products. A thorough lavage will remove these irritating products, thus relieving symptoms and resulting in more favorable conditions for repair.

The procedure is relatively easy to perform and requires very few instruments. In its simplest form, a 1.2mm needle is used for outflow (Fig. 55.5). The irrigation is performed with a syringe and a 0.6mm needle placed in the posterior recess of the upper compartment. A total of 100 ml of isotonic saline solution is slowly injected, and the outflow is constantly checked. The patient may move their jaw slightly during the irrigation.

TMJ lavage is a quick, inexpensive and non-invasive method with hardly any complications. The outcome has been good in several follow-up studies.¹³⁻¹⁸ The disadvantage with simple lavage or arthrocentesis is that the irrigation is not effective for breaking up adhesions or mobilizing the disc which is the main problem in, for example, chronic closed lock and osteoarthritis.

Lysis

Adhesions frequently occur in patients with chronic closed lock, osteoarthritis, and rheumatic disease. These are thought to result from repeated inflammatory episodes and require lysis or removal to improve function. The lysis can be performed blindly, using a blunt trocar with sweeping movements from anterior to posterior (Fig. 55.6). An alternative is to use the Holmium YAG laser under direct arthroscopic control, as described by Koslin.¹⁹

Combined lysis and lavage after arthroscopic diagnosis provides a better result than lavage only according to recent studies.^{15,20-22}

Disc repositioning

Disc repositioning has been performed extensively in open TMJ surgery (see Chapter 56).

A few studies have reported on arthroscopic disc repositioning.^{23,24} First an anterior release is performed with a laser, the disc is then pushed posteriorly and held in position while a suture is passed through the posterior part of the disc and then tied to either the anterior wall of the meatus or the subcutaneous tissue posterior to the joint. This technique requires considerable skill and is time-consuming. The passing of the suture through the avascular portion of the disc is questionable. A better alternative may be to create scar tissue with the laser in the posterior disc attachment and thereby stabilize the disc in the correct position.

Good short-term results have been reported with disc suturing,²³ but there is a need for long-term outcome studies using MRI.

Synovectomy

It seems an attractive principle to intervene early in the disease process in patients with rheumatic joint

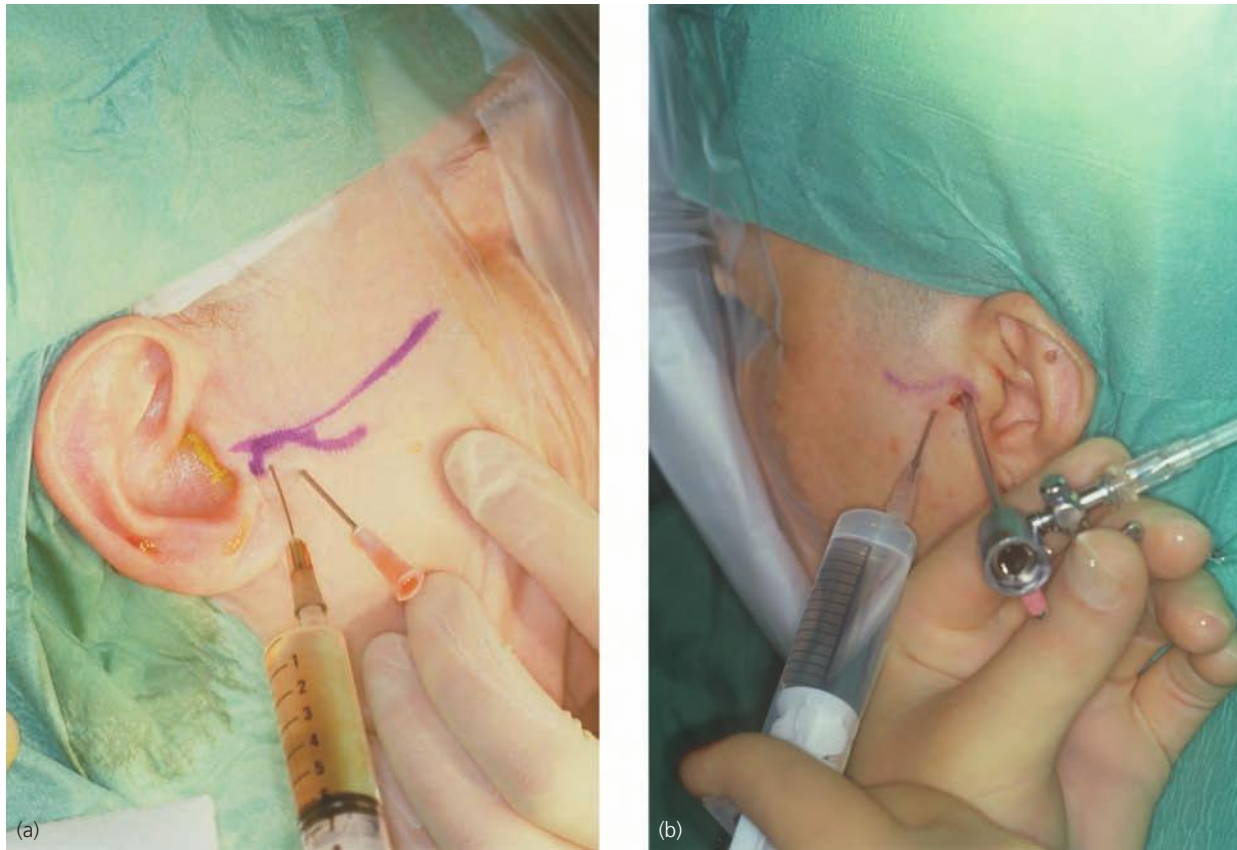


Fig. 55.5 Irrigation (arthrocentesis) of the TMJ. (a) Irrigation using two needles. (b) During arthroscopy using the arthroscopic sheath for outflow providing effective irrigation.

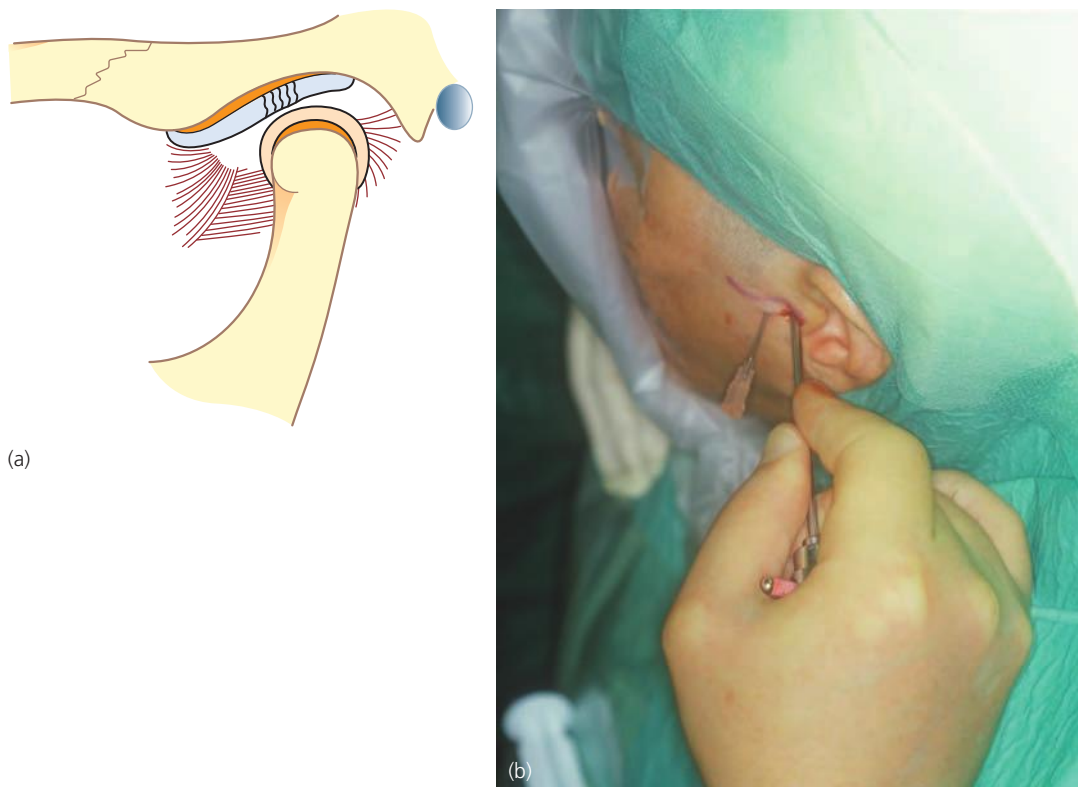


Fig. 55.6 Lysis of adhesions. (a) Simplified drawing of adhesion between the temporal fibrocartilage and the disc. (b) Semi-blind release of adhesions in the joint.

disease and to remove areas of focal granulation in the synovial lining in order to prevent spread to the fibrocartilage and disc. In the knee, controversy exists regarding the long-term effect of arthroscopic synovectomy. However, it has been shown to reduce pain and improve function for up to 5 years.²⁵

Synovectomy should be performed under direct arthroscopic control using a Holmium YAG laser that vaporizes the tissue, which is then removed by irrigation (Fig. 55.7). At present, there are few outcome studies of synovectomy of the TMJ.

Debridement and abrasion

Joints with osteoarthritis and arthritides frequently display irregularities of the fibrocartilage and disc. These irregularities may interfere with the smooth functioning of the joint and, if so, they must be removed. They can be removed with a mini-shaver. However, the use of a shaver in a small joint like the TMJ requires considerable surgical skill and removal should always be done under direct arthroscopic control in order to avoid complications such as damage to vessels and nerves close to the joint capsule. The Holmium YAG laser is a better alternative for the debridement because of improved access to the more distant parts of the joint.

Abrasion arthroplasty has previously been discussed in the orthopedic literature. However, much controversy exists regarding its efficacy. The procedure for the TMJ was described by Quinn in a preliminary study²⁶ but requires further evaluation.

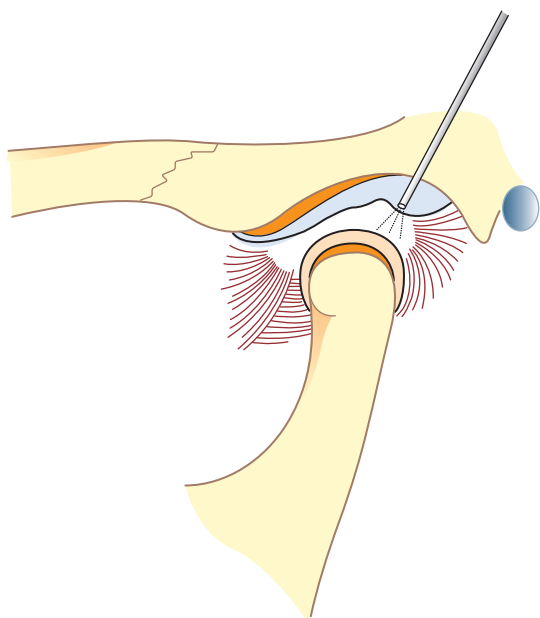


Fig. 55.7 Simplified drawing of laser synovectomy in the TMJ. In patients with hypermobility, the laser may also be used in a coagulating mode to create scar tissue, thereby limiting translation of the disc-condyle complex.

Restriction

Temporomandibular joint hypermobility or recurrent luxation may also be treated arthroscopically. This method has focused on producing scar tissue in the the posterior disc attachment, thereby limiting forward translation of the disc-condyle complex. Under arthroscopic control, a sclerosing solution is injected subsynovially in the posterior disc attachment.²⁷ Another possibility is to use cautery or laser.²⁸ The laser is used in a coagulating mode to create scar tissue in the posterior disc attachment (Fig. 55.7). A third method is to pass a suture through the posterior part of the disc and then tie it to the anterior wall of the meatus.²⁹ Short-term outcome studies have shown good results with all three methods. However, as yet no long-term evaluation has been published.

Intra-articular pharmacotherapy

Intra-articular injections with corticosteroids and hyaluronan have been used in combination with TMJ arthroscopy. Both of these drugs have alleviated pain and improved mandibular function in patients with chronic arthritis.³⁰ Moreover, hyaluronan seems to have a good effect on clicking joints.³¹

At the end of the arthroscopic procedure corticosteroids are usually injected. However, in the TMJ this always entails extravasation of the steroid to some extent through the portals created, which, in turn, may cause atrophy of the subcutaneous tissue. It is therefore better to inject easily dissolvable steroids into small joints such as the TMJ and preferably to wait 1 week after arthroscopy before doing this.

Hyaluronan should also be injected strictly intra-articularly, because an extra-articular injection causes severe pain.

Postoperative care

Arthroscopy under local anesthesia has many advantages compared with that performed under general anesthesia and the patient is ready to go home after only 1 hour's rest in the clinic. Any anesthetic effect on branches of the facial nerve has usually disappeared by then.

The patient is given a simple training program. A physiotherapist is contacted if needed. Control of any parafunctional habits is also maintained postoperatively. Information about hygiene and food is then given to the patient. In cases of postoperative pain, a mild analgesic is prescribed.

Prophylactic antibiotics may be given to patients at high risk for infections (immunodeficiency, heart valve prosthesis, etc.). In normal cases, there is no need for prophylactic antibiotics.

If the arthroscopic procedure is performed under general anesthesia the postoperative care follows the protocol of that for open TMJ surgery. Usually, the

procedure can be performed as day-case surgery limiting the cost for the patient and society.

Complications

Intraoperative and postoperative complications are seldom associated with minor arthroscopic procedures. This accords with data in the orthopedic literature regarding knee joint arthroscopy.³² However, it should be emphasized that a complication in the TMJ area can be disastrous for the patient. A few such complications have been reported, concerning damage to the middle ear and the facial nerve.^{33,34} In arthroscopic surgery, particularly the more complicated procedures, the risk of complications is obviously greater. An adequate knowledge of the external and internal anatomy of the TMJ is a prerequisite for clinical use of the arthroscope. Preoperative radiographic examination should include CT. Training on cadavers before clinical use is recommended. Experience of open TMJ surgery facilitates the management of a complication.

Possible complications include:

- vascular injury;
- extravasation;
- scuffing of cartilage;
- broken instruments;
- otologic complications;
- intracranial damage;
- infection;
- nerve injury.

Vascular injury

Vascular injury is rare. Carter and Testa noted hemorrhagic complications in 1.8% of the cases in a survey of 2225 TMJ arthroscopies.³⁵ In about 80%, the temporal vessels were involved.

If well designed trocars are used, these rarely cause bleeding during puncture of the TMJ. More often it is the outflow needle that causes the bleeding, especially if the needle is difficult to position. The bleeding is usually venous in origin and easily controlled, using a tamponade. In persistent cases the cutaneous incision can be extended slightly and the source of the bleeding is sought, and ligated or controlled with cautery. Intra-articular bleeding may occur in more advanced arthroscopic surgery. The protocol suggested by McCain seems appropriate.³⁶ All instruments are first removed and the condyle is then moved to the compartment where bleeding has occurred and is kept there for about 5 minutes. The instruments are then reinserted to irrigate and debride clots that may have formed. If this procedure does not work, the joint must be opened and bleeding controlled.

Intra-articular hemorrhage is common in trauma to the mandible.³⁷ However, the accumulated blood

seems to be resorbed within a couple of weeks.³⁷ Thus, the risk of developing an arthrofibrosis because of intra-articular hemorrhage is low.

Vascular malformations, such as arteriovenous fistulas, resulting from TMJ arthroscopy are unlikely, but they have been reported.^{38,39}

Extravasation

Excessive extravasation of irrigation fluid into the surrounding tissue sometimes occurs. In prolonged arthroscopic surgery, the risk becomes greater and cases of extensive edema in the upper airway with subsequent postobstructive pulmonary edema have been reported.⁴⁰ If the surgeon or the nurse always checks the outflow, this is unlikely to occur.

Scuffing

The cartilage may be scuffed by the trocar during the puncture. The scuffed fibrocartilage should not be mistaken for arthrotic changes. This lesion can be avoided if the trocar is directed towards the crest of the glenoid fossa until it touches the bone. The sharp trocar is then exchanged for a blunt one which is directed slightly inferiorly to slip into the upper compartment.

Broken instruments

Instruments that break during arthroscopic manipulation result in an unpleasant complication. Such complications have been reported by McCain⁴¹ and Tarro.⁴² They show that the surgeon must be trained in open TMJ surgery, since this may be the only way to retrieve a broken instrument. For many reasons, a broken instrument should not be left in place. One reason is that it may be potentially dangerous, another reason is that it may be blamed for a poor result.

This complication can be avoided by using a correct technique and impeccable instruments.

Otologic complications

A few otologic complications have been noted in association with TMJ arthroscopy. Van Sickels *et al.* reported a case with perforation of the tympanic membrane and partial dislocation of the malleus.³³ Appelbaum *et al.* described three patients who largely lost their hearing.³⁴ Serious complications, such as those mentioned above, can be avoided only by proper training in TMJ anatomy and practice on cadavers before the clinical use of TMJ arthroscopy.

The influence of TMJ arthroscopy on normal hearing has been evaluated by audiometric measurements pre- and postoperatively.^{43,44} Both studies indicate that the risk of adverse effects on hearing is extremely low.

Intracranial damage

Damage to intracranial structures during TMJ arthroscopy must not occur. Nevertheless, such damage has been reported.⁴⁵ The roof of the glenoid fossa can be extremely thin but it is the author's experience from autopsy studies that it takes considerable force to penetrate the roof of the glenoid fossa, even with a sharp trocar. In patients whose cartilage and bone show marked destruction (e.g. advanced rheumatoid arthritis) great caution must be exercised and routine radiographic examination supplemented with CT.

Infection

In an extensive review of 3146 patients (4831 joints) by McCain in 1992,⁴⁶ where complications associated with TMJ arthroscopy were also reported, the frequency of infections was low (15 cases; 0.3%). This brings into question the common use of prophylactic antibiotics. The author has performed more than 1500 TMJ arthroscopies without using any antibiotics and no patient has developed an infection. Considering recent reports on increase in the frequency of bacterial resistance to antibiotics, it seems unnecessary to use antibiotic prophylaxis in routine cases.

Nerve injury

Injuries to nerves are almost never associated with TMJ arthroscopy. In more than 4800 TMJ arthroscopies, McCain *et al.* reported only three cases (0.06%) with permanent nerve damage.⁴⁶ Thus the frequency is about the same as that reported for knee joint arthroscopy. The auriculotemporal and facial nerves are most often involved. Excessive extravasation of irrigation fluid may exceptionally have a transient effect on the infraorbital, inferior alveolar, and lingual nerves.

Concluding remarks

Similar to developments in the field of orthopedic surgery, TMJ arthroscopy has become an important method for diagnosis and treatment. Its accuracy in diagnosing TMJ diseases is high and simultaneous biopsy can be performed to improve diagnosis. Complications are minor and infrequent. The various new surgical methods show good short-term results. A recent retrospective study of 488 arthroscopies showed that only 20% needed additional surgeries and a randomized clinical trial has shown comparable good results when comparing arthroscopic lysis and lavage with discectomy.²¹ The application of surgical lasers to the TMJ has extended the possibilities for arthroscopic surgery. Some arthroscopic procedures, such as the frequently performed lysis and lavage, can be done under local anesthesia in an outpatient clinic. It is cheaper than open TMJ surgery since it

means a shorter hospitalization and little, if any, sick leave for the patient. Finally, arthroscopy provides unique possibilities for research to elucidate the complicated etiopathogenesis of TMJ disorders.

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Chapter 56

Temporomandibular Joint Surgery

Anders Holmlund

The temporomandibular joint (TMJ) has many similarities with other synovial joints and thus the joint diseases that occur in other body joints are also found in the TMJ. However, the TMJ also shows specific features regarding anatomy and function which have to be considered when surgical treatment is intended.

This chapter will discuss etiology and pathogenesis for common TMJ diseases and propose diagnostic criteria and indications for surgery based on recent research. The techniques for established surgical procedures are described as well as common complications. The procedures are critically reviewed regarding outcome and suggestions are given on how to further develop TMJ surgery.

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Development of temporomandibular joint surgery

Many of the temporomandibular joint (TMJ) diseases that we see today were also recognized centuries ago. However, it took until the late 19th and early 20th centuries before it was possible to perform TMJ surgery. Many of the procedures that we perform today are in fact improvements and modifications of surgical techniques first performed by these pioneers. Examples of this are Annandale who performed the first TMJ disc repositioning in 1887¹ and Lanz who performed the first discectomy in 1909.² TMJ ankylosis was operated on in the 1850s and surgical reports by Rizzoli and Verneuil³ described gap-interposition of gutta-percha and temporalis muscle.

The most common TMJ problem leading to TMJ surgery has been, and still is, TMJ clicking and locking. Pringle in 1918⁴ and Wakely in 1929⁵ described disc displacement and contributed to the understanding of disc displacement. A more detailed description was made later by Karl Bohman,⁶ who also presented results from more than 50 patients.

Schwartz⁷ in the 1950s and Laskin⁸ in the 1960s pointed out the importance of muscle disorders and psychosocial mechanisms as part of TMJ symptoms. In the 1970s, there was a renewed interest in the role of the disc after publications by Farrar and McCarty.⁹ Later, Eriksson and Westesson,¹⁰ Wilkes,¹¹ Hall,¹² and Holmlund¹³ have further contributed to the development of TMJ surgery. It is beyond the aim of this chapter to go deeper into the history of TMJ surgery,

but it cannot be emphasized enough how valuable it is for the contemporary surgeon to also study the past.

Classification of surgical TMJ disease

TMJ disease can be classified as follows:

- Mechanical disorders:
 - reciprocal clicking – disc displacement;
 - mandibular dislocation.
- Degenerative joint disease:
 - chronic closed lock – disc displacement;
 - osteoarthritis.
- Chronic polyarthritis with TMJ involvement.
- TMJ trauma:
 - contusion;
 - intracapsular fracture;
 - condylar neck fracture.
- TMJ abnormalities:
 - congenital;
 - acquired.
- TMJ tumors:
 - benign;
 - malignant.
- TMJ infection.
- Extra-articular connective tissue diseases.

The widely used term "temporomandibular dysfunction" (TMD) is not used in this classification. It is the authors' opinion that this term should be avoided when a surgical classification is intended as it makes no separation between muscle conditions and true joint afflictions. This does not mean that muscle conditions have no importance. It simply means that these conditions are not to be treated surgically except for a few rare cases. Moreover, the term "internal derangement" is avoided in this classification. Instead, the two clinical variants of disc displacement (reciprocal clicking and chronic closed lock) are classified under "mechanical disorders" and "degenerative joint disease" respectively. A rationale for this will be given in the following paragraphs.

Reciprocal clicking and chronic closed lock

Biological background

Based on recent research the following statements can be made:

- Reciprocal clicking is common and affects at least 7% of the adult population.¹⁴
- The reciprocal click correlates with a displacing disc and a callus formation in the posterior part of the disc.¹⁵ Except for the callus formation the disc has a normal structure. The fibrocartilage is unaf-

ected and thus no osteoarthritic changes are found.¹⁵

- In joints with reciprocal clicking a mild T-cell dominated inflammation with interleukin (IL)-1 α and β expression is found.¹⁶

Chronic closed lock is characterized by a permanently displaced disc. It may be preceded by reciprocal clicking but in many patients it is not.¹⁵ It is also the author's impression that there are no differences with regard to tissue appearance between patients with or without previous reciprocal clicking.¹⁵ The disc shows clear degeneration.¹⁵ The fibrocartilage shows varying degrees of osteoarthritis.¹⁵ A chronic inflammation is present in the synovium and both T-cells and macrophages are present.¹⁶ Moreover, the cytokine expression is more complex. In addition to IL-1, tumour necrosis factor (TNF) α , transforming growth factor (TGF) β and, in particular, interferon- γ are also expressed.¹⁶ As a result of the prolonged chronic inflammation, adhesions between the disc and the temporal fibrocartilage may form, further limiting the translation of the disc-condyle complex.

The relationship between reciprocal clicking and chronic closed lock has been described in a recent publication by the author.¹⁷

Surgical implications

Only a small number of patients with reciprocal clicking develop intermittent locking or a socially disturbing loud clicking that should be considered for surgery. The treatment has to consider the displaced disc but more importantly the callus formation in the posterior part of the disc. This can be done by removal of the callus, by repositioning of the disc, by altering the condylar position, or by increasing the joint space.¹⁷

In chronic closed lock, there is an ongoing degenerative process in the disc which seems to spread from posterior to anterior.¹⁵ Moreover, a (probably) secondary chronic synovial inflammation develops, and as a result of this adhesions form between the disc and the temporal fibrocartilage. The disc is permanently displaced anteriomedially which also impairs the translation of the condyle.

In about 40% of the patients with chronic closed lock, the pain may subside and function improve.¹⁸ The reason for this is not clear but it seems to correlate with development of a disc perforation which releases the tension and blockage caused by the displaced disc.¹⁹ In these patients, the development of osteoarthritis has also been found radiographically.¹⁹ Symptomatic treatment for a certain period may thus be used, but the problem is that it may take up to 2 years before a spontaneous remission is seen. Moreover, as only about 50% of the patients will improve spontaneously, it is not possible to predict if the patient will fall into this group or will continue to have symptoms.

It should be understood that in chronic closed lock (as well as many other joint diseases), there is no cure for the disease. Until the disease has been explained, we can, at best only prevent further development of the disease, improve mandibular function, and reduce pain. This may be achieved by manipulating the blocked disc and by breaking up the adhesions. Once translation of the disc–condyle is improved, lubrication and nutrition will also improve, providing the basis for tissue healing. The removal of irritating debris and inflammatory mediators may also contribute to improvement (see Chapter 55).

Surgical approaches to the TMJ

Preparation of the surgical site

Preparation and draping should expose the entire ear and also the lateral canthus of the eye. The hair may be shaved in order to keep the draping in place and to expose the temporal area. A small sponge is placed into the external auditory canal. An anesthetic with vasoconstrictor may be used to control subcutaneous bleeding after incision. However, the injection should only be kept superficial as anesthesia in the deeper tissue layers may hamper the use of a nerve stimulator. The skin incision may be marked with dye but there is usually a natural skin fold to follow.

Incision

The standard approach to the TMJ is the preauricular approach (Fig. 56.1a). It uses the preauricular skin fold and, if needed, may be extended superiorly and slightly anteriorly. Normally, when an intra-articular procedure is intended there is no need for a superior or anterior extension of the incision but in cases where access to the area anterior to the eminence is needed such an extension of the incision is practical.

Some surgeons prefer to bring the incision behind the tragus (endastral incision) in young individuals, advocating that this will reduce the visible scar. It is true that the preauricular incision in a few young patients creates a scar that is slightly erythematous, but this will blanch within a couple of months. However, a tragal extension can create more of a problem from an esthetic point of view.

The retroauricular incision has been suggested, mostly in older publications. This approach comprises a curved incision behind the ear and thereby the incision is completely hidden. The access to the anterior part of the joint is somewhat limited. The major drawback of this approach is that it involves transection of the external auditory canal. Problems such as stenosis of the ear canal and infection have been reported.

In summary, the preauricular approach with or without temporal extension provides access for almost all TMJ procedures, including surgery for trauma, ankylosis, and tumors.

Dissection of the joint

The dissection commences in the superior part of the incision (Fig 56.1b–d). The temporalis fascia is exposed and any vessels running posteriorly are ligated or cauterized. The fascia is then followed inferiorly down to the superior part of the zygomatic arch. The temporal artery and vein may be ligated if they interfere with the exposure of the joint. The auriculotemporal nerve usually runs together with the artery and vein and may be dissected free and retracted anteriorly. It is often rather thin and may therefore be either torn or accidentally transected during the dissection. As a consequence there will be a decreased sensitivity in the skin over the TMJ area. The sensitivity will reappear at least partly in most patients within a year and even if a hyposensitivity is permanent, this seems not to disturb the patient. The facial nerve is rarely a problem. In a few cases the temporal branch may be encountered close to the joint capsule anteriorly. If the dissection is kept close to the tragus this is avoided. However, there is always a risk of traction on the nerve from the anterior retractor. It will occur in about 6% of cases. This temporary palsy will subside within 3 months.

The dissection is then continued with an antero-superior incision through the superficial layer of the temporalis fascia which is then extended inferiorly. The scissors are used to identify the zygomatic arch. An almost constant finding is a fairly large vein running over the zygomatic arch in a superior direction. This vein is a useful landmark for the anatomical location of the glenoid fossa. After exposure of the zygomatic arch, the lateral capsule is easily found inferiorly. The structures anterior and inferior to the capsule are protected with retractors. A horizontal incision in the superior part of the capsule provides access to the upper compartment. There is usually a recess between the lateral capsule and the lateral disc attachment but sometimes the capsule has to be transected vertically to gain access to the inferior part of the lateral disc attachment. The retractor is now placed between the capsule and the disc and the lateral disc attachment is cut while lifting the structure somewhat. This protects the condylar cartilage from being traumatized. Condylar retractors are now placed anterior and posterior to the condyle. In order to open up the joint space, a 1.1 mm Kirschner wire is now drilled into the condyle and zygomatic arch and a retractor is positioned and the joint space widened. If the joint space is not opened up it may be difficult to visualize the disc, especially in the medial part. If a discotomy is performed it may result in incomplete removal of the medial part of the disc.

Closure of the incision is made in the usual manner beginning with the lateral capsule and the temporal fascia and then a few sutures in the deeper subcutaneous tissue and finally a running 4/0 subcuticular suture to close the skin.

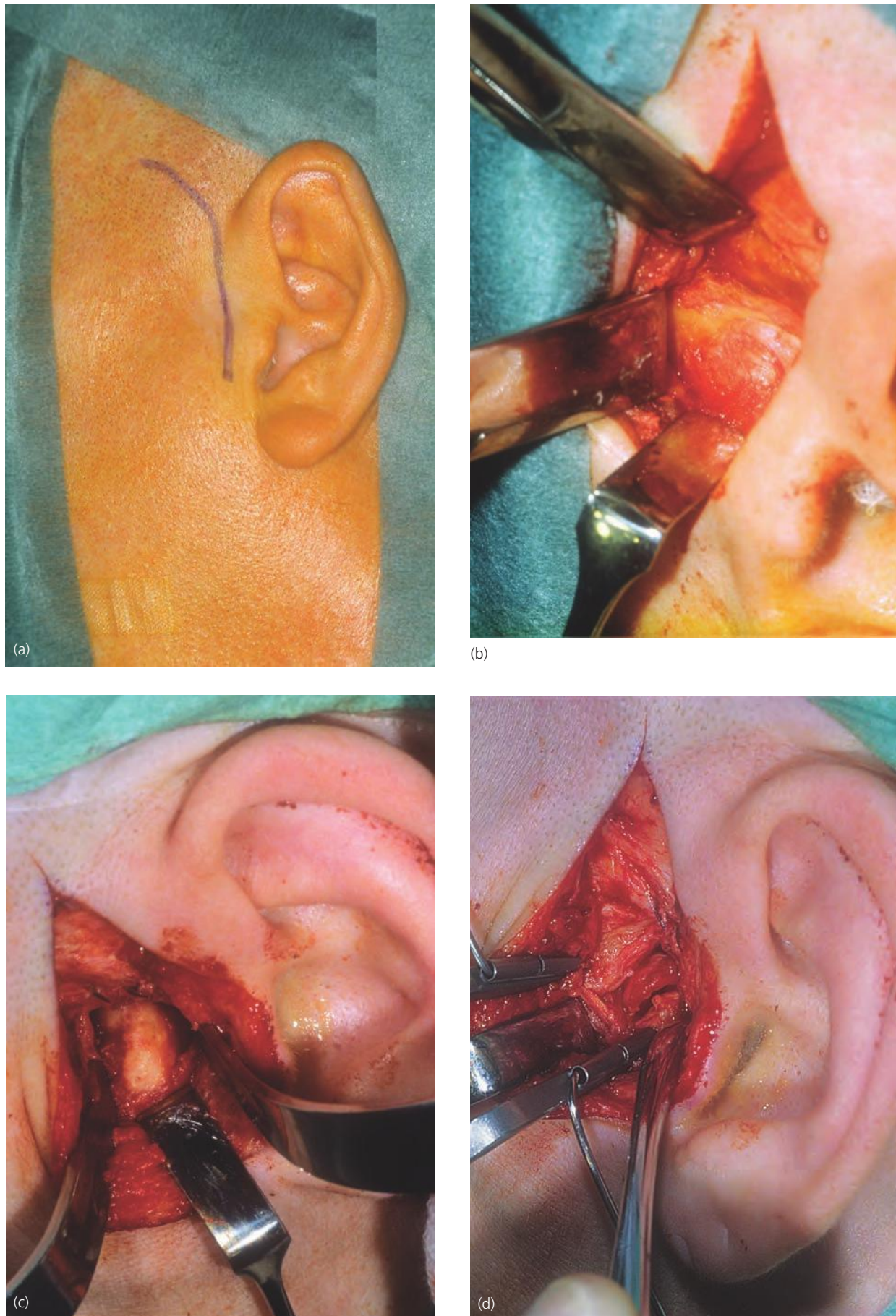


Fig. 56.1 Surgical dissection of the TMJ. (a) Preauricular incision line. (b) Zygomatic arch and lateral capsule exposed. (c) Lateral capsule opened and condylar retractors placed. (d) Kirschner wires drilled into the eminence and condyle and a Wilkes self-retaining retractor (W. Lorenz Surgical, Jacksonville, FL) applied to increase access to the joint.

Reciprocal clicking

Clinical diagnosis

The patient usually complains of loud clicking and catching of the joint. The clinical examination reveals a reciprocal click (Fig. 56.2). This sign is strongly correlated to a displacing disc and callus formation in the posterior part of the disc. Thus, a diagnosis based on clinical examination is sufficient and there is no need for radiographic examination unless surgery is intended. In most patients the reciprocal clicking also occurs in protrusion and laterotrusion to the opposite side. The patient experiences pain only when the joint locks temporarily. There is usually no pain over the joint area upon palpation. Mandibular movements are usually normal and radiographic examination shows normal features in the majority of patients.

Surgical treatment

Disc repositioning

Disc repositioning means that the anteriorly displaced disc is surgically repositioned back to its original position on the condyle (Fig. 56.3).

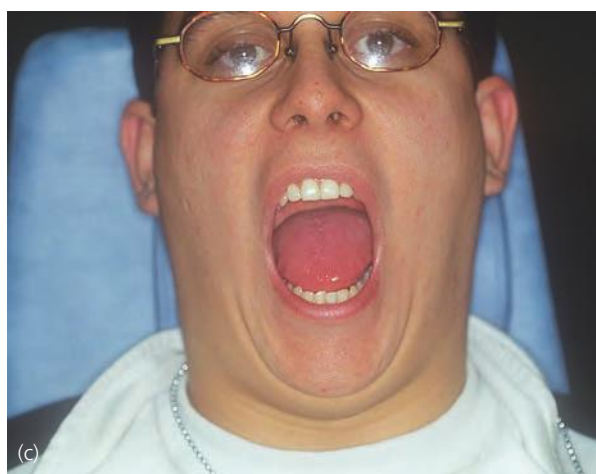
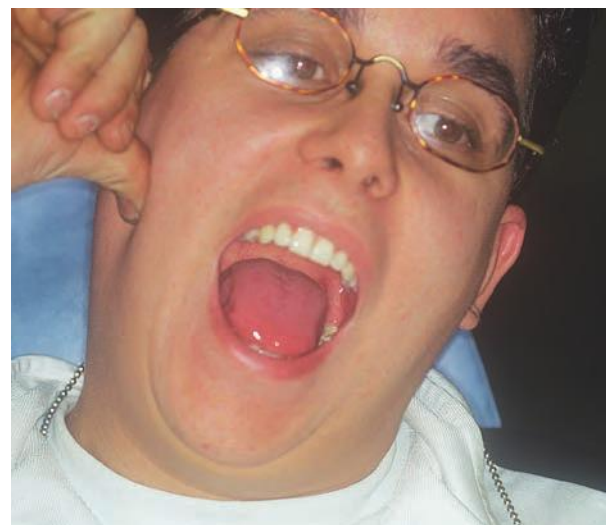
Biological considerations

The central part of the disc is thin and avascular and has the properties to take up the load during function. It also facilitates the sliding of the disc–condyle complex down the posterior slope of the eminence.

In TMJs with reciprocal clicking the disc is displaced anteriorly and there is a callus formation in the posterior part of the disc. When surgery is planned it is therefore important to address the altered position and also the fact that the disc is deformed. If the callus is not removed, or at least reduced, the disc will displace again. A problem is that the callus is usually most prominent in the posteriomedial part where access is limited and sometimes even a large part of the disc itself has been displaced medially.

Outcome studies

Disc repositioning has been performed extensively and there is a number of case series reported. The overall results have been good.²⁰ There is no convincing evidence (e.g. magnetic resonance imaging (MRI) studies) showing that a repositioned disc remains so after surgery. In general, there is a lack of randomized clinical trials and the outcome criteria or end-points are poorly defined. Therefore, the procedure needs to be validated in randomized clinical trials



(b)

Fig. 56.2 Patient with reciprocal clicking and catching of the right TMJ. (a) Maximum opening before the click. (b) Manipulation of the TMJ with the thumb. (c) Maximum opening after click.

where it is compared with a proper reference procedure.

Complications

There are very few complications reported with the methods described above. A temporary palsy of the temporal branch of the facial nerve is seen in about 6% of cases. A slight but clinically insignificant hyposensitivity in the skin anterior to the ear may be found in most patients. Deep infection is very infrequent. Superficial infection adjacent to a remaining suture may be seen occasionally. Whether a prophylactic antibiotic should be given or not remains unanswered. Until such a study has been performed, the author routinely prescribes clindamycin 600mg IV given 1 hour before surgery. Serious scar formation is extremely rare. Malocclusion, demanding slight occlusal adjustment, may occur in a few patients.

Modified condylotomy

The hypothesis of this method is that if the position of the condyle is altered the load on the posterior disc attachment will be reduced, which in turn will facilitate a spontaneous repositioning of the disc on the

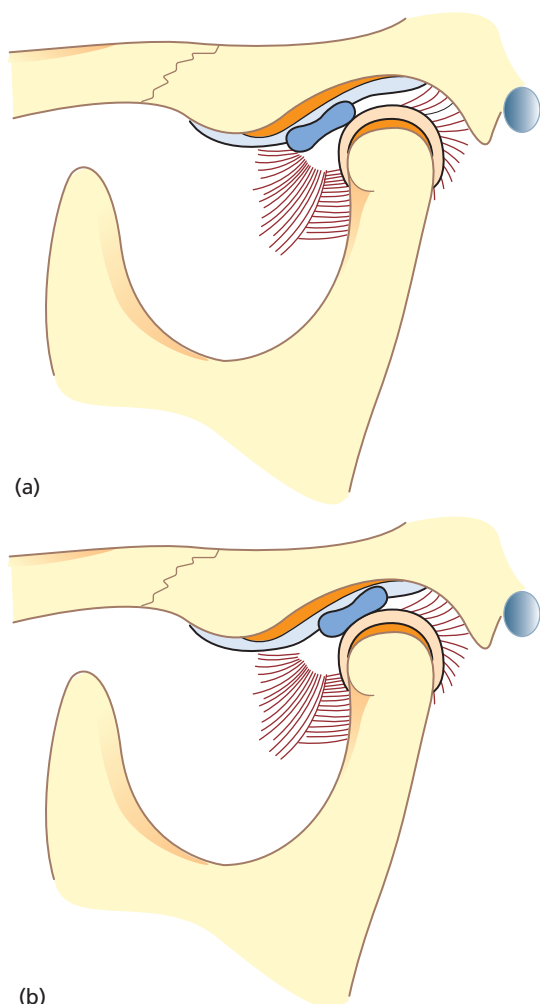


Fig. 56.3 Simplified drawing showing surgical repositioning of the disc. (a) Displaced disc. (b) Disc after surgical repositioning.

condyle (Fig. 56.4). In the knee, the same method has been used in the past by performing an osteotomy of the tibia, thereby changing the load on the meniscus.

The technique comprises an intraoral vertical ramus osteotomy via an incision of the buccal fold posteriorly. The posterior segment containing the condyle is allowed to drop a few millimeters but the segment is not fixed to the distal portion of the mandible. The wound is sutured and intermaxillary fixation with elastics is then applied and held for 2 weeks.

Outcome studies

There have been a few long-term follow-up studies done but no randomized clinical trials comparing modified condylotomy with, for example, discectomy.²¹ These studies also show considerable drop-out of patients during the follow-up period, decreasing the value of the studies. In conclusion, modified condylotomy may also be a useful method for treatment of reciprocal clicking and catching of the TMJ but it has to be validated in comparative randomized studies comparing it with discectomy.

Complications

Complications are usually minor. Wound infection may occur but is usually not a problem. The condyle may displace in some cases and create an open bite on the operated side.

Discectomy

Biological considerations

Discectomy treats both the displaced disc and the problematic callus in the disc and does it effectively (Fig. 56.5). Thus, the loud click disappears as well as the catching of the joint. However, removal of the disc also has some negative consequences. In most cases a slight reduction of the translation of the condyle is seen at follow-up, probably because of a slight scar tissue formation in the joint. Crepitation is common as are radiographic signs of remodeling or

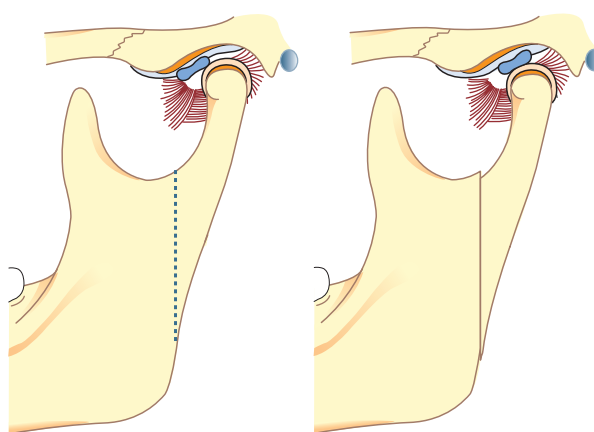


Fig. 56.4 Simplified drawing showing modified condylotomy of the TMJ.

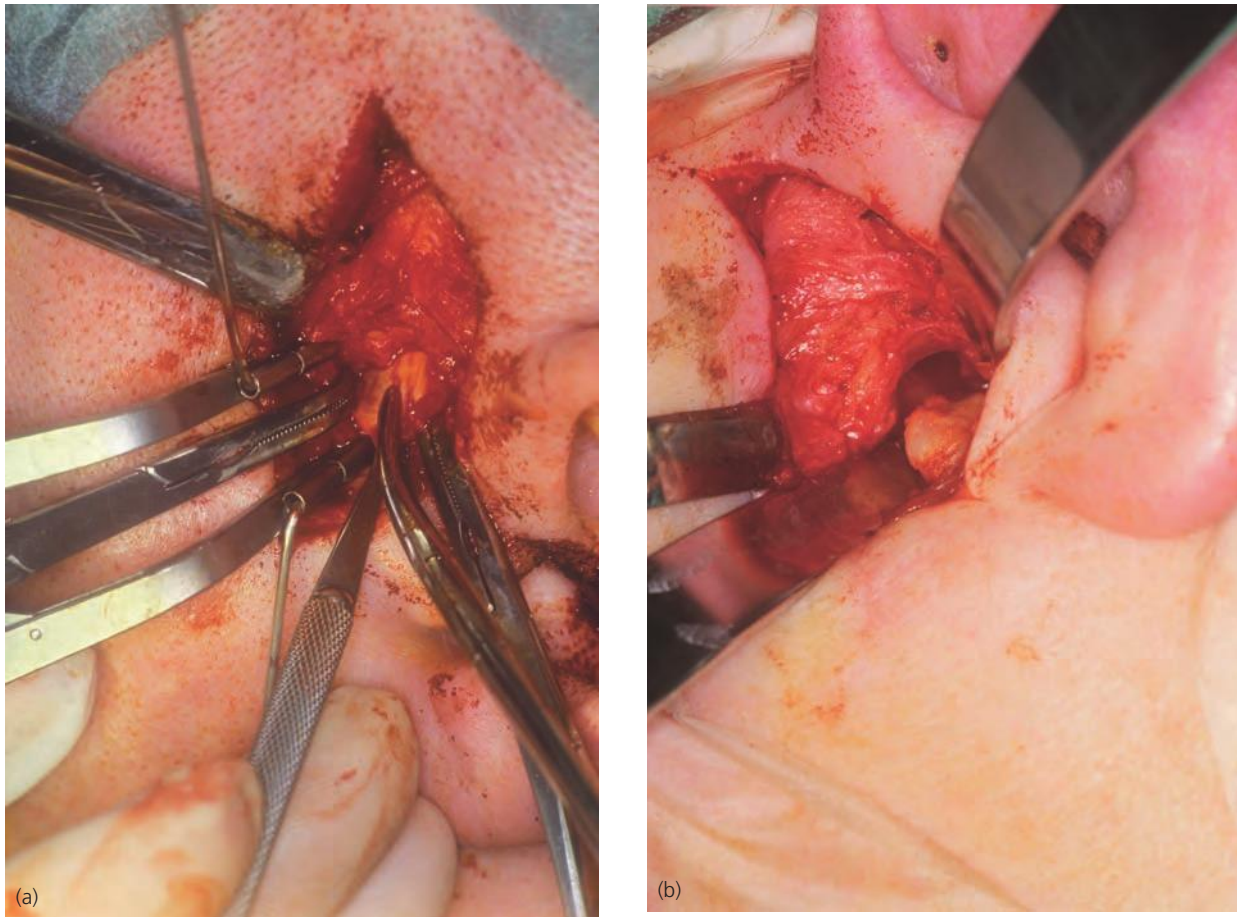


Fig. 56.5 (a) Excision of the disc. (b) Exposure of an interfering osteophyte on the condyle.

arthrosis of the articular surfaces.²² However, it seems that this arthrotic-like appearance does not correlate with impaired function or pain.

Outcome studies

Discectomy without replacement has been thoroughly evaluated in well designed long-term follow-up studies.^{13,22–24} In prospective 5-year follow-up studies at four different oral and maxillofacial centers in Sweden the success rates were high, about 85%, and were also stable over time. Moreover, in a long-term retrospective study (mean follow-up 29 years) the results were very good. No patient had TMJ pain and the maximum opening was more than 39 mm. Similar to what was found in the prospective studies, crepitation and radiographic signs of arthrosis were common.

In conclusion, discectomy is thoroughly evaluated and should be regarded as the first choice for surgical treatment in patients with TMJ reciprocal clicking. Discectomy should also be the reference procedure in randomized clinical trials when new surgical methods are to be evaluated.

Complications

The complications are the same as for disc repositioning surgery.

Disc replacement

In the same context as discectomy, disc replacement must also be discussed. Several methods using alloplastic materials as well as autogenous tissue have been used. Alloplastic materials used in the past include Silastic and Proplast, which are no longer used because of complications including fragmentation and giant cell reactions. The implants were held in place with sutures or screws drilled into the zygomatic arch. Among the autogenous procedures suggested for use today the temporalis muscle flap seems to have the greatest potential.

Outcome studies

There are no randomized clinical studies comparing disc replacement with discectomy without replacement and the reported success rates for disc replacement surgery have not exceeded those of discectomy without replacement.

Complications

Compared with discectomy without replacement there is an increased risk of complications (inflammatory reactions and deep infection) when alloplastic materials are used. Donor site morbidity may occur when autogenous tissues are used.

Mandibular dislocation

Epidemiological studies have revealed an increased incidence of general joint hypermobility in children, in women compared with men, and in some racial groups (Eskimos and Chinese).²⁵

Clinically it is difficult to decide when the TMJ should be regarded as hypermobile. The reason for this is the great variation in translation capacity between normal subjects. In TMJ patients, based on TMJ arthrographic findings, a frequency of 29% has been reported. Symptomatic hypermobility of the TMJ has been recently investigated in children, young adults, and adults.²⁶ Girls/females were predominant. The overall prevalence was about 18% in 15-year-old children, 19% in 22-year-old young adults, and about 15% in 43-year-old adults. Risk factors for the development of symptomatic hypermobility were gender (females), race other than Caucasian, masticatory muscle pain, and increasing maximum mouth opening.

TMJ hypermobility may be subclassified into:

- acute dislocation;
- recurrent dislocation;
- long-standing dislocation.

Acute dislocation has been known since the time of Hippocrates or even earlier. Although rather dramatic in its appearance it is quite easily managed. Most acute dislocations occur only once. Recurrent dislocations are rare, but the situation is often very disturbing for the patient with frequent visits to emergency units. Another category of recurrent luxations are the patients with so-called habitual luxations. These patients can dislocate and reposition the mandible at will and therefore rarely pose a clinical problem. Yet another category are the very few patients with long-standing dislocations. These patients are usually old and chronically ill with a variety of neurological disorders making them unable to communicate their

problem. The author has had five such patients through the years where the mandibular dislocation was not diagnosed by the nursing personnel.²⁷

Clinical diagnosis

Clinical diagnosis is usually not a problem. In bilateral dislocations there is a frontal open bite, in unilateral dislocations there is a deviation of the mandible to the opposite side and a lateral open bite on the same side (Fig. 56.6). The condyle is not seated in the glenoid fossa and thus the area anterior to the tragus appears "empty". Radiographic examination confirms the dislocated mandibular condyles.

Differential diagnosis involves traumatic contusion of the TMJ and possibly also unilateral growth of the condyle. It is important to have a proper medical history to rule out these conditions. For the inexperienced surgeon a radiographic examination may be helpful.

Surgical treatment

Acute dislocation

Acute dislocation is best managed with manual repositioning. In the author's experience, manual repositioning is easy and quick provided that a proper technique is used. The patient should be reassured and then the repositioning should be done as quickly as possible without any unnecessary delay (Fig. 56.7). In order to reposition the mandible it is important to press downwards on the mandibular molars and avoid pushing the mandible backwards. If some time has elapsed since the dislocation occurred, either local anesthesia over the joint or sedation/analgesic may be given. Only when the dislocation has persisted for days may it be necessary to perform the repositioning under general anesthesia.

Usually, there is no need for any bandage. In cases where there is a tendency for recurrent luxation a

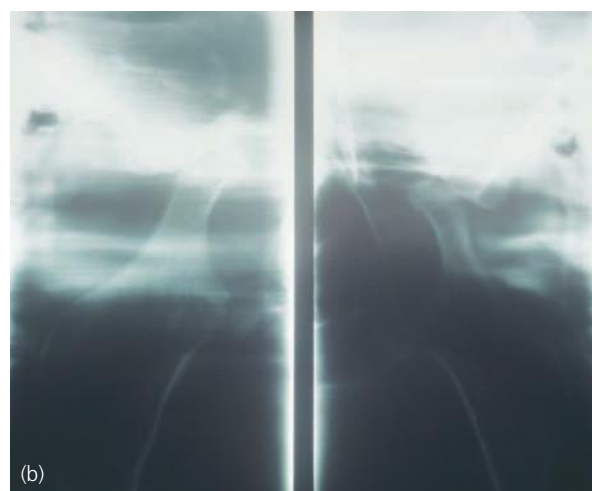


Fig. 56.6 Patient with recurrent dislocation of the mandible. (a) Clinical appearance. (b) Radiographic examination confirming the dislocated condyles.

loose intermaxillary fixation may be used for a week or so.

Recurrent dislocation

Several methods for management of recurrent dislocation have been proposed through the years, such as blocking procedures, anchoring procedures, methods inducing scar tissue, myotomy, eminectomy, and discectomy. Both open surgery and arthroscopic procedures have been described in the literature.

Myotomy was first described by Bohman in 1949 (Fig. 56.8a).²⁸ The method comprises sectioning of the muscle attachment anterior to the condyle. However, reattachment of the muscle seems to occur and Laskin therefore suggested the use of a Silastic sheet anterior to the condyle to prevent reattachment of the muscle.²⁹ In 1988 Sindet-Pedersen reported on intraoral myotomy of the pterygoid muscle where scar tissue was also induced anterior to the condyle.³⁰

Eminectomy was first described by Myrhaug in 1951 (Fig. 56.8b).³¹ The rationale is to facilitate spon-

taneous repositioning instead of blocking the excessive translation. Thus, the eminence is reduced as far as possible from the lateral side. The backward translation of the disc-condyle complex is facilitated but it is the author's experience that ingrowth of the lateral capsule also contributes by making the joint more rigid. An arthroscopic method for eminectomy has also been reported recently.³²

A blocking procedure that gained popularity in the 1980s was the Le Clerc procedure (Fig. 56.8c).³³ An osteotomy is performed in the zygomatic arch anterior to the eminence and the arch is then down-fractured and placed under the eminence, blocking disc-condyle translation anterior to the eminence.

Discectomy has also been suggested, for example by Wakely.³⁴ This method addresses the problem that hypermobility and mandibular dislocation often also have an associated disc displacement with clicking. Discectomy seems to both reduce the translation of the condyle (probably because some scar tissue is formed) and eliminate the disc-related catching of the joint.



Fig. 56.7 Patient with acute mandibular dislocation. (a) Before repositioning. (b) Manual repositioning. (c) After repositioning.

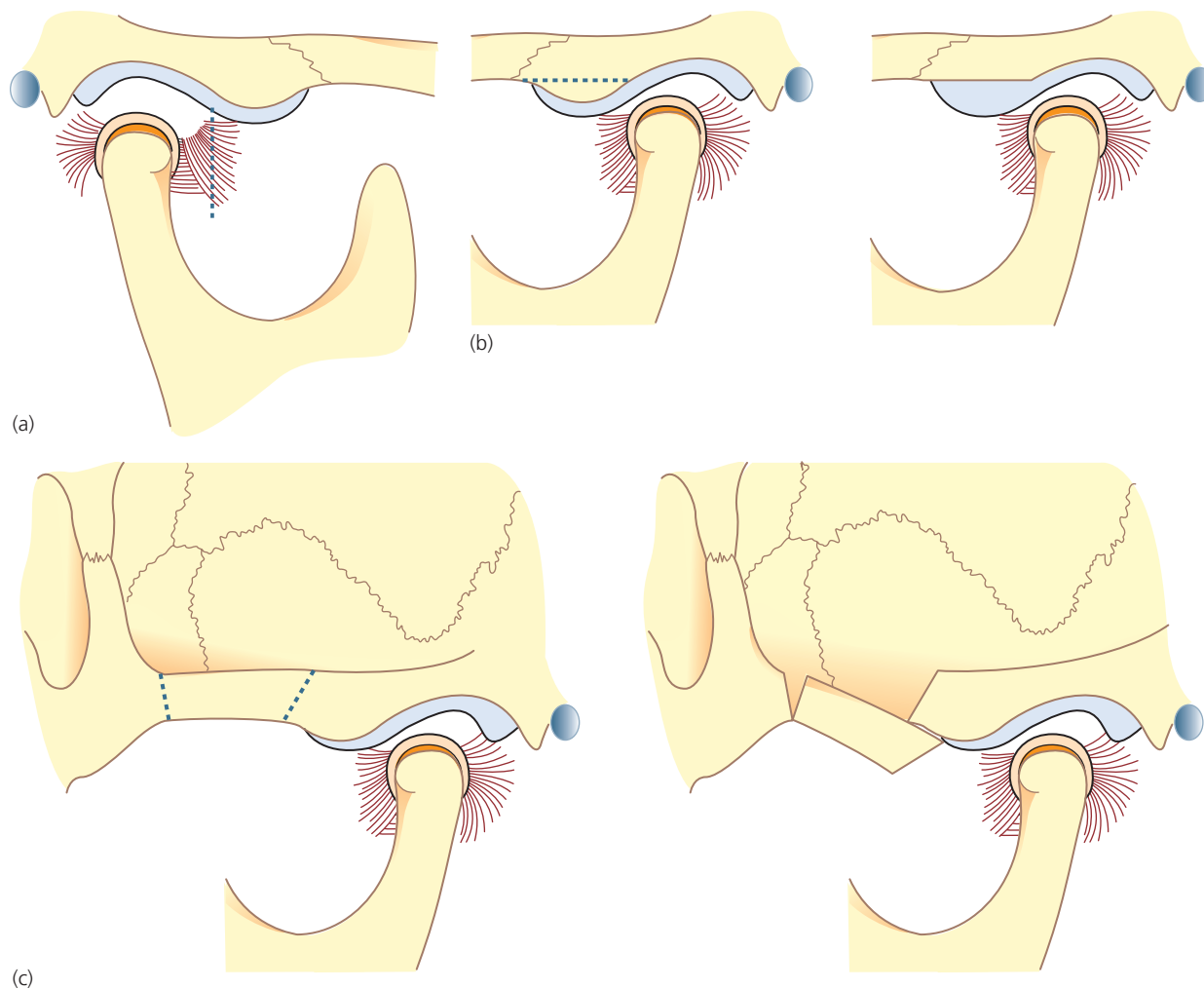


Fig. 56.8 Surgical methods for treatment of recurrent mandibular dislocation. (a) Myotomy of the lateral pterygoid muscle. (b) Eminectomy. (c) Le Clerc procedure.

Outcome studies

In general, there is a lack of randomized clinical trials. It is problematic that even single follow-up studies comprise small numbers. Considering these limitations eminectomy and discectomy seem to be the methods of choice. Arthroscopic methods have to be further evaluated.

Complications

Regarding myotomy and eminectomy there is an increased risk of injury to the temporal branch of the facial nerve as the dissection has to be extended anterior to the eminence. For discectomy, the risk of complications is very low, as previously mentioned.

Chronic closed lock and osteoarthritis

Chronic closed lock and osteoarthritis are described under the same heading as they frequently occur together.

In its early phase, chronic closed lock may show no lesions in the fibrocartilage but sooner or later they will appear. In a recent study, all patients with chronic

closed lock showed osteoarthritic changes in the TMJ operated on.¹⁵ Chronic closed lock may or may not be preceded by reciprocal clicking.¹⁷ Chronic closed lock usually has a rapid onset, a so-called acute closed lock. The reason for this may be a tear of the posterior disc attachment with a resulting anterior displacement of the disc.

Osteoarthritis (also called degenerative joint disease) is thought to be a primarily degenerative disease of the cartilage. Synovial inflammation may occur secondary to the primary disease process. Cartilage debris and inflammatory mediators are thought to provoke the synovitis. Osteoarthritis may also occur in a generalized form. The criterium for this diagnosis is that more than three joints (the spine excluded) should be affected. The TMJ may also be affected.

Clinical diagnosis

Chronic closed lock usually shows the following clinical features (Fig. 56.9):

- pain in the TMJ upon mandibular movements;
- laterotrusion to the opposite side is reduced and painful while laterotrusion to the same side is not;

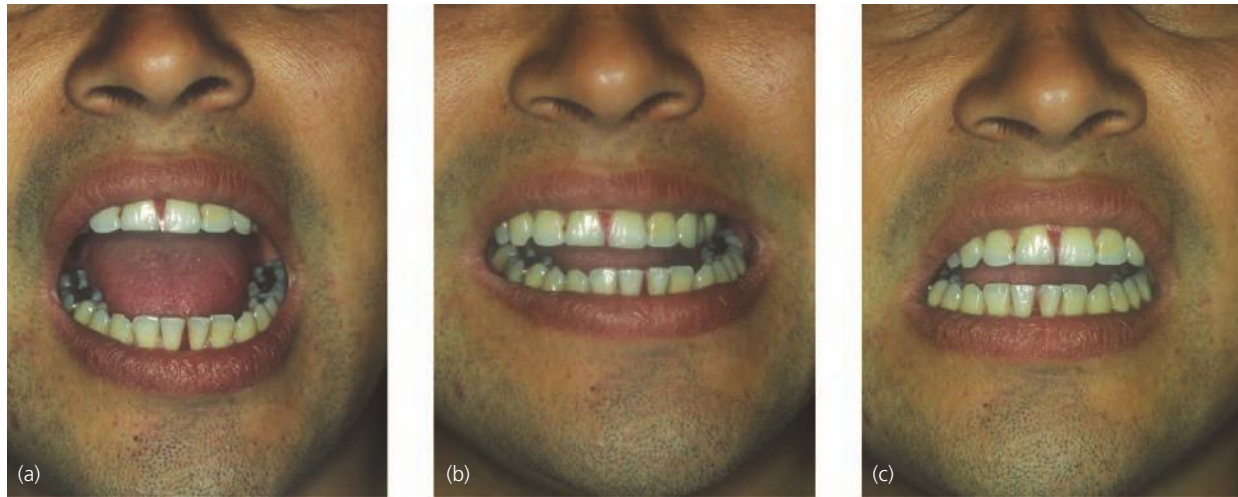


Fig. 56.9 Patient with chronic closed lock of the left TMJ. Red wax indicates the dental midline. (a) Maximum opening reduced and deviation of the mandible to the affected side. (b) Laterotrusion to the affected side is normal. (c) Laterotrusion to the opposite side is reduced.

- maximum opening and sometimes also protrusion deviates towards the affected side;
- maximum opening and protrusion are reduced;
- the affected TMJ is tender to palpation laterally and sometimes also posteriorly.

Normally, there are few muscle symptoms. If present, these are localized to the jaw muscles on the affected side. If parafunction is also present, muscle symptoms may be more prominent.

Radiographic examination may or may not show arthrotic changes of the subchondral bone (erosion, sclerosis, flattening, reduced joint space). MRI findings are an anteriorly (sometimes medially) displaced disc and some fluid in the joint indicating inflammation.

Osteoarthritis is clinically diagnosed if there is crepitation in the joint. Crepitation has been found to correlate well with manifest osteoarthritis and disc perforation. However, early phase osteoarthritis can only be diagnosed arthroscopically. Clinical signs and symptoms may be similar to those of chronic closed lock but some patients have quite good mobility of the joint. In these patients the complaints are mainly joint pain when opening and chewing. In some patients, the inflammation may be more pronounced and sometimes an interfering osteophyte may occur (Fig. 56.10).

Arthroscopy will reveal advanced destruction of the fibrocartilage, chronic inflammation, and adhesions. Radiographic examination shows arthrotic changes in the subchondral bone and reduced joint space. MRI may add to the diagnostic information by demonstrating a displaced and deformed disc.

Surgical treatment

In chronic closed lock, the disc is permanently displaced, resulting in a load on the vascularized and innervated posterior disc attachment. The translation will be impaired by the displaced disc as well as

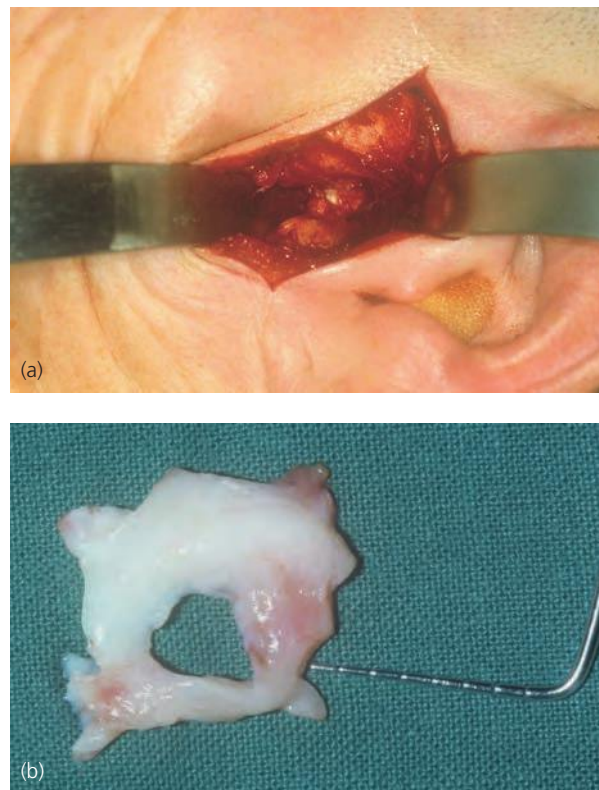


Fig. 56.10 Patient with osteoarthritis. (a) Interfering osteophyte on the condyle. (b) Excised disc with a large perforation.

adhesions resulting from the chronic synovitis. The reduced translation, in turn, impairs the vascular supply to the posterior disc attachment and, if this is prolonged, the nutrition and lubrication of the joint will be affected, aggravating the degenerative process.

The TMJ has a marked capacity for adaptation and repair. Thus, the TMJ may develop a spontaneous remission similar to that seen in other joints. This has also been shown in a study by Kurita *et al.*¹⁸ who found that about 40% of the patients with chronic closed lock obtained a spontaneous remission within

2.5 years after only symptomatic treatment with non-steroidal anti-inflammatory drug (NSAID) medication. However, this and previous studies have not been able to predict which patients will follow this advantageous route. Moreover, it is the author's experience that very few patients, although informed of this fact, are willing to wait such a long time for symptom relief.

Until more is known about the disease mechanisms for chronic closed lock and osteoarthritis, we can at best prevent further development of the disease, improve mandibular function, and reduce the pain.

In patients with chronic closed lock, results in a recent randomized clinical study comparing arthroscopic lysis and lavage with discectomy indicate that the first choice of treatment should be the former.³⁵ The results regarding clinical outcome were comparable and both methods had a high success rate. However arthroscopy is less invasive, can be performed under local anesthesia, and is thus also less expensive both for the patient and society. For more information about arthroscopic lysis and lavage, see Chapter 55.

Acute closed lock may be treated in the same manner as a contusion (see TMJ trauma). If the acute closed lock is very recent (within 24 hours) local anesthesia of the joint and gentle manual manipulation may unlock the joint. In the author's experience, this method does not work if the lock has existed more than 2 days, and prolonged manipulation can instead cause more trauma to the joint.

Discectomy

Discectomy has been described previously (see under Reciprocal clicking).

Outcome

Discectomy without replacement has shown good results in several well designed long-term follow-up studies.^{13,22–24,35} It is therefore the method of choice if arthroscopic lysis and lavage has failed.

Complications

The complications have been listed previously under Reciprocal clicking (disc repositioning).

Modified condylotomy

The method has been described under Reciprocal clicking. The same hypothesis is suggested, i.e. that a decreased load on the disc will facilitate adaptation and repair.

Outcome studies

Follow-up studies have been performed showing good outcome.³⁶ The limitations of these studies are high levels of drop-out of patients. The method has yet to be validated in a randomized study comparing it with discectomy.

Complications

The complications are those described under Reciprocal clicking (modified condylotomy).

Osteoarthritis (degenerative joint disease)

As previously mentioned, osteoarthritis frequently runs in conjunction with chronic closed lock and shows mainly the same symptoms except that crepitation is also frequently present.

Outcome studies

There are no specific assessment studies regarding TMJ osteoarthritis. As osteoarthritis and chronic closed lock frequently occur together the studies done for chronic closed lock may also be applicable to osteoarthritis. Thus arthroscopy is the first treatment of choice. Discectomy may be used if arthroscopy fails or if an interfering osteophyte or remodeling is seen on the computed tomography (CT) scan (see Fig. 56.10).

In patients where the TMJ osteoarthritis is part of a generalized osteoarthritis it seems that the success rate is lower.³⁷ In a small number of patients with osteoarthritis the degenerative process seems to induce pronounced adhesions with a high recurrence rate. In these patients an arthroplasty may be indicated.

Complications

The complications are essentially the same as for surgical treatment of reciprocal clicking and chronic closed lock.

Chronic polyarthritis with TMJ involvement

Chronic polyarthritis may involve the TMJ. The most common form is rheumatoid arthritis (RA) and juvenile rheumatoid arthritis (JRA), followed by psoriatic arthritis and ankylosing spondylitis. The prevalence of RA is about 2% in the western world. Women are twice as often affected as men. Reported incidence for TMJ involvement varies from 2–86%, reflecting the fact that definite clinical and radiographic criteria for RA of the TMJ are missing. For psoriatic arthritis and ankylosing spondylitis, there are no reliable figures regarding TMJ involvement. Arthroscopic studies have shown an increased frequency of adhesions and more pronounced synovitis in patients with RA of the TMJ compared to patients with chronic closed lock of the TMJ. Synovial biopsies from TMJs with RA involvement also show more pronounced inflammation.³⁸ The cytokine pattern also seems different from that of osteoarthritis.³⁹

Clinical diagnosis

Chronic polyarthritis usually first appears in joints other than the TMJ and therefore the diagnosis has already been established when the patient comes to the dentist or the oral and maxillofacial surgeon.

However, occasionally the first joint to evince symptoms may be the TMJ.

Acute RA usually demonstrates a swelling over the TMJ and the serology markers are usually raised considerably. In these cases several milliliters of synovial fluid can be aspirated from the joint. Arthroscopy shows typical hyperplasia and hyperemia of the synovium.³⁸ In a small group of patients

with rheumatic disease a frontal open bite and mandibular retrusion will develop (Fig. 56.11). In chronic RA the clinical features may be the same as for chronic closed lock and osteoarthritis, i.e. reduced mandibular movements, joint pain, and crepitation.

Surgical treatment

Surgical treatment comprises intra-articular injection, arthroscopic surgery, discectomy, arthroplasty, and orthognathic surgery. It is the author's experience that the vast majority of patients with chronic polyarthritis and TMJ involvement will benefit from arthroscopic lysis and lavage with or without a ste-



Fig. 56.11 Patients with chronic polyarthritis and TMJ involvement. (a) Patient with ankylosis following TMJ polyarthritis. (b) Another patient with a frontal open bite. (c) Micrognathia of the mandible in another patient.

roid injection. However, in a small group, open bite, mandibular retrusion, and TMJ ankylosis may complicate surgical treatment.

Arthroscopic treatment is described in Chapter 55.

Discectomy

In TMJs affected by chronic polyarthritis the disc is frequently displaced and deformed. The treatment may therefore be similar to that of TMJ osteoarthritis and chronic closed lock. If arthroscopic lysis and lavage fails, discectomy may be performed. However, in TMJs with chronic polyarthritis the chronic inflammation is more pronounced and a selective synovectomy may therefore also be done.

Outcome studies

Very few outcome studies have been performed. Bjornland performed discectomy and synovectomy in 15 patients (25 joints) and followed them in the long term.⁴⁰ Seventy-three percent of the patients improved regarding pain and opening capacity. This study is promising and indicates that discectomy with a selective synovectomy may be the method of choice when arthroscopic lysis and lavage has failed.

Complications

The complications are similar to those described previously for discectomy and disc repositioning surgery.

Arthroplasty

As previously mentioned there is a small group of patients with chronic polyarthritis that develop ankylosis or open bite/mandibular retrusion. Arthroplasty is described in Chapter 55.

Ankylosis induced by chronic polyarthritis is a complicated condition and is more difficult to treat than ankylosis caused by trauma. The recurrence rate is higher because the disease process is difficult to control. Moreover the anesthetic risk is sometimes higher, especially when the neck is involved. Sometimes the reduced mobility in the neck can limit the access to the operation field.

For ankylosis, several methods have been suggested, such as gap osteotomy, chondrochondral graft, auricular graft, dermis, alloplastic grafts, TMJ prostheses, and distraction osteogenesis. The suggested wide variety of methods indicates that there is still not a satisfactory treatment. More tissue-related research is needed to understand why some patients do not respond to the treatment. All that are involved in TMJ surgery recognize a small group of patients that seem to have recurrence of ankylosis, whatever treatment is given. They show ectopic bone formation in the TMJ and probably represent a genetic variant. The solution to this problem is not yet another prosthesis but to focus more on tissue research.

Sound principles for surgical treatment are that the least invasive method in terms of complexity, trauma, social complication, and cost that benefits the patients should be preferred. At present, both for children and adult patients, the author prefers to use a gap osteotomy and interposition with temporalis muscle. This method may serve as the reference procedure in future randomized clinical trials.

TMJ trauma

In all cases with mandibular trauma, there is also an associated injury of the soft tissues of the joint. The term contusion or traumatic arthritis may be used.

Clinical findings

In unilateral cases there is a lateral open bite on the affected side and the mandible deviates to some extent towards the opposite side (Fig. 56.12). Joint pain is provoked if pressure is applied to the chin. In rare cases the contusion can be bilateral and in these cases a frontal open bite occurs.

Differential diagnoses are fracture of the condylar neck and mandibular luxation. The first is ruled out because the clinical features differ from that of a contusion. In unilateral condylar neck fractures the mandible deviates towards the affected side and the lateral open bite develops on the opposite side. Unilateral mandibular luxation may show the same clinical features as a contusion but there is usually no history of trauma. Radiographic examination is of value in these cases.

Treatment

In most cases with contusion of the TMJ symptomatic treatment is sufficient. The patient is put on a soft diet and is given anti-inflammatory medication and analgesics. Usually, the symptoms subside within 10 days and the occlusion returns to normal. In a few patients an acute closed lock may develop because a lacera-



Fig. 56.12 Traumatic synovitis of the right TMJ after a blow to the chin resulting in a lateral open bite on the right side.

tion in the posterior disc attachment occurs resulting in an anterior displacement of the disc. In these cases, arthroscopy or TMJ surgery may be indicated.

Long-term consequences

It has been shown that patients with TMJ reciprocal clicking and chronic closed lock have a much higher reported occurrence of previous trauma to the jaw compared to controls.⁴¹ Clicking and locking symptoms may develop many months after the trauma episode which is important to remember when insurance matters are dealt with.

TMJ abnormalities

TMJ abnormalities are infrequent compared to functional disorders. However, the treatment can often be complicated and demanding, particularly in children. TMJ abnormalities may be either congenital or acquired.

Congenital abnormalities

The most common congenital abnormalities are hemifacial microsomia (incidence 1 in about 5600 living births) and Treacher Collins syndrome (incidence 1 in about 10000 living births). These conditions are best treated in specialist centers where a sufficient number of patients enable development of experience and there are more possibilities for evaluation. Some of the surgical treatment methods are described in Chapters 49a and 49b.

Acquired abnormalities

Condylar hyperplasia

Excessive growth of the mandible may affect only the condyle, the condyle and the ramus, or the whole mandible. The condition is usually unilateral. Several thorough reviews have been published but it is still unclear how the condition develops and progresses. It is more common in females than in males and it usually becomes detectable in the early teens. In these patients the excessive growth usually ceases when normal growth has ended. However, there are also cases where a recurrence of growth takes place after cessation of normal growth. In these patients the growing condyle is not only hyperplastic but also shows asymmetric growth; it has been questioned whether these cases are true condylar hyperplasias or may instead be development of an osteochondroma.⁴²

Classification

The condition may be either of a vertical or rotational type. In patients with the vertical type the growth has a mainly vertical direction leading to a long ramus of

the mandible and a lateral open bite on the affected side depending on the age at which it occurs. If the patient is young, the maxilla can grow down to match any open bite, instead producing an occlusal cant. Usually there is no contralateral crossbite and only a slight deviation of the chin point or dental midline.

In patients with a rotational type, besides a long ramus, the chin point deviates to the contralateral side. Intraorally, there is a crossbite on the contralateral side. The dental midline also deviates to the normal side.

Diagnosis

It is most important to determine whether the condition is in a residual (inactive) stage or not. Repeated occlusal assessments may be one method of detecting growth. However, compensatory growth of the maxilla may occur concealing the actual growth. Radiographic examination may reveal a size difference of the condyle between the right and left sides which may indicate condylar hyperplasia. However, it has to be remembered that the condyle normally shows variation in size and form. Especially when only a panoramic radiograph is taken the distortion can be considerable depending on the angle of the long axis of the condyle. A CT scan is therefore more reliable. Bone scintigraphy, especially when combined with CT, is a reliable method for detection of metabolic activity in the condyle and may also show if the activity is found in other parts of the mandible or only related to the condyle. The uptake in the condyle must be pronounced and clearly differ from that of the contralateral condyle. It must also be remembered that other conditions, such as osteomyelitis and arthritis, may also show a high uptake.

Surgical treatment

In the growing child there are two options. If the child has a mild anomaly and is close to when cessation of normal growth is predicted, observation may be an option. However, if the anomaly is considerable and there is a long time until cessation of growth, a resection of the abnormal growth center in the superior part of the condyle is recommended (Fig. 56.13). After normal growth has ceased and if no further growth is seen in the condyle, orthognathic surgery is undertaken to correct the mandibular asymmetry. In the adult patient, the growing condyle has to be resected and a simultaneous reconstruction performed.

Another important thing to understand is that mandibular hyperplasia is a disease of the mandible. This means that the disc and the temporal component are not affected. Another thing that can be advantageous is that the condylar neck is frequently thicker than normal and may therefore be large enough to act as a new condyle. This observation has formed the basis for an elegant and simple solution to the reconstruction task (Fig. 56.14). The condyle is first resected via a preauricular approach. The lateral part of the ramus is then exposed via a submandibular approach

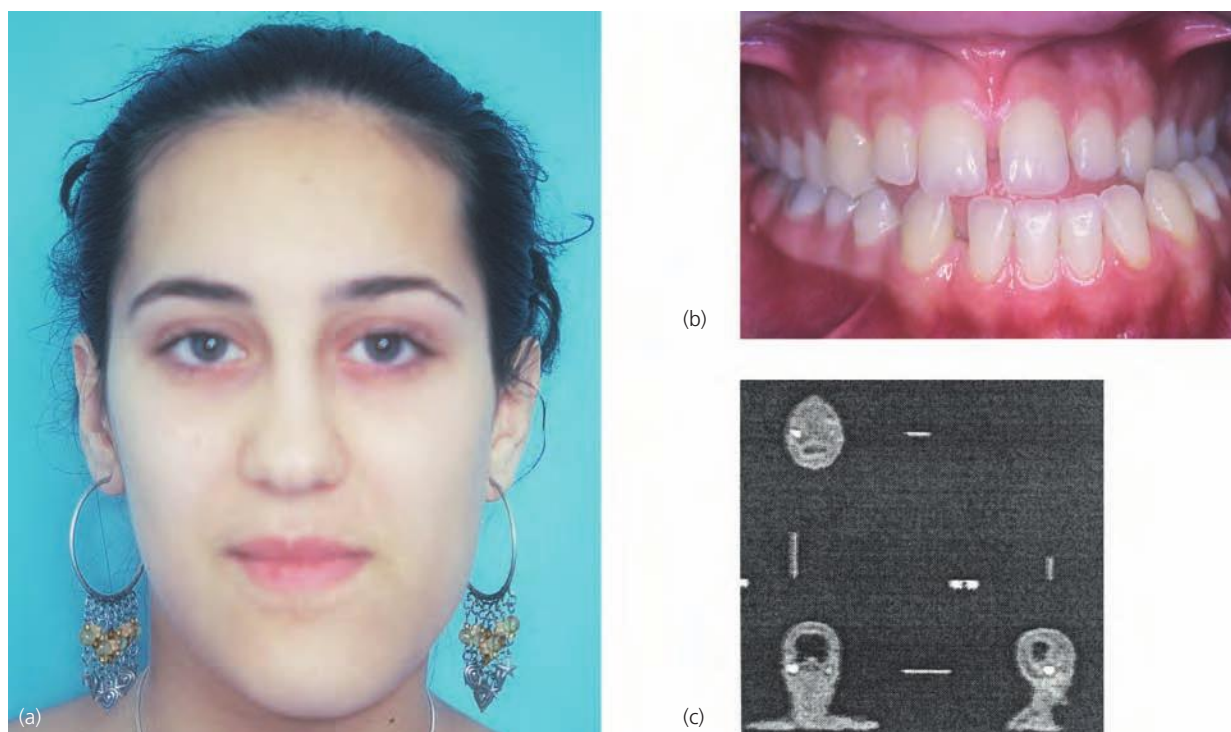


Fig. 56.13 A 14-year-old girl with an actively growing condylar hyperplasia. (a) Clinical picture showing deviation of the chin to the opposite side. (b) Occlusal view showing crossbite on the opposite side. (c) SPEC-SCAN showing increased uptake in the right TMJ.

and a vertical osteotomy performed in the posterior part. This posterior segment may be regarded as a bone-muscle flap as it has muscle attached to it medially. The superior part is smoothened and pushed up against the disc. Fixation to the remaining part of the ramus is done with miniplates.⁴²

Another method recently described for hemifacial microsomia is to use distraction osteogenesis.⁴³ The condyle is resected and an L-osteotomy is then performed in the posterior part of the ramus. The segment is then gradually advanced superiorly to form the new condyle. The method avoids the problem with donor site morbidity but it is complicated and expensive.

The condyle can also be reconstructed using a costochondral graft; this was often used in the past. It involves a certain risk of donor site morbidity and asymmetric excessive growth of the graft, which in turn can result in a mandibular asymmetry.

In cases with the rotational form, simultaneous orthognathic surgery is frequently required as well as pre- and postoperative orthodontic treatment.

Patient numbers are often very small which makes reliable evaluation difficult. A solution for the future may be to perform multicenter studies. Considering the publications up to the present, it seems that the method using the bone-muscle flap is to be recommended.

Complications may include a temporary palsy of the temporal and marginal mandibular branches of the facial nerve or the inferior alveolar nerve. If simultaneous orthognathic surgery is performed there is also a small risk of postoperative infection.

Ankylosis

Ankylosis, by definition, means that the joint is fused with bone. However, the term has often been broadened to also comprise fibro-osseous or fibrous fusion of the joint. The condition may occur after trauma (most frequent), infection, chronic polyarthritis, or as a result of radiation therapy. It occurs both in adults and children, more often in the latter.

Diagnosis

The diagnosis is rarely difficult. There is no translation of the disc-condyle complex and only a limited rotation. In unilateral cases, this is reflected by a marked deviation to the affected side in maximum opening and protrusion. The laterotrusion to the unaffected side is usually zero. In bilateral cases, the interincisal opening usually varies between 5 and 15 mm and there is no protrusion or laterotrusion.

CT examination reveals a bony mass in the joint area. In cases with fibrous ankylosis there may be an arthrotic appearance of the joint surfaces. CT is also valuable to display the medial extent of the bony mass.

Surgical treatment

The surgical protocol involves resection of the bone mass, creating a gap of about 10 mm both laterally and medially. If interincisal opening is still limited after this procedure, an osteotomy (or ostectomy) of the coronoid process may be performed as well. This will usually lead to an interincisal opening of about 35 mm or more.



Fig. 56.14 Patient with a rotational type of condylar hyperplasia. (a) Clinical picture. (b) Intraoral view showing open bite on the right side. (c) Radiograph showing enlarged condyle, collum, and ramus. (d) Simplified drawing showing the surgical technique used to reconstruct the TMJ. (e) Occlusion 5 years postoperatively. (f) Radiograph 5 years postoperatively.

The TMJ may then be reconstructed in several ways. In the past costochondral graft was the method of choice. However, excessive growth of the graft can result in mandibular asymmetry. This occurs most frequently in the child but may also occur in adults. The costochondral graft may be used with or without an interpositional material. Several such materials have been used in the past, autogeneous such as auricular cartilage, dermis, temporalis fascia, and temporalis muscle, or alloplastic, such as Silastic, Proplast, and several models of TMJ metal prostheses. Another recently suggested method is to use distraction osteogenesis (see under Condylar hyperplasia).

Reviewing the literature indicates that TMJ prostheses and costochondral grafts should be used with caution. There are no reliable data showing how long a prosthesis is going to function before it has to be replaced. Since most of the TMJ patients are relatively young this may lead to repeated replacements with a considerable risk of nerve damage. Costochondral

grafts have the disadvantages of donor site morbidity and the possibility of excessive asymmetric growth. Moreover, interposition of alloplastic materials (Proplast and Silastic) has led to problems of fragmentation, giant cell formation, and failure.

As previously mentioned there is in general a lack of well-designed outcome studies comparing different methods. Moreover, the numbers are small. As mentioned before we therefore have to rely solely on clinical experience which is not ideal. Until more research has been carried out, it seems most recommendable to rely on gap osteotomy with interposition of a temporalis muscle flap as the standard procedure.⁴⁴ The thick muscle flap may fill the gap and prevent an open bite. A new condyle may also be created surgically by advancing the posterior segment of the ramus after performing a vertical osteotomy (Fig. 56.15). An alternative method may be to use distraction osteogenesis. However, this method is more complicated and expensive.

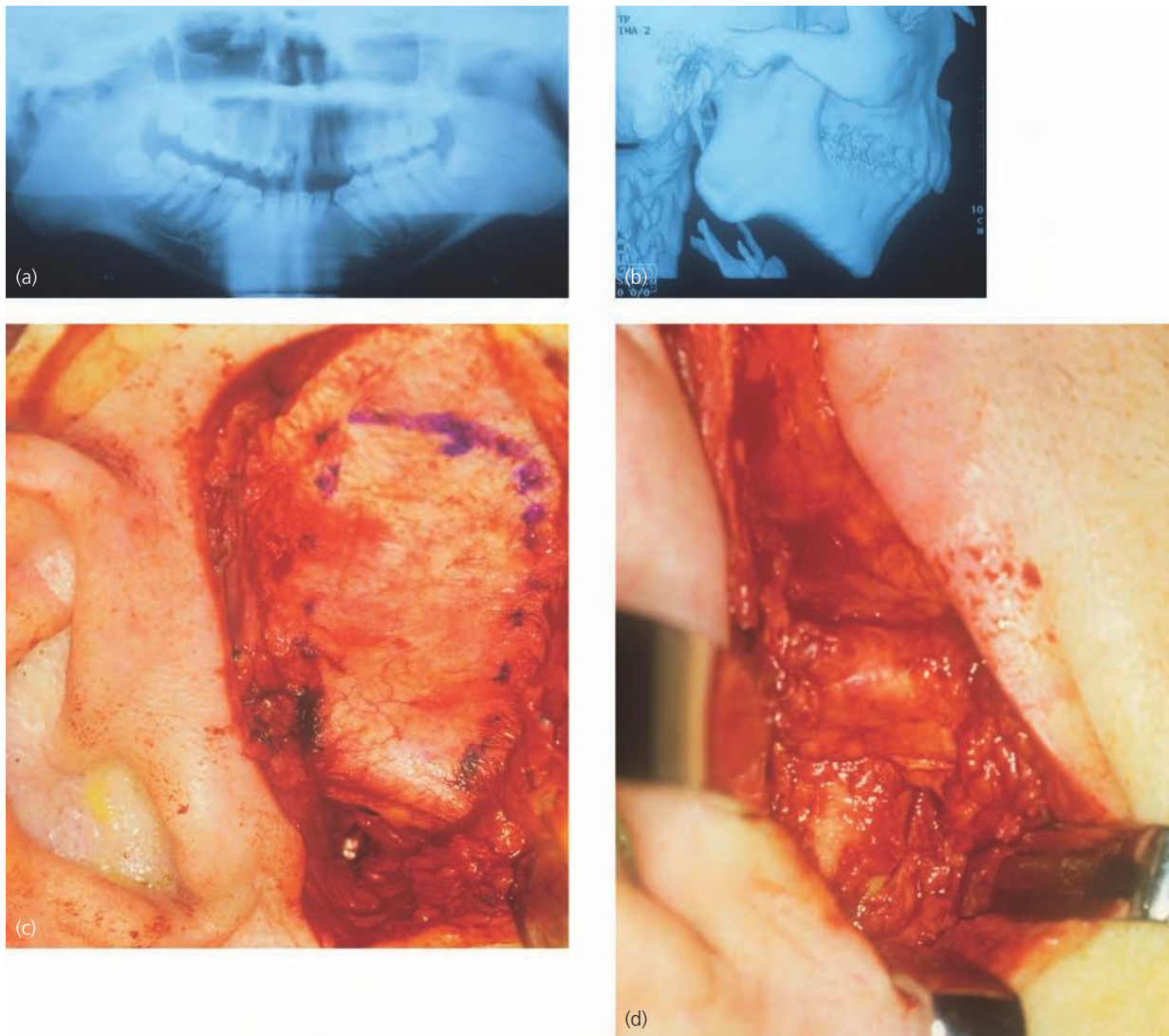


Fig. 56.15 Patient with ankylosis of the right TMJ after trauma. (a) Preoperative panoramic radiograph. (b) Preoperative three-dimensional CT (notice the elongated coronoid process on the right side). (c) The right TMJ after resection of the ankylotic bone. The temporalis muscle/fascia flap is outlined. (d) The flap rotated down and sutured into the joint. The condylar stump is advanced superiorly below the interposed muscle flap after a vertical ramus osteotomy.



Fig. 56.15 (cont'd) (e) Postoperative panoramic radiograph. (f) Maximum opening of 41 mm at 3-year follow-up examination (compares with preoperative opening of 10 mm). (g) Occlusion at 3-year follow-up examination.

The most disturbing complication after TMJ ankylosis operations is reankylosis. The reported figures vary from 0–37%. Complications with costochondral grafts are overgrowth of the graft, malocclusion, persistent pain, and donor site morbidity. Gap osteotomy may have the least complications. Temporalis muscle flap may create muscle symptoms at the donor site. In patients with TMJ prostheses, joint failure, Frey's syndrome, and persistent pain seem to be most common.

TMJ tumors

Tumors of the TMJ are extremely rare. In a review of 3200 tumors of the head and neck only seven (0.2%) were located in the TMJ. The majority of these tumors are benign.

Benign tumors

Benign tumors include osteoma, osteochondroma, chondroma, chondroblastoma, fibro-osteoma, sarcomatosis, and pseudotumors such as synovial chondromatosis and pigmented villonodular synovitis. In this chapter, the most common of these rare tumors will be described, namely osteochondroma and synovial chondromatosis.

Osteochondroma

Osteochondroma is a cartilage-capped bony outgrowth of the condyle and in the literature it has often been confused with condylar hyperplasia or chondromas.⁴² The outgrowth is most common in the anterior and medial part of the condyle. Other loca-

tions in the mandible have also been found, such as the coronoid process. As the outgrowth seems to follow the tendons of the lateral pterygoid and temporal muscles the suggested explanation has been that a continuous strain on these tendons results in an overgrowth of cells with chondrogenic potential.

Osteochondroma of the TMJ seems to occur more often in females, with a later onset than in the long bones where the onset usually takes place within the first two decades.

Diagnosis

The tumor is usually very slow growing, with a gradual deviation of the chin to the opposite side and a lateral open bite on the same side. Usually the condition is symptom-free but slight pain may be present in a few cases. Thus, the indication for surgery is mainly if the patient is disturbed by malocclusion or the esthetics. Two of the author's patients had had their osteochondromas for several years, having no symptoms. They also had a malocclusion and deviation of the chin but declined surgery as they had no symptoms. They were followed for a decade until they passed away showing no additional growth or symptoms. Thus the indication for surgery may not be clear-cut in these cases but has instead to be determined on an individual basis.

Surgical treatment

The surgical protocol is similar to that for condylar hyperplasia. In most cases the TMJ disc is not affected. If the disc is perforated and degenerated it has to be removed and replaced by a temporalis muscle flap. The condyle has to be resected but the condylar neck may be used as a "neocondyle" after performing a vertical ramus osteotomy with superior advancement

of the posterior segment as previously described for condylar hyperplasia (Fig. 56.16). In a few cases simultaneous orthognathic surgery may also be needed.

Outcome studies have been small and no comparative studies have been performed. The limited experience that exists until now seems to favor the above described surgical treatment.

The complications are the same as described for surgical treatment of condylar hyperplasia.

Synovial chondromatosis

The disease is not a true neoplasm but is better regarded as a metaplastic activity within the synovial membrane. For unknown reasons, the synovial membrane starts to produce small cartilage particles which are then released into the joint cavity.⁴⁵ It seems that some of them can grow considerably and the author has found free bodies more than 1 cm in size. The

number of free bodies may be as many as 100. Interestingly, the disease seems to be limited to the upper compartment. If free bodies are found in the lower compartment there is always a disc perforation present allowing the particles to slip into the lower compartment.

Some reports have appeared in the literature indicating that synovial chondromatosis has a malignant potential, but this is not generally believed. On the contrary the disorder seems benign and is easily treated. Nevertheless, until we have more data a long follow-up period is needed in these patients.

Diagnosis

Clinical signs and symptoms may be rather varied (Fig. 56.17a–c). Recurrent swelling lateral to the TMJ without any signs of infection, degenerative or rheumatic disease may indicate synovial chondromatosis. Pain may be present but not always.



Fig. 56.16 Patient with osteochondroma of the right TMJ. (a) Clinical picture. (b) Occlusion is prenormal and deviates to the opposite side. (c) Panoramic radiograph showing a large osteochondroma of the right TMJ. (d) Postoperative panoramic radiograph showing the surgical procedure (resection of the tumor, advancement of the condylar stump, sagittal split on the opposite side). (e) Postoperative occlusion.

A CT scan usually displays free bodies in the TMJ as at least some of the fragments are calcified. However, there are also exceptional cases where no calcified bodies are seen and where the free bodies are also difficult to depict on MRI, because of the high water content of the cartilage fragments.⁴⁶

Arthroscopy is very accurate in diagnosing synovial chondromatosis. Symptomatic relief can also be obtained by flushing out the majority of the smaller fragments and the excess synovial fluid that creates

the swelling. However, the bigger fragments cannot be removed arthroscopically and open surgery is therefore often necessary.

Surgical treatment

Surgical treatment (Fig. 56.17d, e) involves exposure of the joint via a preauricular approach. The free fragments are removed, paying attention to the anterior and medial recess where a number of fragments may hide. Synovial areas with evident chronic inflamma-

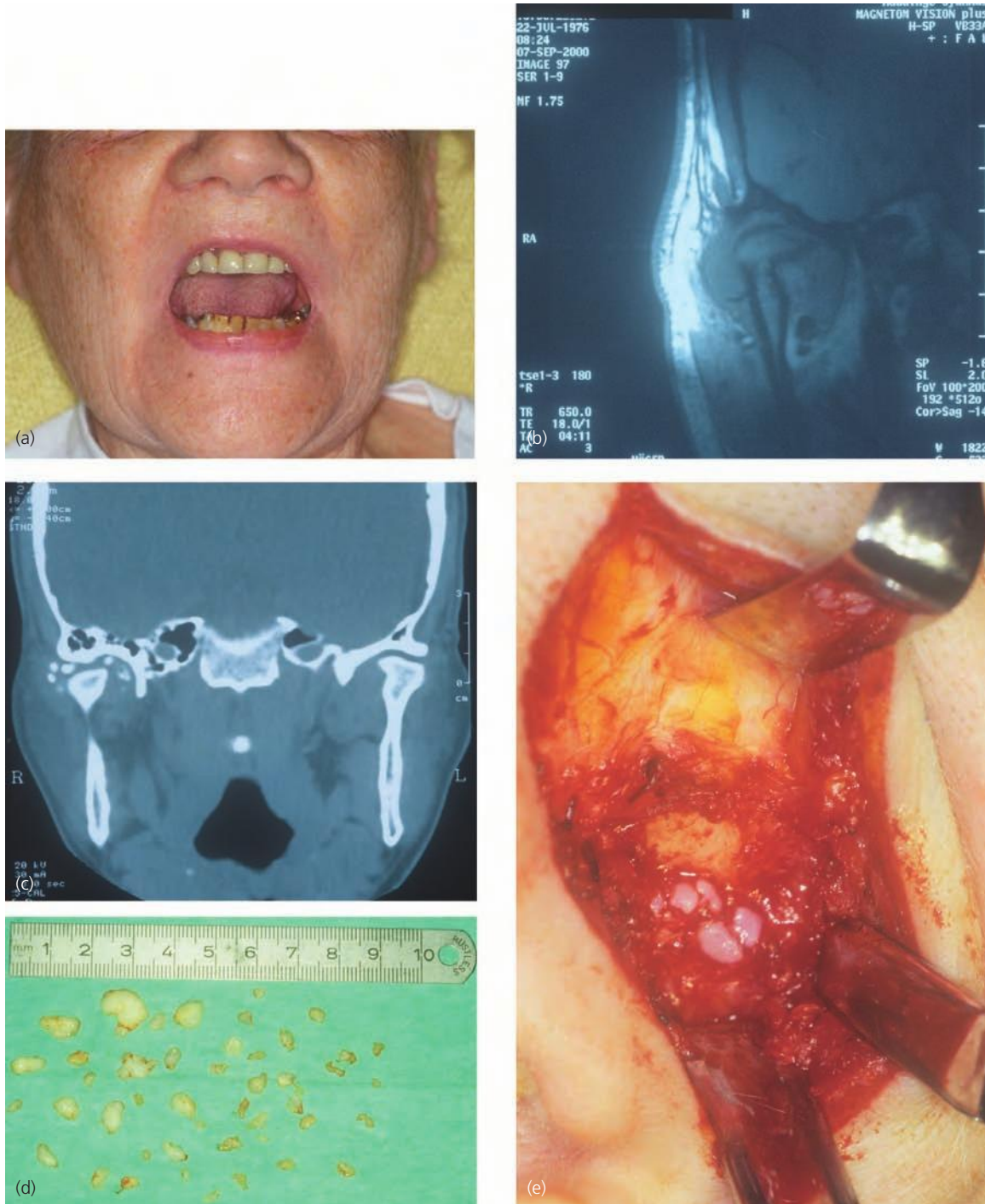


Fig. 56.17 Synovial chondromatosis of the right TMJ. (a) Clinical picture. Notice the swelling over the right TMJ. (b) Preoperative MRI showing an expanded TMJ capsule and fluid. (c) CT showing some calcified free fragments in the TMJ. (d) Surgical exposure of the right TMJ. Fragments emerge from the joint on incision of the lateral capsule. (e) The fragments found in the joint. Some of them are almost 10mm in diameter.

tion may be resected, using diathermy or a laser. If an associated perforated disc is found the disc may be excised without any replacement.

Similar to other rare TMJ conditions, there are few reports in the literature. A recent study of nine patients with a clinical and MRI follow-up between 1 and 6 years indicated that the above described conservative approach for surgical treatment may be best,⁴⁵ at least until multicenter comparative follow-up studies provide more thorough information.

Recurrence may be a complication as may malignant transformation. The risk seems to be extremely low. Other complications may be similar to those for TMJ disc derangement.

Malignant tumors

Malignant tumors in the TMJ are extremely rare. The author has only diagnosed two malignant tumors, one in the TMJ and the other in an extra-articular location. The tumors may be either primary or metastatic. Metastatic tumors are usually carcinomas originating from a wide variety of primary tumors. Primary TMJ tumors are sarcomas of chondral, fibrous, synovial, or bony origin. It is necessary to try to describe the criteria that differentiate a malignant tumor from a benign one.

Diagnosis

A malignant tumor may have clinical signs and symptoms that show great resemblance to that of TMJ arthritis or chronic closed lock, e.g. swelling, impaired mobility of the TMJ, and pain. The author can relate a case where the patient was initially treated for a chronic closed lock of the TMJ for a period of several months before referral. The clinical signs and symptoms indicated no malignant tumor. The panoramic radiograph did not indicate any arthritis or tumor and it was not until a CT scan was performed that the diagnosis became evident (Fig. 56.18). In cases with signs of TMJ arthritis and chronic closed lock, a CT (or MRI) scan is therefore strongly recommended.

Surgical treatment

Surgical treatment usually includes a craniotomy with a wide excision of the tumor, radiation therapy (if the tumor is not radioresistant), and chemotherapy. Reconstruction may include temporalis muscle flap and distraction osteogenesis or orthognathic surgery.

Complications may include permanent palsy of the facial nerve, Frey's syndrome, and recurrence of the tumor.

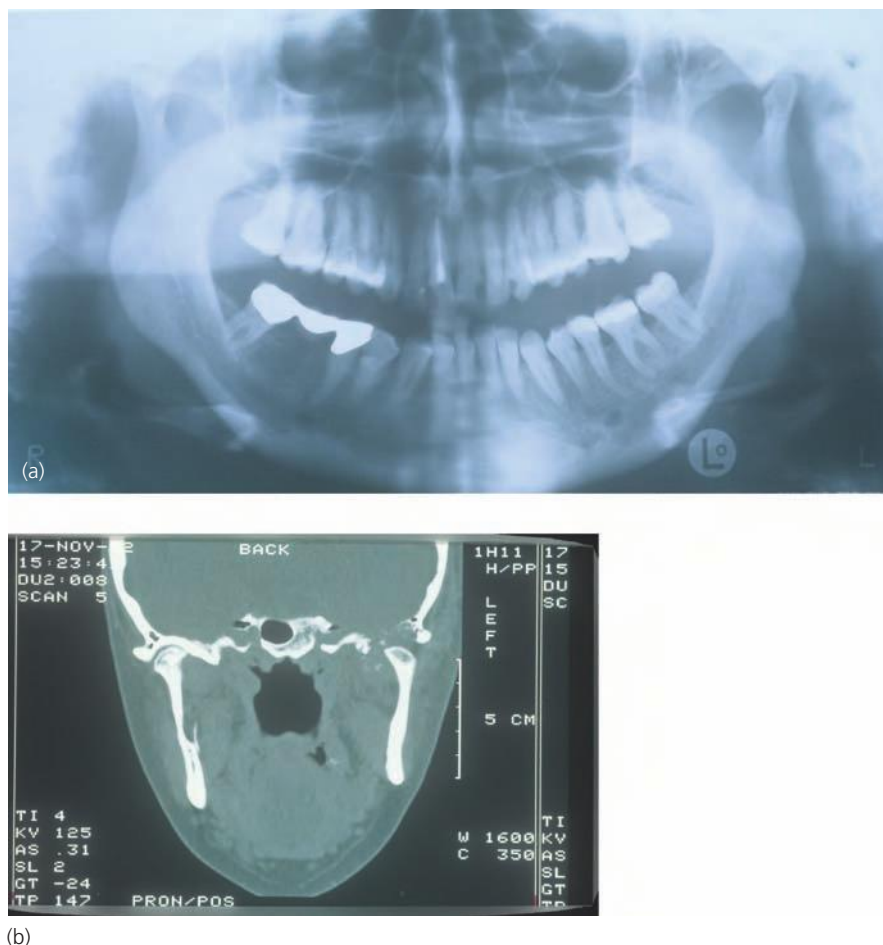


Fig. 56.18 Patient with an osteogenic sarcoma of the TMJ. (a) Initial panoramic radiograph giving no indication of a destructive process in the TMJ. (b) CT showing the typical features of a malignant process in the TMJ.

TMJ infections

In the past infections of the TMJ were not uncommon and even nowadays such infections exist in countries where access to health care is limited. Infections may reach the TMJ from a penetrating wound, adjacent tissues, or by hematogenous spread.

Penetrating wounds may include TMJ surgery or condylar fractures with laceration of the external auditory meatus. Ear infections may spread directly to the TMJ because of the very thin barrier between the ear and the TMJ. Infection may spread from the mouth and the teeth through the pterygoid and infratemporal spaces. Recently, much interest has been focused on the possibility of hematogeneous spread of some microorganisms known for their ability to induce reactive arthritis. One such microorganism of interest is *Chlamydia trachomatis*.⁴⁷

Clinical diagnosis

The clinical signs and symptoms may vary. In some patients the swelling is evident, yet in others the swelling is discrete and may be difficult to distinguish from a traumatic synovitis or chronic polyarthritis. A thorough history is important: Is there a generalized joint disease? Any trauma? Bruxism? Teeth infection? Ear infection? Are there any systemic signs of infection such as fever and leucocytosis?

Usually there are no hard tissue changes in the early phase of the infection. However an increased joint space may be seen as well as changes in the occlusion (lateral open bite on the same side). MRI is more accurate and may also demonstrate abscesses in the adjacent structures. Fig. 56.19 shows a patient with a suppurative TMJ infection which originated from a postoperative infection after removal of the lower left third molar.

Surgical treatment

Incision and debridement of the abscess is important. Before incision, aspiration is performed for culture and determination of suitable antibiotics. The joint may be irrigated arthroscopically in some cases. If the joint tissues have been damaged it may be necessary to perform an arthrotomy and remove necrotic tissue and adhesions.

Extra-articular connective tissue diseases

Hyperplasia of the coronoid process

Mandibular hypomobility is most often related to TMJ pathology or has a muscular origin. However, there are also other conditions that can impair mandibular mobility. Hyperplasia of the coronoid process

is such a condition, and is sometimes overlooked. In the literature it has been reported occasionally⁴⁸ since the end of the 19th century when Jacob described the first case.

Diagnosis

The clinical complaint is usually slowly progressing restricted mandibular opening. The patient usually points to the anterior part of the superior insertion of the masseter muscle and not to the TMJ. There is no history of clicking or ear pain. In most cases translation can be palpated in the TMJ when performing protrusion and laterotrusion. Sometimes crepitation may be experienced by the patient in the area of the coronoid process.

CT provides valuable information. The picture may vary from patient to patient. In some patients the coronoid process is elongated and inclined with a clear impingement against the posterior surface of the zygomatic bone or the zygomatic arch. In some patients an osteochondroma has formed and in yet other patients the elongated process seem to have created a "pseudo joint" with the temporal bone. In some patients the coronoid process is very broad, causing impingement on adjacent soft tissues.

If the condition develops in young individuals it seems to be combined with a "square mandible", class II occlusion, and a deep bite (Fig. 56.20).

Differential diagnosis may include buccal eruption of the third molars in the maxilla and hyperplasia of the maxillary tuberosities both of which may narrow the space for forward translation of the coronoid process upon opening. The author has seen two such cases where the coronoid processes were clearly elongated in combination with buccal eruption of maxillary third molars. After removal of the third molars bilaterally the maximum opening increased from 20mm to more than 40mm within a week and no impingement of the coronoid processes was noted despite the suggestion of it by the radiologist (Fig. 56.21).

Surgical treatment

If a clear impingement is diagnosed, the coronoid processes can be removed through an intraoral approach. The coronoid process is exposed in the same manner as for a vertical osteotomy. The process is cut at the base with a bur or a saw and removed.

No long-term, well designed outcome studies have been published as yet. The occasional case reports all seem to report a good outcome.^{48,49} Very few complications have been reported. Infection may be one, which is easily managed.

Fibrosis of temporalis tendon

This condition may show the same clinical features as hyperplasia of the coronoid process.



Fig. 56.19 Odontogenic infection with spread to the TMJ. (a) Clinical picture at admission. (b) Panoramic radiograph showing an empty alveolar socket after extraction of left lower wisdom tooth. (c) Incision and drainage of the abscess. (d) Tomogram 1 year after treatment showing severe destruction of the TMJ but remarkably there were no symptoms.

Diagnosis

In these patients the coronoid process is usually not elongated or hyperplastic but may be broad and short. The tendon is usually painful when palpated intraorally and feels tight.

Surgical treatment

The surgical treatment mainly follows the same protocol as for condylar hyperplasia. However, as there is no impingement, an osteotomy of the coronoid process may be sufficient in these cases. In some of

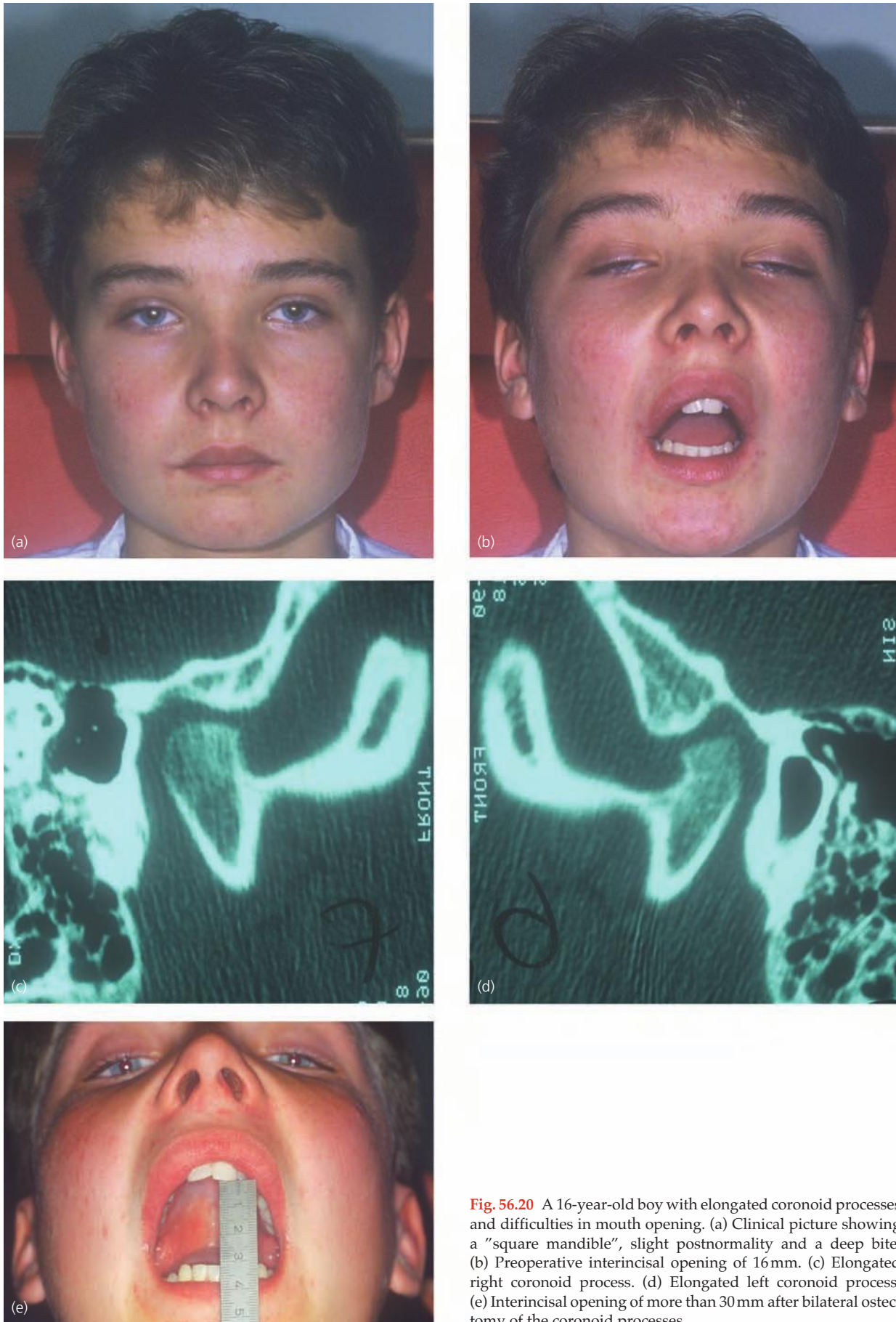


Fig. 56.20 A 16-year-old boy with elongated coronoid processes and difficulties in mouth opening. (a) Clinical picture showing a "square mandible", slight postnormality and a deep bite. (b) Preoperative interincisal opening of 16 mm. (c) Elongated right coronoid process. (d) Elongated left coronoid process. (e) Interincisal opening of more than 30 mm after bilateral osteotomy of the coronoid processes.

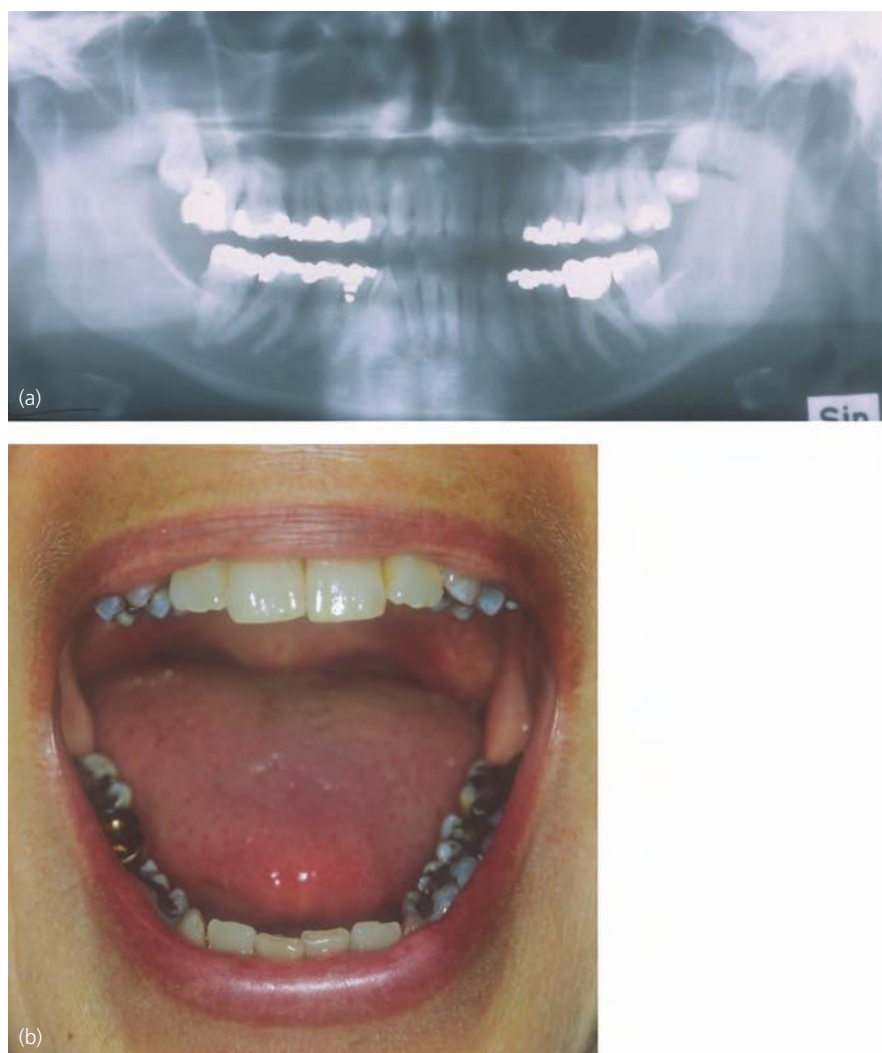


Fig. 56.21 Patient with chronic pericoronitis of impacted upper wisdom teeth. The teeth were partly erupted and caused impingement on the surrounding soft tissue. Maximum opening before extraction of the teeth was 15 mm. (a) Preoperative panoramic radiograph. (b) Maximum opening 2 weeks after extraction.

these patients the masseter muscle and its attachment also show fibrosis and sometimes even thick bands. These can often be released through the same intra-oral incision.

The occasional reports published indicate that the above protocol may have a good outcome in some patients. However, the recurrence rate seems high for these patients. The complications are the same as described for condylar hyperplasia.

Eagle's syndrome

The condition is related to an elongated styloid process. Normally the process is about 2.5 cm long and a process longer than 3 cm is regarded anomalous. Styloid process anomaly was first mentioned by Marchetti in 1652. Eagle described two forms based on the clinical symptoms.⁵⁰ The first category was related to the elongated styloid process and is characterized by pharyngeal pain located to the tonsillar fossa. The second category corresponded to the stylo-carotid syndrome and is related to persistent pain in the carotid area. The incidence varies in the literature,

reflecting the difficulty in finding precise diagnostic criteria.

Diagnosis

A problem is that many patients show calcification of the stylomandibular ligament on radiographs but have no symptoms. Therefore, only patients presenting with a clearly elongated and deviated styloid process may be relevant. In these patients the tip of the process may also be palpated in the fossa tonsillaris. The clinical symptoms may be a foreign body sensation in the fossa tonsillaris and sometimes pain when swallowing, chewing, or rotating the head. A diagnostic local anesthetic block of the fossa tonsillaris may be useful in cases of uncertainty.

Surgical treatment

Access to the elongated styloid process is achieved through a transpharyngeal approach.⁵¹ An incision is made in the tonsillar area over the palpated styloid tip. The tip is then exposed and resected (Fig. 56.22).

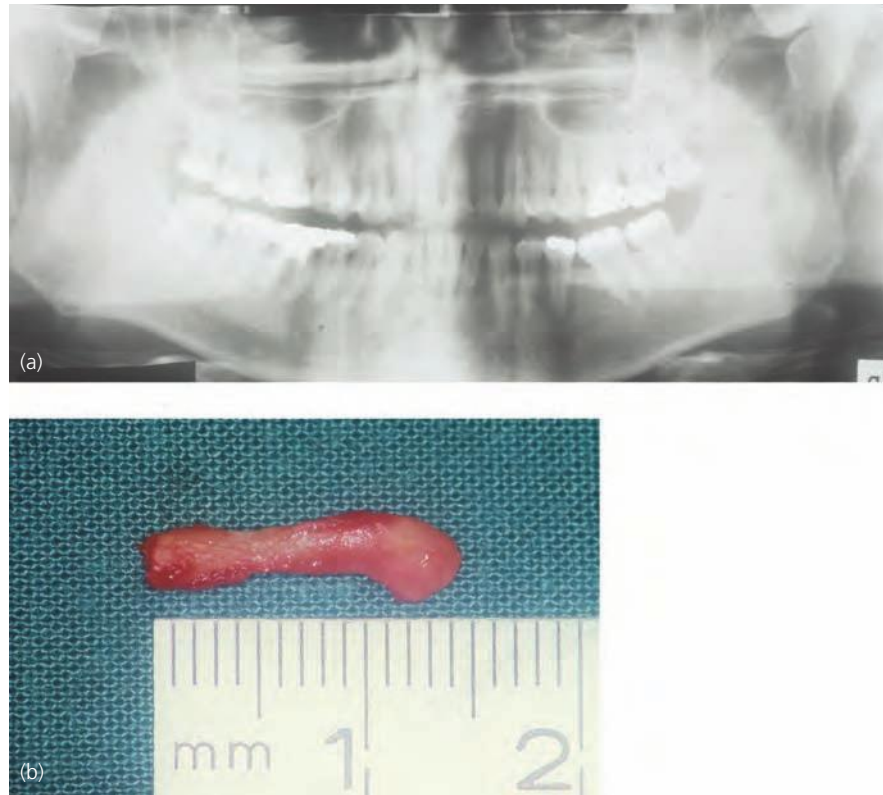


Fig. 56.22 Patient with elongated styloid process on the left side causing pain in the tonsillar area. (a) Panoramic radiograph showing the elongated styloid process on the left side. (b) Excised part of the styloid process.

There is usually no need for tonsillectomy. An extra-oral approach has also been described, utilizing a retromandibular approach.

There are only case reports published. These indicate a good outcome with the above-described technique. Complications may include intraoperative bleeding and postoperative infection.

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Chapter 57

Temporomandibular Joint Reconstruction

Anders Westermarck

When severe destruction or ankylosis of the temporomandibular joint occurs, joint replacement may be necessary. Although there are a number of autogenous sites that have been used over the years, the costochondral graft is the one that is most frequently employed. Alloplastic joints are now available and there are a handful of different types that are manufactured and commercially available. Most are similar to orthotopic alloplastic joints in that they are often made of titanium with a chrome/cobalt surfaced head and an ultra high molecular weight polyethylene glenoid fossa.

This chapter will describe the indications for joint replacement and the materials and techniques available.

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Temporomandibular joint (TMJ) reconstruction is not a treatment for general TMJ problems. It should be considered end-stage surgery, and is therefore a rather exclusive treatment modality. Indications for TMJ reconstruction include:

- severe degenerative joint disease;
- bony ankylosis;
- irreparable condylar fracture;
- neoplasia requiring resection of joint components;
- congenital malformation;
- previously failed reconstruction.

The term TMJ reconstruction should be reserved for the situations when both the condyle and the fossa are reconstructed. Otherwise, one should use terms such as condylar replacement and fossa reconstruc-

tion, respectively. In various reconstructive procedures of the TMJ one can choose between autogenous material and alloplastic devices.

Autogenous reconstruction

As with most things in medicine and surgery, there is little that is truly new. In 1927 Wassmund described the use of free metatarsal grafts for condylar replacement.¹ Other donor tissues that have been used for condylar replacement include clavicle,² iliac crest,³ and vascularized fibula.⁴ Of all the autogenous donor sites, however, ribs have been used more than anything else. Rib grafts can be used either as free⁵⁻⁸ or vascularized⁹ costochondral grafts. The costochondral graft consists of both bone and hyaline cartilage.

The popularity of costochondral grafts for condylar replacement is based on several factors. Ribs are relatively easy to harvest. The size of the graft usually fits the native TMJ dimensions, and it is rather easy to adapt the rib and its cartilage to the local conditions. In patients who are not skeletally mature, the costochondral graft may have some persisting growth potential.

The growth potential is, however, a potential disadvantage of the costochondral graft. The graft may grow too much, too little, or not at all. Children who have had condylar replacement with rib grafts may have to undergo further surgical procedures to correct the jaw position later on.

The downside of costochondral grafts also includes things they have in common with other autogenous grafts, such as ankylosis/reankylosis (Fig. 57.1), resorption (Fig. 57.2), and donor site morbidity. While costochondral grafts for condylar replacement do relatively well in many instances, they are not overly reliable in the treatment of TMJ ankylosis. Saeed and Kent found that a preoperative diagnosis of ankylosis was associated with an *“increased complication and further surgery rate, suggesting caution in this group of patients”*.⁷

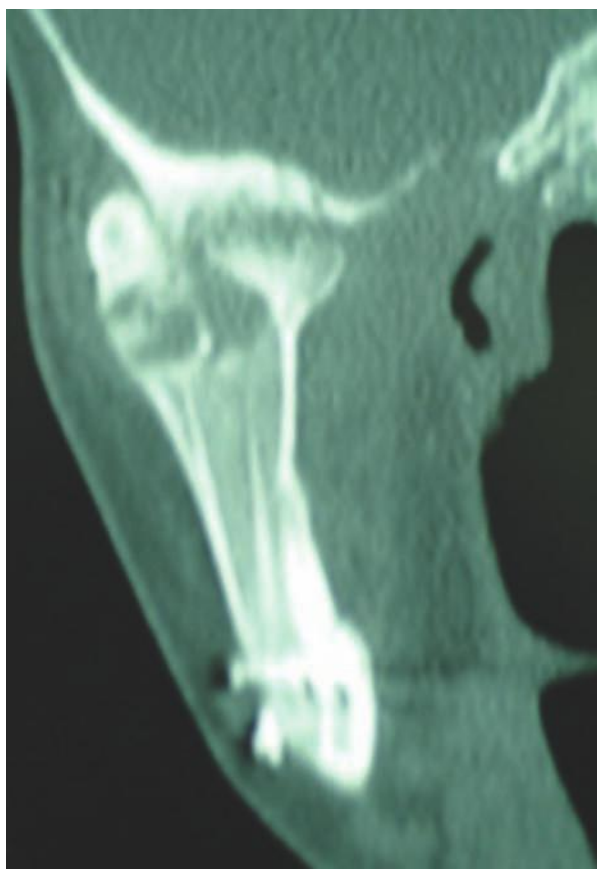


Fig. 57.1 CT scan with a frontal view of a costochondral graft attached with screws to the lateral aspect of the mandibular angle. The radiolucent area under the skull base represents the cartilaginous part of the costochondral graft. The contour of the graft can also be identified, although it is surrounded by a robust heterotopic bone formation.

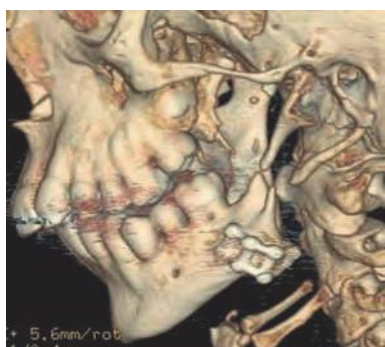


Fig. 57.2 Along with resection of the mandibular ramus in an 8-year-old boy, the ramus and condyle were reconstructed with a costochondral graft. The graft has undergone degeneration that has resulted in loss of continuity mid-way. Note that the cartilage of the graft does not appear in this three-dimensional image.

Costochondral grafts for condylar replacement

Usually, the costochondral graft is used as a free, non-vascularized graft. The recipient site should be prepared first, therefore, so that the time from rib harvest to installation can be kept as short as possible.

Kaban *et al.* represent a group of surgeons who have great faith in costochondral grafts for condylar replacement, including the treatment of ankylosis.⁵

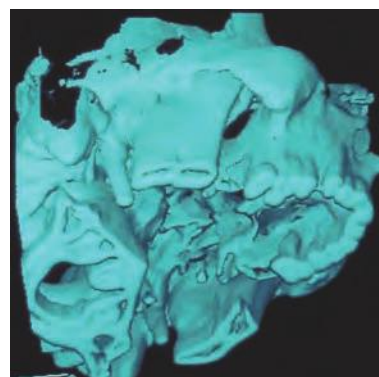


Fig. 57.3 Three-dimensional image of a CT scan obtained from a patient with severe TMJ ankylosis. Note the massive fusion between the mandible and the zygomatic complex. The heterotopic bone formation eliminates the contours of condyle, sigmoid notch, and coronoid process. In this case the coronoid process was fused with the temporal bone.

Their protocol for management of TMJ ankylosis has become more or less the standard approach for costochondral replacement of the condyle in general, not only in ankylosis. Their protocol consists of seven stages: (1) aggressive resection; (2) ipsilateral coronoidectomy; (3) contralateral coronoidectomy when necessary; (4) lining of the TMJ with temporalis fascia or cartilage; (5) reconstruction of the ramus with a costochondral graft; (6) rigid fixation; and (7) early mobilization and aggressive physiotherapy.

The coronoid process often becomes enlarged in association with TMJ ankylosis. This enlargement may interfere with the mobilization of the mandible. Sometimes, the coronoid process may even be fused with the temporal squama or the zygomatic arch (Fig. 57.3). In cases where the condyle is replaced for other reasons than ankylosis, such as rheumatoid arthritis, the coronoid process may be left untouched.

Surgical access to the TMJ area is limited by the extent of the facial nerve. Therefore, the costochondral replacement of the condyle will require two surgical approaches: one preauricular, and one retromandibular or subangular. Because one must harvest a pedicled temporalis fascia flap to line the fossa, the preauricular incision must reach rather high, with a posterior–superior convexity of the incision line. The temporal fascia flap is based just over the root of the zygomatic arch. It is then swung over the lateral part of the arch, to line the roof of the fossa and, ideally, also the medial wall of the wound cavity (Fig. 57.4). It is often surprising how long the flap must be to achieve all this. If, however, the condyle is replaced in patients with a persisting disc, there is no need for a temporal fascia flap.

The access to the mandibular angle and ramus can follow several routes. Either a submandibular or a retromandibular incision is made.¹⁰ The latter has gained increased popularity in spite of the delicate transparotid route and close encounter with facial nerve branches. The reward of this route is excellent

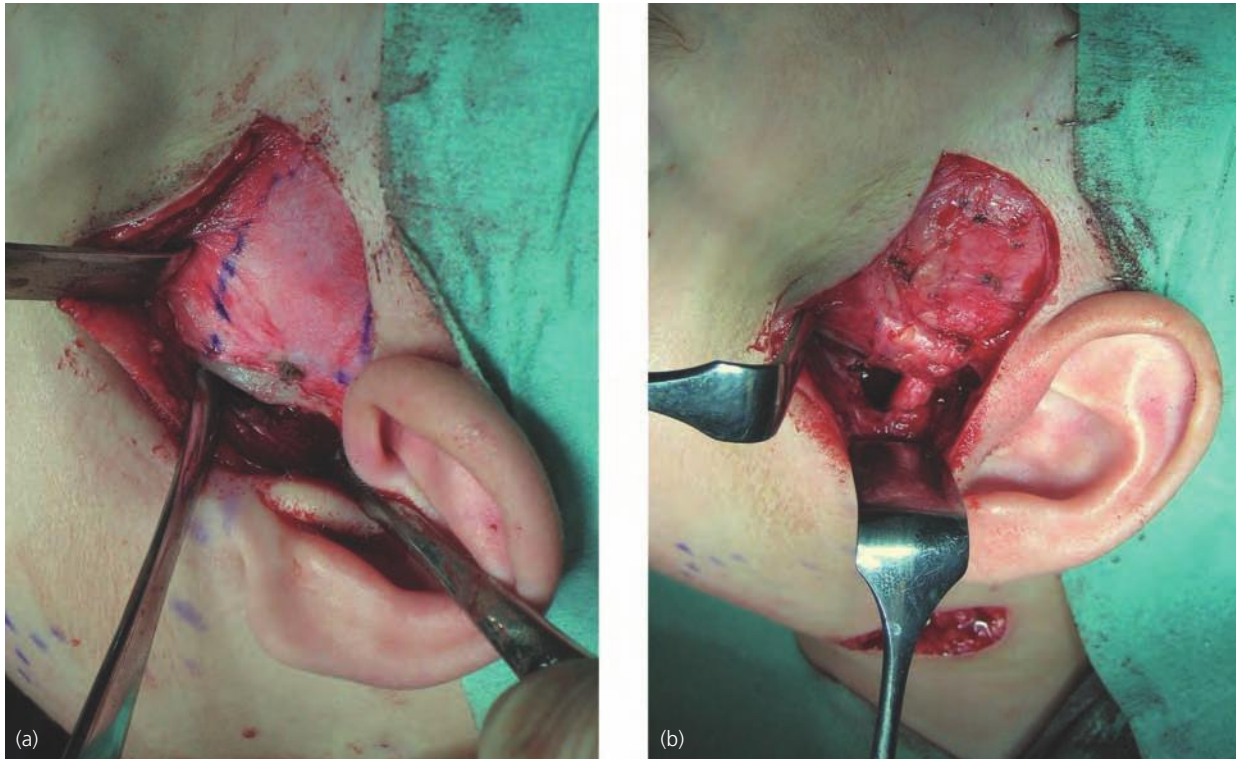


Fig. 57.4 (a) A 3-year-old boy has developed a unilateral TMJ ankylosis following urosepsis. After release of the ankylosis and preparation of the fossa region, a temporalis fascia flap is designed to line out the fossa. (b) The temporalis fascia flap has been brought around the base of the zygomatic arch, and is sutured medially in the fossa cavity. The cartilaginous part of the costochondral graft is resting on the soft tissue lining created by the temporal fascia flap.

exposure of the posterior part of the ramus. The submandibular incision only interferes with the marginal mandibular branch of the facial nerve, but does not reach as high on the ramus as the retromandibular incision does. For most purposes, however, the submandibular approach is sufficient.

The rib harvest is done through a more or less horizontal incision below the inferior border of the pectoralis muscle. Usually, rib harvest is made from rib number five, six, or seven. Sometimes it is suggested that a rib from the contralateral side is easier to fit than one from the ipsilateral side. Thereby, in bilateral cases, one should harvest one rib from each side. On the other hand, it may be preferable to only harvest from one side, to reduce the risk of bilateral pneumothorax. After dissection through the skin and subcutaneous tissues the rib periosteum is cut through parallel to the rib. The subperiosteal dissection is carefully carried out around the rib, 4–5 cm in length. About 1 cm of the hyaline cartilage is enough to serve as the condylar head. It is important to maintain a strip of periosteum/perichondrium around the rib/cartilage junction (Fig. 57.5). This will create a support for the cartilage, which otherwise may separate from the rib. The dissection of this periosteal/perichondrium sleeve is much more difficult than the subperiosteal dissection around the bony part of the graft. The rib is preferably cut with a special rib cutter, which is designed to give as much rib as the incision will allow. After the rib has been removed, it is important to examine the tissue bed for any sign of



Fig. 57.5 Subperiosteal dissection is carried out around rib number six in the same patient as in Fig. 57.4. Note that the periosteum/perichondrium around the junction between bone and cartilage is preserved to prevent the cartilage from falling off the bony part.

pleural tear. The wound is filled with saline, and the anesthetist inflates the lungs maximally. A pleural tear will show up as a bubbling in the fluid. If such a tear is present, it should be closed at this time. Postoperatively, a chest radiograph must be obtained to ensure absence of pneumothorax.

The cartilage part of the rib can easily be carved with a regular scalpel blade, and thus be given an optimal fit into the fossa (Fig. 57.6). The costochondral graft will require a certain period of fixation to fuse with the native mandibular bone. This creates a conflict with the need for early mobilization and



Fig. 57.6 After harvest, the cartilage of the costochondral graft has been cut to fit the contour of the reshaped fossa.

aggressive physiotherapy. Ribs are sometimes also rather soft, especially in younger patients, so rigid fixation may be difficult to achieve. Screws alone may not be enough, and in such cases a plate can be used, to serve as a washer. Steel wires can also be used for fixation of the graft (Fig. 57.7). It is sometimes suggested that both the mandibular ramus and the rib graft should be decorticated over the surfaces that meet, in order to increase healing.⁸ Other authors claim that this is not necessary.¹¹ Kaban *et al.* recommend an intermaxillary fixation (IMF) for 3–10 days, “depending on the thickness and rigidity of the costochondral graft”,⁵ while Quinn recommends IMF for 4–6 weeks.¹¹

After release of the maxillomandibular fixation the patient can start on a soft diet and jaw-opening exercises. Active opening and manual finger stretching are advised at this time. During the next few weeks the diet can be more advanced and the exercises increased. Heat, massage, ultrasonography, gum chewing, and manual stretching exercises can be used in order to reach the opening capacity that was achieved during surgery. If this degree of opening

cannot be reached with physical exercises, one might consider tissue stretching under general anesthesia.⁵

Fibula grafts for condylar replacement

Costochondral condylar replacements are usually applied as non-vascularized grafts. Such an option does not exist for fibula grafts. The fibula is composed of very dense, cortical bone, which requires a vascular supply for survival. With the development of microvascular anastomoses, the fibula became one of the most popular grafts for mandibular reconstruction. The majority of such cases concern mandibular body resections in the treatment of tumors or osteoradionecrosis. Sometimes such a resection also includes the mandibular ramus with the condyle. In such an instance the fibula can be angulated to build up both the mandibular body and the ramus (Fig. 57.8). A triangular piece of the fibula must be cut out for this body and ramus design. Care must be taken that this procedure does not injure the vessels that are feeding both ends of the fibula graft. The joint disc usually can be preserved in these cases, so the top of the fibula will be seated on to the disc, and no further soft tissue lining is needed. Sometimes, the lateral pterygoid muscle can be preserved, and can be sutured to the top of the fibula graft, allowing translational movement of the reconstructed condyle to be achieved.

Autogenous reconstruction of the fossa

Tumors, or tumor-like lesions, may destroy the fossa while leaving the condyle and disc undisturbed. In such situations, the fossa alone needs reconstruction. This can be achieved with autogenous, non-vascularized bone. Access to tumors with this localization

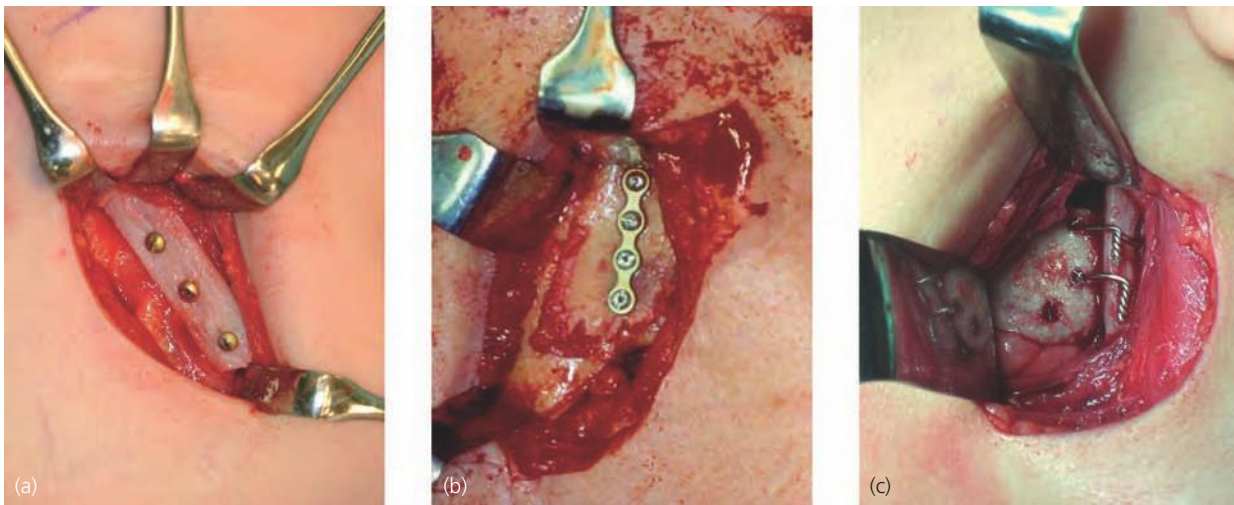


Fig. 57.7 (a) A costochondral graft in a 5 year old, attached with screws only. Care must be taken not to insert the screws with too much force. If the rib is very soft, a plate must be added as a washer, such as in Fig. 57.7b. (b) A four-hole miniplate serves as a washer for the osteosynthesis of a costochondral graft to the mandibular angle. (c) Same patient as in Figs 57.4–57.6. In this case the costochondral graft has been placed along the posterior border of the mandibular ramus, and is attached with steel wire osteosynthesis.

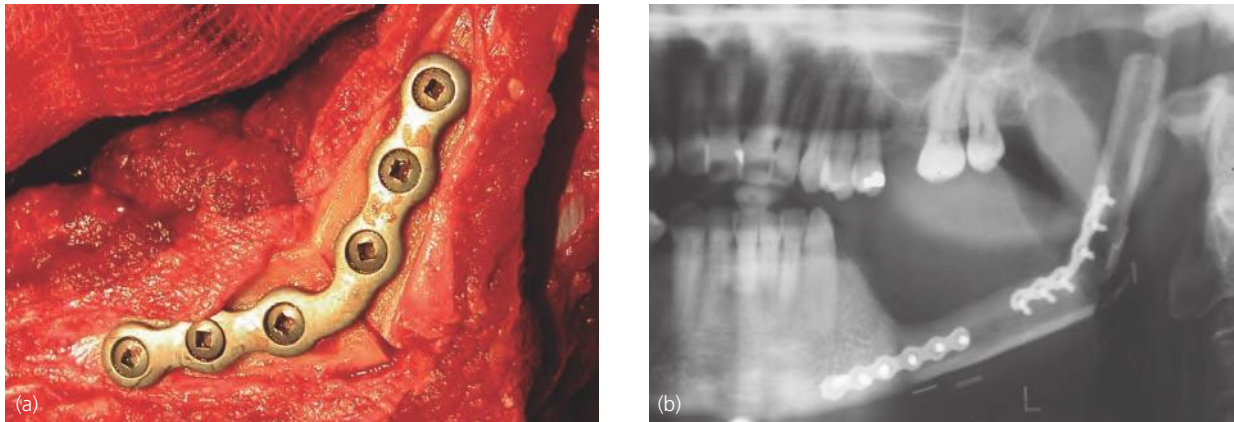


Fig. 57.8 (a) A triangular piece of a fibula graft has been cut out to permit contouring of the fibula to match both the body and ramus of the mandible. If a locking plate system is used, one can refrain from stripping the periosteum under the plate. (b) A vascularized fibula graft reconstructing both the body and ramus of the mandible. Note the bony fusion between the fibula and the native mandible.

requires coronal, or hemicoronal flaps, therefore the calvarial bone is readily available as reconstruction material. A piece of bone corresponding to the fossa defect can easily be harvested as a full-thickness graft. The outer and inner tables of the calvarial graft can be separated with thin chisels. One part can then be used to reconstruct the fossa, and the other goes back to the donor site. The graft can be fixed in the fossa with screws, plates, or combinations thereof. One can also use bone from other donor sites (Fig. 57.9).

Prosthetic reconstruction of the TMJ

In his book, *The Rise and Fall of Modern Medicine*, James Le Fanu writes, "John Charnley's hip replacement is, by definition, a successful operation. It is straightforward (the skills can readily be acquired as part of general orthopaedic training), it works, and it lasts. From this one might readily surmise that it is also 'simple', but that would be misleading."¹² This quotation could just as well have been made about prosthetic reconstruction of the TMJ. Not

only do the surgical requirements resemble those of the hip, but unfortunately, the early mistakes do too. With respect to the TMJ, however, the early mistakes were made some 20 years after the same mistakes had been made in total hip reconstruction.

In the early version of the total hip prosthesis, the cup was lined with Teflon. In about 1 year the cup surface had worn out. Wear debris created severe inflammatory reactions. The joint components had to be removed and replaced with new ones. Later, Teflon was replaced with ultra high molecular weight polyethylene (UHMWP) and the results improved dramatically.

In spite of those early bad results in hip replacement, Teflon was also used in a widespread, early type of total TMJ prosthesis, the Vitek-Kent prosthesis.¹³ Just as with the early hip prostheses, the early results were very good. Soon enough, however, problems arose with severe foreign body reactions due to wear debris. The results were just as disappointing as after early Teflon hip reconstructions. Roughly 26000 Vitek-Kent prostheses were installed, and a large

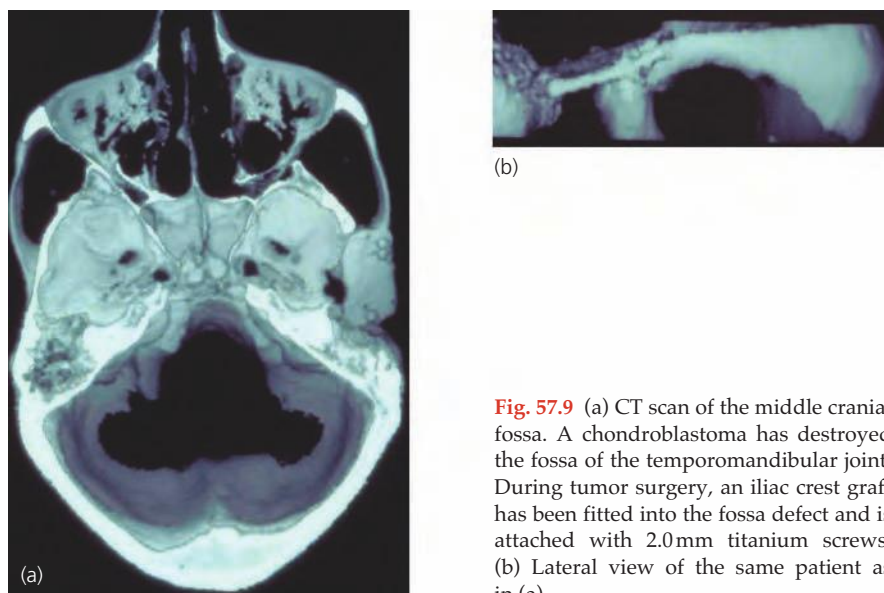


Fig. 57.9 (a) CT scan of the middle cranial fossa. A chondroblastoma has destroyed the fossa of the temporomandibular joint. During tumor surgery, an iliac crest graft has been fitted into the fossa defect and is attached with 2.0mm titanium screws. (b) Lateral view of the same patient as in (a).

number of them had to be replaced later on. In fact, the American Association of Oral and Maxillofacial Surgeons, AAOMS, published guidelines on how to handle failed TMJ prostheses.¹⁴

While the early bad results in orthopedic surgery were soon forgotten and replaced with a faith in the new and better results from improved materials, the same did not occur in the oral and maxillofacial surgical community. For years, the bad results of the early attempts plagued the opinion of prosthetic reconstruction of the TMJ as a reliable treatment modality.¹⁵ Today, however, there are also excellent prosthetic reconstruction devices for the TMJ. For a detailed presentation of the history and development of TMJ prosthetic devices the interested reader is encouraged to read a paper by van Loon *et al.*¹⁶

TMJ prostheses can be divided into two major categories: custom made and stock products. A custom-made prosthesis is one that is made on a model of the patient's skeletal components. Thus, the prosthesis is made to match the patient's individual anatomy. A stock product, on the other hand, is a standard design that comes in various sizes, small, medium, and large. For a stock prosthesis, the bone contour is adjusted to fit the prosthesis. In this chapter, the custom-made TMJ prosthesis is represented by TMJ Concepts, Ventura, California, USA,¹⁷ and the stock prosthesis by Biomet, Biomet Microfixation (previously W. Lorenz Surgical), Jacksonville, Florida, USA.¹⁸

These two prostheses have one thing in common. They both have a fossa made of UHMWP. In the Biomet prosthesis, the fossa is made of UHMWP all through, and the attachment flange comes in three different sizes (Fig. 57.10). In the TMJ Concepts prosthesis the fossa cup is attached to a titanium mesh plate, which is made to fit the patient's individual anatomy (Fig. 57.11). In both types the actual cup of the fossa has only one size. In both types, the fossa



Fig. 57.10 Skull model with a set of Biomet total TMJ prosthesis Biomet microfixation (previously W. Lorenz; Jacksonville, Florida, USA). The mandibular component is made of CoCr with a titanium spray on the surface that faces the bone. There is also a narrow mandibular component. The anterior bulb of the prosthesis here was designed to meet the demands of bony support for screws in patients with previously failed reconstructions, where the posterior part of the ramus might lack good bony support for screw installation. The fossa component is made of UHMWP, and is attached with screws to the lateral aspect of the zygomatic arch.

component is attached to the zygomatic arch with 2.0mm titanium screws.

The mandibular components differ more between the two types. In the Biomet case, the mandibular component is made of cobalt–chromium (CoCr), with a titanium spray on the surface that faces the bone. The mandibular component comes in two different shapes, one standard and one narrow, and they exist in various lengths (Fig. 57.10). In the TMJ Concepts prosthesis, the mandibular component is machined out of titanium. The condylar head is covered with a cup made of CoCr. The mandibular component in the TMJ Concepts prosthesis is first waxed on the patient's model, and then shaped after this model to create the individual fit of the component (Fig. 57.11).

The use of CoCr for the condylar head in both these products is dictated by the wear properties of this material. Both the Biomet and TMJ Concepts prostheses have been subject to experimental testing under conditions vastly exceeding those under normal wear and both are approved by the Federal Drug Administration (FDA) in the USA. Based on the experimental results, one might assume a lifespan of up to 20 years for a total joint prosthesis of such a type. In 2007 Mercuri *et al.* published a study on TMJ reconstruction with up to 14 years of follow-up.¹⁹ They concluded that “outcome data presented show that the patient-fitted total temporomandibular joint reconstruction system continues to be a safe, effective, and reliable long-term management modality for the specific patient population surveyed in this study”. In a recent review Guarda-Nardini *et al.* concluded that “total alloplastic TMJ replacements have shown promising treatment outcomes, and reported improvements are good for both subjective and objective clinical parameters”.²⁰ Basically, the two systems described here, although different in terms of stock vs custom made, are based

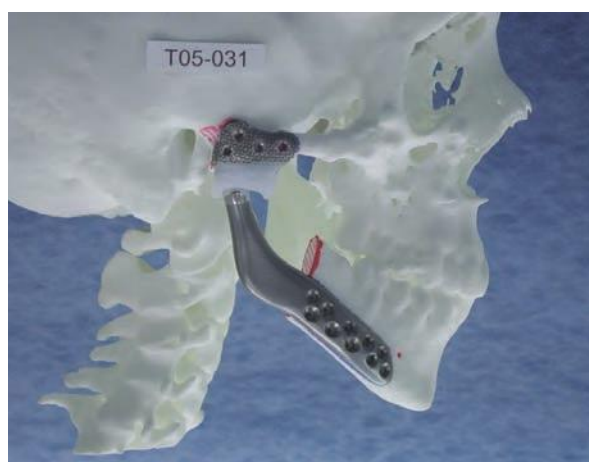


Fig. 57.11 An individual three-dimensional model of a patient where a critical size defect has been created in the preparation for a custom-made prosthesis by TMJ Concepts (Ventura, California, USA). The mandibular component is machined out of titanium, and has a condylar head of CoCr pressed on top. The fossa component is, just like in the Biomet device, made of UHMWP, but is attached to a titanium mesh plate formed according to the individual anatomy of the patient. (Reproduced from [26]. Copyright © 2007 Elsevier.)

on modern orthopedic prosthetic principles, developed over decades of hip and knee reconstruction. The collected experience in alloplastic joint reconstruction in the orthopedic community is vast. Installation of knee and hip prostheses in the USA alone runs around half a million per year, while the corresponding figure for alloplastic TMJ reconstruction worldwide, is a couple of hundred per year.

Surgical considerations in prosthetic TMJ reconstruction

The surgical approaches are basically the same as for costochondral condylar replacement. It is important to keep the surgical fields free from oral contamination. Thus, the oral cavity should be excluded from the surgical field with a surgical drape.

The preauricular incision can usually be made somewhat lower than when a costochondral graft is installed, since there is no need to swing down a temporal fascia flap to line the fossa. For the approach to the lateral aspect of the ramus, personal preferences, just as much as anything else, may guide the approach of choice. The dissection should create a subperiosteal tunnel between the two incisions. This tunnel should be wide enough for a little finger to pass through. That is approximately the width required to pass the prosthetic mandibular component through, as well. If this tunnel turns out to be too narrow, it will be much more difficult to widen it once the fossa component has been installed.

In cases with TMJ ankylosis it is important to work meticulously on the mobilization of the mandible. Experience shows that one should be able to pull down the mandibular angle up to 2 cm before one installs the prosthesis. Sometimes such a mobilization will require thorough muscle detachment not only on the lateral, but also on the medial aspect of the ramus. Even after coronoidectomy, the most anterior part of the temporal tendon attachments may persist on the anterior part of the ramus. Once the mandible has

been thoroughly mobilized, the fossa component can be installed. If a stock prosthesis, such as the Biomet, is used, the eminence must be flattened out (Fig. 57.12). The eminence represents the area with the widest range of irregularity in the joint. In the Biomet kit there is a diamond burr which has a contour that matches that of the fossa component. Once the eminence has been flattened out, the sizers (or trials) for the fossa component can be tested. The cup of the fossa is the same in all sizes, only the flange that attaches the fossa to the zygomatic arch differs between the various sizes. Care must be taken to preserve enough bone to attach the screws on the lateral aspect of the arch. Stability is crucial. With the stock fossa one should aim at a tripod support. With a finger one can push the fossa component upwards, and it should stay there without wobbling. Thereafter, the sizer, or “dummy” is removed, and the true prosthetic fossa component is installed. It should be attached with as many screws as the flange holes and available bone will allow (Fig. 57.13).

After the fossa installation, intermaxillary fixation (IMF) must be performed. At this point it is important to realize that the installation of a total TMJ prosthesis requires similar precautions as installation of other joint prostheses. Any contamination of the prosthetic

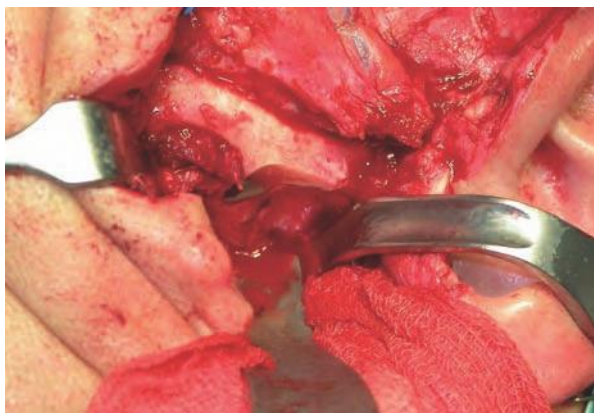


Fig. 57.12 The eminence has been flattened out with a special diamond burr to create space for the standard fossa component of a Biomet prosthesis. Note that the width of the zygomatic arch is maintained to allow for screw installation.

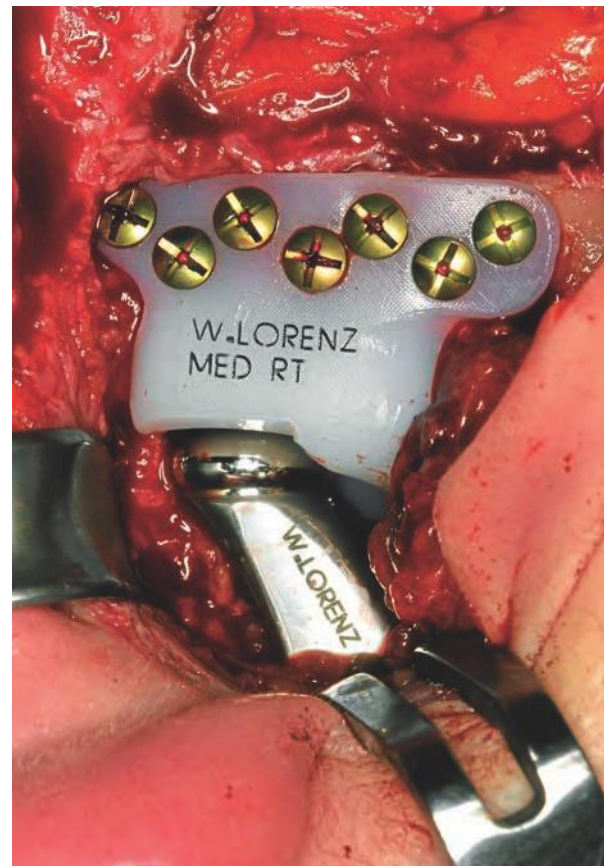


Fig. 57.13 A Biomet total joint prosthesis has been installed. The signs on the fossa component verify that it is a right-sided, medium-size component. All screw holes have been used. Note that the condylar head is seated in the posterior part of the fossa cup. This is the same patient as in Fig. 57.15. (Reproduced from [26]. Copyright © 2007 Elsevier.)

components may establish a biofilm on the surface of the components. This may result in infection that will require the removal of the implants.²¹ Therefore, when IMF is applied, the wounds must be covered and the oral field must be kept separate from the rest of the surgical fields. Once IMF has been established, the mouth must again be covered with a surgical drape. The instruments from the intraoral procedure must be kept aside, and the surgeon and assistant who performed the IMF must change gown and gloves before they continue the surgical work.

When the mandibular component is installed, care should be taken to seat the condyle in the posterior part of the fossa (Fig. 57.13). This will create the best possible conditions for a good postoperative range of motion. If it is a stock prosthesis, the correct size is tested out with the aid of the sizers. If it is custom made, it is made to fit directly.

Stock components may require some bone surface flattening before they lay passively on the bone surface. This part of the procedure is important, since stability is one of the most critical factors for treatment success. For this reason it is a good idea to study a three-dimensional model of the patient before deciding on what type of prosthesis to use. A stock prosthesis may be problematic in cases with major transverse discrepancies between the fossa region and the ramus. If the fossa is seated too far lateral to the ramus, the mandibular component, with the condyle resting in the fixed cup, will be leaning medially

and reach bony contact only towards the basis of the mandibular angle. Then, the bony work to create a wider bony support for the mandibular component will also reduce the thickness of the bone that will hold the screws. Even if bone harvested from, for example, the coronoid process is installed in the gap superiorly, the stability of the mandibular component may be suboptimal. In such a case a custom-made component is a better choice.

When the first screw is installed in the mandibular component, it is imperative that it is properly centered in the hole. If the first screw hole is made eccentric, the screw will force the mandibular component out of the chosen position and this may disrupt the occlusion. After the first two or three screws are inserted, it may be a good idea to release the IMF and test the occlusion before installing the rest of the screws. Such a procedure should be done with the same aseptic technique as when the IMF was applied before installation of the mandibular component.

It is sometimes advised that the prosthetic components be soaked in antibiotic solution before installation, and also that the wound bed is rinsed with such solution before it is closed. Intraoperative intravenous antibiotic prophylaxis, as well as a postoperative course of oral antibiotics should be maintained. It should also be mentioned that the American Dental Association and American Academy of Orthopedic Surgeons have produced guidelines for antibiotic prophylaxis in patients with joint prostheses. They

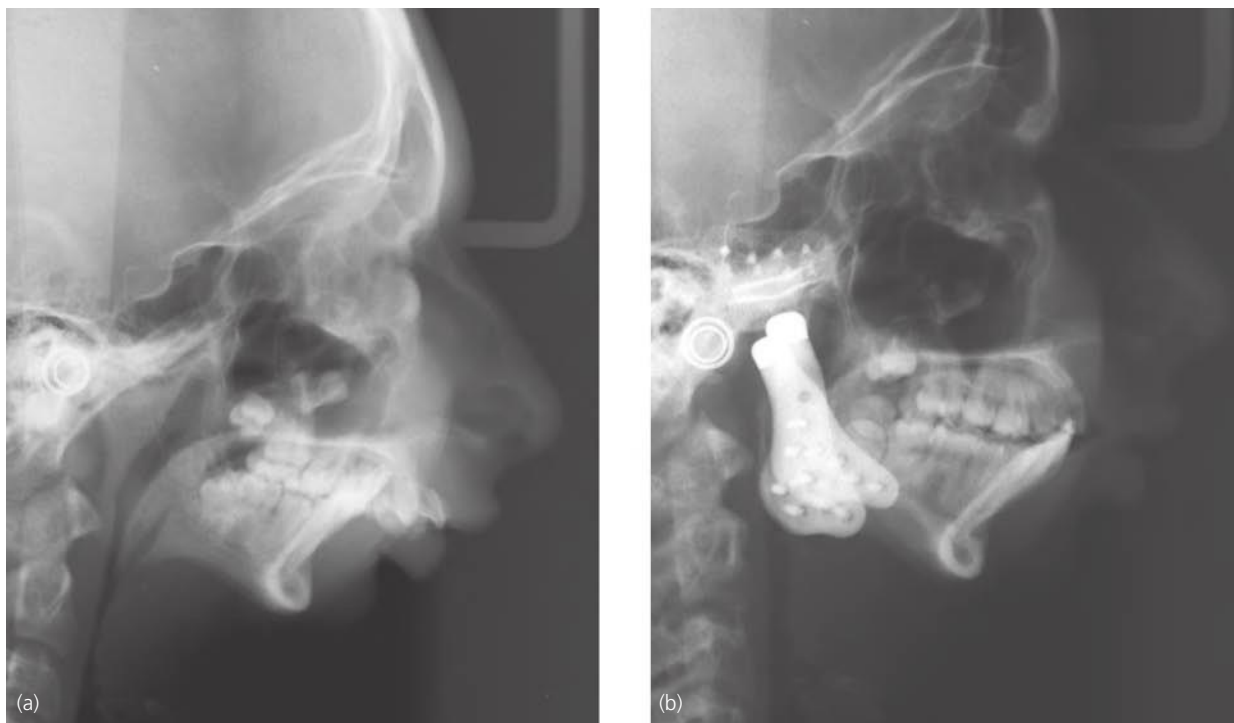


Fig. 57.14 (a) Profile radiograph of a 15-year-old patient who has been ankylosed since a very early age. The resulting growth impairment has been so severe that the patient can no longer defend her airway. Shortly after this radiograph was obtained, the patient had a tracheostomy to secure the airway. (b) Resection of bilateral ankylosis followed by installation of TMJ Concepts patient-fitted total TMJ prostheses has resulted in improved pharyngeal airway space. The patient is released from the tracheostomy. The prosthetic devices withstand the posterior pressure put upon them by the suprahyoid muscles. The mandible was advanced as much as the dentition allowed. A genioplasty will follow, to further improve both the airway space and the patient's profile.

recommend that patients who had their prostheses installed up to 2 years previously, should have antibiotic prophylaxis 1 hour before high-risk dental procedures, such as dental extractions, periodontal treatment, etc.²²

One big advantage with prosthetic reconstruction over costochondral or other autogenous reconstruction, is that the patient can, and actually should, exercise right after surgery. Another advantage with prosthetic reconstruction of the TMJ is that the prosthetic devices can withstand the reactive forces that will arise in cases where the mandible is advanced at the time of TMJ reconstruction. Such situations may occur in patients whose joints are severely destroyed by rheumatoid arthritis (RA). One typical feature of a patient with longstanding RA is the bird face, with a variably severe mandibular micrognathia and retrognathia. In such cases an offensive mandibular advancement can be combined with installation of total joint prostheses. A costochondral graft will not give the same postoperative stability. Similar situations may occur in patients who have been ankylosed since an early age. TMJ ankylosis in early childhood will dramatically impair mandibular growth. The resulting micrognathia may become so severe that the patient cannot defend the airway. With the release of the ankylosis and the advancement of the mandible, the pharyngeal airway will increase (Fig 57.14).²³

For the ankylosed TMJ, the risk of recurring ankylosis is always present. It has already been mentioned that costochondral replacement of the condyle in patients with TMJ ankylosis may result in suboptimal outcome, often because of reankylosis. Reankylosis is, however, also a risk after prosthetic reconstruction of the joint. The prosthetic reconstruction requires a rather large gap between the fossa and the ramus. The gap can reach the width of a critical size defect; in this area such a defect is considered to be 2–2.5 cm (see Fig. 57.11). Recently, fat grafting has been intro-

duced as a new treatment modality to further reduce the risk of reankylosis.^{24,25} A free fat graft can be harvested from just under the umbilicus. The fat graft is packed around the joint components to fill empty spaces, before the wounds are closed

Prosthetic replacement of the condyle alone is not recommended.²⁶ On the contrary, it has been demonstrated that fossa erosion frequently occurs if the native condyle is replaced with a metal one without a corresponding fossa component.²⁷ Fig. 57.15 shows a computed tomography (CT) image of a patient who had the condyle replaced with a metal condyle and no fossa prosthesis. The result is dramatic erosion with the condyle seated in a bony cavity just under the middle cranial fossa. The patient suffered from severe pain and restricted jaw movement. Because of that, and, for protection of the brain, a total joint prosthesis had to be installed (Fig. 57.13). As a rule, alloplastic condylar replacement should only be used for temporary purposes (Fig. 57.16).

Congenital malformations may include complete lack of TMJ or deformed joint parts (Fig. 57.17). For TMJ reconstruction of such cases the treatment modality must be chosen after thorough consideration of available alternatives. Often, a custom-made TMJ prosthesis will offer an excellent treatment option. Both the fossa component and the mandibular component can be designed and manufactured to bridge even major bony defects, and to create support for the soft tissue drape. Fig. 57.18 shows a three-dimensional image of the facial skeleton of a patient with Goldenhar syndrome. In the past the patient had been treated with orthognathic surgery procedures in order to mask some of the facial irregularities that were part of the condition. A TMJ Concepts custom-made prosthesis was chosen for the TMJ (re)construction. The joint was designed partly to give the patient joint movement, and partly to give stability to the mandible and the occlusion. Furthermore, the attachment plate for the fossa was designed to build up the missing zygomatic arch, and the mandibular component to give the contour of the mandibular angle.

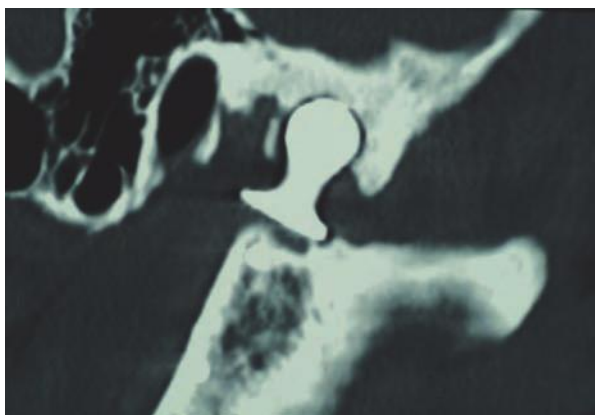


Fig. 57.15 CT scan of a patient who, 15 months earlier, had a metal condylar replacement installed without corresponding fossa component. The condylar head has eroded into the skull base. The patient suffers from severe pain and a very limited jaw opening capacity. The patient went on to have a total joint reconstruction (Fig. 57.13). (Reproduced from [26]. Copyright © 2007 Elsevier.)



Fig. 57.16 In situations like these, a condylar replacement can be used for temporary purposes. A partial mandibulectomy is carried out because of a rare tumor in the angle of the mandible. The unfamiliar behavior of the tumor calls for an observation period before bony reconstruction is considered.



Fig. 57.17 In a situation like this a stock prosthesis is not a treatment option. A lateral facial cleft and hemifacial microsomia have created a situation where lack of bony tissue calls for bony reconstruction or custom-made prosthetic devices.

Treatment expectations after prosthetic reconstruction of the TMJ

In the very beginning of this chapter it was pointed out that TMJ reconstruction is a treatment option for the joint that is no longer a functioning joint. Sometimes TMJ reconstruction is, actually, TMJ construction, such as in cases with congenital absence of joint components. A long-standing ankylosis means that surrounding muscles and soft tissues have adapted to the immobilized joint, sometimes with severely impaired mobility as a result. In congenital deficiencies there may be total absence of muscles to perform the joint movements. Also, because of loss of function of the lateral pterygoid muscle in most TMJ reconstruction cases, translation is severely impaired. There may be some translational movement because of the lowered rotation point. This inferior hinge axis gives a false translational image to the joint movement. A realistic expectation of opening capacity after TMJ reconstruction is around 35mm. It should be emphasized, however, that the opening capacity to a large extent depends on the patients' compliance with their postoperative physiotherapy. Patients who have suffered from ankylosis must be prepared to maintain a lifelong exercise program to maintain a good opening capacity. There are physical therapy appliances to support these exercises, such as the TheraBite (Cranio Mandibular Rehab Inc, Denver, Colorado, USA) (Fig. 57.19). With respect to pain one must remember that, while most of the joint-related pain will be reduced by the installation of a TMJ prosthesis, muscle pain and myofascial pain may persist. As long as one maintains the correct indications for treatment, one can expect an overall improvement of the patient's quality-of-life scores.¹⁹

Many authors demonstrate a direct correlation between a high number of previous surgical interventions in the TMJ, and a poorer outcome of a final TMJ

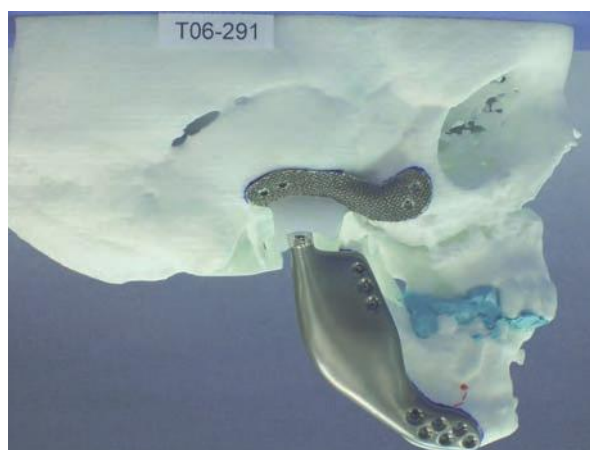


Fig. 57.18 A three-dimensional skull model of a patient with Goldenhar syndrome shows lack of the major part of the mandibular ramus, as well as the entire zygomatic arch and the fossa. All missing parts have been reconstructed in a custom-made total joint prosthesis by TMJ Concepts. The fossa component is designed to also create a zygomatic arch. The mandibular component is designed to give soft tissue support in the angular region. Based on the CT scan, the mandibular canal is outlined. The screw holes in the mandibular component can therefore be oriented to avoid interference with the inferior alveolar nerve.



Fig. 57.19 The TheraBite (Cranio Mandibular Rehab Inc, Denver, Colorado, USA) is an example of a useful device for jaw exercise.

reconstruction.²⁰ This probably depends on the extent of scarring following numerous surgical procedures. Dense scarring creates a fibrous wall that impairs healing conditions both after costochondral grafting and after prosthetic reconstruction of the TMJ. It appears that some patients who have had multiple operations would have done better if they had had their TMJs reconstructed at an earlier stage. These observations may serve as a challenge for the future oral and maxillofacial surgeon to identify, at an earlier stage, which patients should have a total joint reconstruction.

Future perspectives

This chapter was written in 2008. There has recently been a rapid development of both experimental and pragmatic tissue engineering.²⁸⁻³⁰ Thus, various facial bone structures designed with computer assistance

have been produced inside the patients' own bodies. Those achievements have used growth factors and stem cells to build bone. The growth factor in these cases has been bone morphogenetic protein 2 (BMP2). The stem cells have been either those present in a muscle that has been transformed into bone, or stem cells derived from bone marrow or body fat. The author of this chapter is confident that the surgical community in the future will develop substances and methods with which we can engineer complex, multipotent autologous structures to become spare parts identical to those they replace. The rate of the development leading to such ideal TMJ reconstructions remains to be seen.

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